

MEDICAL SURVEY OF THE PEOPLE OF RONGELAP AND UTIRIK ISLANDS ELEVEN AND TWELVE YEARS AFTER EXPOSURE TO FALLOUT RADIATION (MARCH 1965 AND MARCH 1966)

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MEDICAL SURVEY OF THE PEOPLE OF RONGELAP AND UTIRIK ISLANDS ELEVEN AND TWELVE YEARS AFTER EXPOSURE TO FALLOUT RADIATION (MARCH 1965 AND MARCH 1966)

Introduction

The results of a medical survey of the people of Rongelap in the Marshall Islands, carried out in March 1965 and March 1966, 11 and 12 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 rads 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, re-

ceived only an external gamma dose of about 69 rads. 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 an estimated 14 rads 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 2 days after the accident and taken to Kwajalein Naval Base about 150 miles to the south, where they received extensive examinations for the following 3 months. During this period vigorous efforts were necessary to decontaminate the skin completely (see Figure 2).

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 slight enough to allow safe habitation. Because Rongelap Atoll was considered to be too highly contaminated, a tempo-

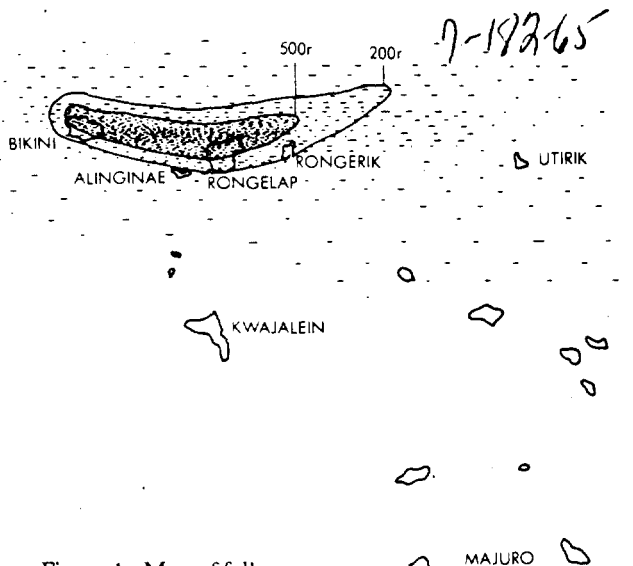


Figure 1. Map of fallout area, Marshall Islands, March 1, 1954.



Figure 2. Marshallese bathing in lagoon at Kwajalein in March 1954 to decontaminate skin and hair after fallout contamination.

rary village was constructed for the Rongelap people (including the 18 from Ailingnae) 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 radioactive contamination, 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. The number has since increased to >200. Following the initial survey of the Utirik people on Kwajalein in 1954, repeat surveys have been carried out on these people about every 3 years, including the 12-year survey. In addition, during the past survey, as in the previous surveys, a visit was made to Kwajalein and Majuro Atolls for examination of a number of Rongelap people now residing at these atolls, and also groups of children who represent parts of the control group used for the growth and development studies of the exposed children.

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.

Table 1 lists exposure data on the various populations involved in the fallout. In the following summary, except where data are broken down into more detail, the exposed group includes the people exposed on Rongelap and on Ailingnae (subject Nos. 1 to 86).

Summary of Past Findings

Reports have been published on the medical 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 4 years,⁶ 5 and 6 years,⁷ 7 years,⁸ 8 years,⁹ and 9 and 10 years.¹⁰ Appendix 1 gives a more complete list of reports, including outside publications, on the results of medical surveys of the Marshallese exposed to fallout and includes a section on the radiation ecological studies of these Islands published largely by the University of Washington group. The following is a brief summary of the medical findings previously reported.

During the first 24 to 48 hr after exposure, about ⅔ of the people exposed on Rongelap Island experienced anorexia and nausea. A few vomited and had diarrhea, many also experienced itching and burning of the skin, and a few complained of lacrimation and burning of the eyes. None of these symptoms was noted in the Utirik people (14-rad group). 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 lym-

Table 1

Summary of Fallout Effects

Group*	Composition	Fallout observed	Estimated gamma dose (rads)	Extent of skin lesions
Rongelap	64 Marshallese	Heavy (snowlike)	175	Extensive
Ailingnae	18 Marshallese	Moderate (mistlike)	69	Less extensive
Rongerik	28 Americans	Moderate (mistlike)	78	Slight
Utirik	157 Marshallese	None	14	No skin lesions or epilation

*Also exposed were 23 Japanese fishermen who received a sublethal dose.

phocytes 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 RADIATION

One of the earliest findings indicative of a significant exposure in these people was lowering of levels of *leukocytes* and *platelets* of the peripheral blood. This was most marked in the 64 people on Rongelap who had received 175 rads, and was less marked in the other groups receiving less exposure. The hemopoietic depression was roughly proportional to the dose of radiation received. Even in the 157 Utrik people who received only an estimated 14 rads, it was possible to distinguish slight platelet depression in the group as a whole. The smaller group on Ailingnae and Rongerik showed peripheral blood levels between those of the high and low exposure groups. The chronological records of blood findings on the group exposed on Rongelap are presented in Figures 49, 50, 56, and 65 and Appendix 8, and on the Ailingnae and Utrik groups in Appendices 9 and 10.

Lymphopenia of about half the level of the comparison Marshallese population was evident when the people exposed on Rongelap were first examined on their arrival at Kwajalein 3 days after exposure (see Figures 49, 50, and 65). In children <5 years of age the lymphocytes dropped to 25% of the levels in the comparison children, but showed a slight rise during the following weeks. The lymphocyte level showed a slight increase by 1 year. In the following year mean counts approached the levels of the comparison population but remained slightly below (see Figures 50 and 65).

Neutrophil levels fluctuated considerably during the first month; possibly this was related to the prevalence of beta burns of the skin during that period. Neutrophil depression became evident by 5 and 6 weeks post exposure (see Figures 49 and 65), with levels reaching about half that of the comparison population in the adults and slightly

lower in the children <5 years of age. This degree of neutropenia was insufficient to result in any apparent increased infectious processes, and, indeed, it was noted that neutrophilic leukocytosis was possible in people showing casual infections at this time. Neutrophil levels recovered more rapidly than lymphocyte levels and reached near control levels by 1 year. Subsequent annual surveys have revealed that recovery does not appear to have been complete, particularly in younger and older age groups, during the 10-year period.

Early *platelet* counts showed less fluctuation than other blood counts and fairly consistently showed increasing depression, reaching levels of about 30% that of the comparison population by the 4th week. A spurt of recovery to about 75% of comparison levels occurred during the following few weeks, which was followed by slower recovery but with mean levels never reaching higher than 90 to 95% that of the comparison population during the 10 years post exposure (see Figures 56 and 65).

Erythropoietic depression has not been a consistent finding as with the leukocytes and thrombocytes. Slight depression of red blood counts, hematocrits, and hemoglobin has been noted at times. *Bone marrow* smears taken at 6 months showed no gross abnormalities. Smears taken at 8, 9, and 10 years showed an alteration in the myeloid-erythroid ratio manifested by an increased number of red cell precursors. Depression of peripheral blood elements in the Ailingnae and Rongerik groups was not so pronounced as in the Rongelap group. However, a slight lag in complete recovery in the Ailingnae peripheral blood count has also been noted. The persistent depression of peripheral blood elements in the exposed people makes it appear likely that there is slight residual bone marrow damage.

A general *anemic* tendency has been evident in both exposed and unexposed Marshallese. Price-Jones curves, on the average, showed a slight microcytic tendency. Serum iron levels have been generally normal, and the cause of this anemic tendency has been undetermined.

Reticulocyte counts have been about the same in the exposed as in the unexposed people.

Clinical findings, except for radiation-induced lesions of the skin, patchy epilation, and early gastrointestinal symptoms, revealed no clear-cut disease processes or symptoms which could be re-

lated directly to radiation effects during the first few years post exposure. No prophylactic or specific therapy for radiation effects was ever considered necessary or given. Epidemics of chicken pox and measles that occurred during the first year showed no greater incidence or severity in the exposed than in the unexposed Marshallese people.

During the first months post exposure about $\frac{2}{3}$ of the exposed people exhibited *loss of weight* of several pounds. This may possibly have been related to their radiation exposure, although it was difficult to rule out possible effects due to change of environment.

At 3 years post exposure the *immune response* to primary and secondary tetanus antitoxin was tested and found not to be significantly different in the exposed compared with the unexposed populations.

There were 10 deaths in the exposed populations over the 10-year period (see Table 6). Of these, 2 deaths were recorded as due to malignancies. The 10 deaths that have occurred in the exposed population represent a *mortality rate* of 12.2 per 1000 population per annum, compared with 8.3 for the Marshall Islands as a whole (1960). The somewhat higher death rate in the exposed group is partly offset by the higher proportion of older people; those >65 years of age were originally 20% in the exposed group and only 7% in the unexposed group.

Growth and development studies on the children (height, weight, anthropometric measurements, and radiographic studies for bone age) have revealed slight retardation in growth and development in the boys exposed on Rongelap who were <12 years of age at the time of exposure, particularly those 12 to 18 months of age at exposure. Only slight immaturity was noted in the female children of this group. In studies of children born of exposed parent(s), it was noted that males showed a slight growth retardation and slightly lower levels of peripheral blood elements compared with male children of unexposed parents; however, this latter finding has not been evident since 1963. The slight growth difference does not appear to justify a conclusion that there is an association with exposure of the parent.

It was difficult to evaluate the effects on fertility. However, a review of the *birth rate* of the exposed groups over the past 10 years seems to indicate no noticeable effects of their exposure on fertility. The

50 births represent a rate of 61 per 1000 population per annum compared with 37.3 for the Marshall Islands (1957). The incidence of *miscarriages* and *stillbirths* in these exposed women was about twice that in the unexposed women during the first 4 years after exposure, but no difference has been noted since then (see Table 8).

A *cardiovascular survey* of the adults (1959)⁷ showed no outstanding differences between the exposed and unexposed groups. The Marshallese people appeared to have less hypertension on the whole than is noted in people in the continental United States.

An *arthritis survey* (1959)⁷ showed no great differences between the exposed and the unexposed people, and about the same incidence as is seen in American populations.

Ophthalmological surveys showed no remarkable differences between the exposed and unexposed groups except possibly a slightly greater number of cases of pterygia, pingueculae, and corneal scars in the exposed groups. It is not known whether these findings are 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 accommodation levels in the Marshallese appeared to be above the average in the U.S. population.

*Dental surveys*⁷ showed no significant differences in caries rate between exposed and unexposed groups. However, the incidence and severity of periodontal disease was slightly greater in the exposed group. It is not known whether or not this finding is related to radiation effects. The poor oral hygiene generally observed in the Marshallese had its usual results, namely, high caries rate in teen-age children, severe periodontal 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.

Aging studies were done in which various parameters usually associated with aging were measured or estimated on a 0 to 4+ scale (skin looseness, elasticity, and senile changes; graying of the hair and balding; accommodation, 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 individ-

uals of the same age groups showed no apparent differences. A biological age score was calculated for individuals 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.

Development of thyroid pathology was noted in 1963 when a teen-age exposed girl was found to have a nodule of the thyroid gland. In 1964, two additional teen-age girls were found to have nodules. These cases were operated upon, and the nodules were found to be benign adenomatoid goiters. These abnormalities are believed to be the result of exposure of the thyroid to radioiodines from the fallout and to the gamma radiation. The dose to the child's thyroid was estimated as between 700 and 1400 rads from radioiodines and an additional 175 rads from gamma radiation for the group exposed to 175 rads. No nodules were detected in the unexposed children.

Two cases of *cancer* have developed in the exposed group. The first appeared at 5 years post exposure, too soon, it is believed, to have been radiation induced. The second occurred at 8 years post exposure.

Leukemia surveys including physical findings, studies of leukocyte counts and morphology, alkaline phosphatase staining, and basophil counts of 4000 white cells showed no evidence of leukemia or leukemic tendency. One child in the irradiated group has had slightly elevated basophil counts 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 examinations in the first-generation children of exposed parents, with the possible exception of suggestive evidence of increased miscarriages and stillbirths in the exposed women.

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 exposed on Rongelap 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 itching and burning sensations. 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 on the dorsum of the feet, continued to show lack of repigmentation with varying degrees of scarring and atrophy of the skin. At 10 years these conditions were still evident in a number of cases. During the past several years an increased number of pigmented maculae and moles have been noted in previously irradiated areas of the skin, but these have appeared to be quite benign.

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 6 months. No further evidence of epilation has been seen.

An interesting observation noted during the first few months after exposure was the development of bluish-brown pigmentation of the semilunar areas of the fingernails and toenails in about 90% of the people exposed on Rongelap. By 6 months this pigmentation had disappeared, having grown out with the nail. 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. At that time 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; even by 6 months activity in the urine was barely detectable.

In 1958 analyses of bone samples on one of the men who died showed a body burden of 3.7 nCi ^{90}Sr . Beginning in 1957, gamma spectroscopy by use of a low-level counting chamber was added to the techniques of radiochemical analysis. 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. During the 4 years after the original contaminating event, additional weapons tests held in the area contributed slightly to the fission products in the environment. Since the diet includes a variety of imported foods, the people may have been delayed in reaching equilibrium with the environmental fission products.

Body burdens of gamma-emitting fission products (such as ^{137}Cs and ^{65}Zn) were measured in a whole-body counter and checked by radiochemical analysis of urine specimens. The levels of internal contamination per unit weight appeared to be about the same for juveniles as for adults, male and female. Wide variations in levels of contamination were found, apparently due to differences in diet, metabolism, and age.

Body burdens of ^{90}Sr 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 ^{137}Cs , ^{65}Zn , and ^{90}Sr body burdens was to be expected when the people returned there in 1957. The ^{137}Cs body burden in 1958 was about 0.68 μCi , about 60 times as great as in 1957, and the urinary ^{137}Cs level rose by a factor of 140; the mean body burden for 1959 was 0.57 μCi . The mean body burden of ^{65}Zn estimated from whole-body counting data was, in 1958, after the return to Rongelap, 0.36 μCi , 8 times as high as in 1957, and 0.44 μCi in 1959. In 1961 the mean ^{137}Cs

body burden was 0.67 μCi , which is slightly higher than the mean value of a similar group obtained in 1959; it was 300 times that of the medical team, who were measured at the same time for comparison. It appeared at this time that the people were approaching equilibrium with their environment. The ^{65}Zn level dropped to 0.071 μCi in 1959. With a larger detector and a longer counting time than previously employed, it was possible to identify and quantify ^{60}Co for the first time in these people; the mean level of ^{60}Co was about 11% of the ^{65}Zn level (7.6 nCi). A small amount of residual activity was still present after the subtraction of ^{40}K and the above radionuclides from the total spectrum. The mean level of urinary excretion of ^{90}Sr was 7.2 pCi/l or 14% higher than measured in the 1959 medical survey. In 1962 the mean urinary ^{90}Sr level was 114 pCi/g Ca, giving an estimated body burden of 12.0 nCi. Analysis of bones from the deceased Rongelap woman (1962) gave an estimated body burden of 11.4 nCi. These levels represent about a 6-fold increase in ^{90}Sr over the 1958 levels. The levels of ^{90}Sr in 1962 and 1963 hovered around the 12.0-nCi level in adults and about 22 nCi in children, about 5 and 10% respectively of the maximum permissible level (for members of the population at large).

It thus appears that body burdens of ^{90}Sr have reached equilibrium with the environmental ^{90}Sr . Little or none of the present body burden of the exposed group can be considered residual from their initial exposure, since little difference has been noted between the body burdens in exposed and unexposed populations living on Rongelap Island. The possible relation of internal absorption of radioiodines initially in the fallout to the recent development of thyroid nodules will be referred to later. No other effects of such exposure have been detected.

OTHER STUDIES

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 R¹ gene frequency, and total absence of Kell and Diego factors.¹¹ These characteristics differ from those of Polynesians and Indonesians. *Haptoglobin studies* showed the frequency of the Hp¹ gene to be higher than in Euro-

pean populations thus far tested and consistent with that of populations living near the equator. The distribution of haptoglobin types showed the population to be relatively homogeneous.¹² *Transferrins* in all sera were type CC, the common European type.¹² β -Amino-iso-butyric acid urinary levels showed the Marshallese to be the highest excretors of this acid of any population thus far reported.^{13,14} 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. *Hemoglobin types* were considered normal (all had type AA₂). *Sickling tests* showed no sickling tendency in any of the people. *Glucose-6-phosphate dehydrogenase* of the red cells appeared to be normal in the Marshallese. Studies of *Gm phenotypes* showed the Marshallese to have 100% Gm^(a+) and nearly 100% Gm^(b+). There was a complete absence of Gm^x and a high frequency of Gm-like (Gm^c).¹⁵ Serum studies for the Ag system reveal that the Rongelapese compared with other world populations have a high frequency of C.deB. antiserum reactors and a low frequency of New York antiserum reactors.¹⁶ 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 homogeneity of the population and closet 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. Numerous blood cultures for chromosome analysis have been taken on past surveys. The results of these studies will be presented in this report.

Other Laboratory Studies: Serum protein levels were generally on the high side of normal; electrophoretic patterns showed the increase in proteins was largely due to an increase in the gamma globulin fraction. The reason for this is not apparent. Numerous chronic infections may be an explanation.

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.¹⁷ It will be interesting to see whether the incidence of hypertension will later increase.

Serum cholesterol levels (1957, 1959) were somewhat lower in the exposed population than in the comparison or Utirik populations but were in the low normal range. No abnormally high or low readings were noted.

Serum creatinine levels (1957) were in the normal range with no abnormal levels noted.

Serum vitamin B₁₂ concentrations (1958, 1959) were generally significantly higher than American levels. The possibility of contamination of the samples with bacteria producing vitamin B₁₂ must be considered, since myeloproliferative and liver diseases were not seen.

Folic acid levels were found to be somewhat low in the Rongelap population and probably reflected low dietary folic acid.

Serum protein-bound iodine levels have repeatedly been shown to be generally slightly elevated. Evidence for thyroid dysfunction has only recently been noted in the people along with the recent development of thyroid nodules. Thyroid studies will be reviewed in detail in a later section.

Glucosuria and elevated blood sugar were found in a number of Rongelap people. A relatively high incidence of diabetes is prevalent in the Marshallese people.

A survey for *intestinal parasites* (1958) showed 75% of the people to be infected with various types.¹⁸ For the three major pathogens found, the over-all infection rates were, for *Entamoeba histolytica*, 18.2%; for hookworm, 5.5%, and for *Trichuris trichiura*, 34.3%.

Eosinophilia (>5%) has consistently been noted in about half the people. The fact that half the cases with eosinophilia showed no helminthic infections at all suggests that other factors besides parasitic infections must be responsible. The eosinophilia may be related to chronic fungus and other infections, particularly of the skin.

Complement fixation studies for parainfluenza 1, 2, and 3, respiratory syncytial, 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.

Immuno-electrophoretic analysis showed neither a paraproteinemia nor a typical picture of antibody-deficiency-syndrome, but a high frequency of increases of some of the immunoglobulins was noted.

Blood volume studies with ^{51}Cr -labeled sodium chromate showed a significant reduction in red cell mass and/or plasma volume. However, there is some indication that Americans living in the Islands for more than 1 year may also have slightly lowered values. Tritiated water has been used to establish the relationship of blood volume to lean body mass. Further studies on blood volume determinations are presented later.

OTHER BACKGROUND INFORMATION

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 imposes a serious difficulty in interpretation and evaluation of the medical data. Trust Territory officials are attempting to improve registration of 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 creates certain difficulties in interpreting some of the studies to be outlined.

4. The conditions of field examinations naturally limit the procedures and methods that can be used.

COMPARISON POPULATIONS

During the first 2 years, two separate groups of Marshallese people were used for comparison, each comparable in size with the exposed Rongelap group and matched for age and sex. However, both groups were found to be unstable, with a large attrition rate over the 2 years, which made them 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. Table 2 shows the various Marshallese populations that have been examined since 1954.

Table 2

Marshallese Populations Examined Since 1954

Group	Original number in group	Number living (1966)	Frequency of exams	Subject Nos.
<u>Exposed</u>				
Rongelap*	67	58	Annual	1- 86
Ailingnae**	19	15	Annual	1- 86
Utirik	157	133	3-4 years	2101-2237
<u>Unexposed</u>				
Rongelap	99	168†	Annual since 1957	801-1073
Rita	57		1955-1956	1000-1082
Majuro	115		1954 only	700- 800
<u>Children Conceived After the Fallout</u>				
Of exposed parent(s)	63††	60	Annual	87- 151
Of unexposed parents		104	Annual	801-1073

*Includes 3 *in utero* children.

**Includes 1 *in utero* child.

†Individuals have been added since 1957 when this group was first available.

††Live births.



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Figure 3. Medical survey team, 1965 (upper picture) and 1966.

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Organization

THE 1965 SURVEY

The survey team consisted of 12 physicians and technicians from the United States and 6 from the Trust Territory (Figure 3). Examinations were carried out on 71 Rongelap people in the exposed group, 48 children of exposed parent(s), and 191 unexposed Rongelap people (adults and children) comprising the comparison population. A Trust Territory cargo ship was used to transport part of the team and equipment between Rongelap and Kwajalein. In addition, air support was furnished by the Search and Rescue Detachment, U.S. Navy, stationed at Kwajalein. The team lived ashore at Rongelap Island during the period of the examination. Two new air-conditioned trailers were recently installed on Rongelap for examination purposes (see Figure 4). Smaller groups of Rongelap people were also examined at Ebeye (Kwajalein Atoll) and Majuro, with local hospital facilities used for the examinations.

THE 1966 SURVEY

The 12-year medical survey was of a more limited scope than the previous one, with only the exposed groups being examined. A total of 177 examinations were carried out on 72 exposed Rongelap people and 105 exposed Utirik people. Personnel and equipment were transported between islands by a ship operated by a Marshall Island trading company.

Prior to each survey, meetings were held with the people to explain the survey procedures and particularly to discuss why the thyroid cases need hospitalization, surgery, and treatment. They seem to have accepted the situation in regard to the thyroid lesions calmly, and there has been no expression of great concern.

In February 1966, the exposed people of Rongelap received compassionate payment for effects of fallout radiation. Each person received nearly \$11,000 and some an additional amount as inheritance from those exposed people that had died. They were quite happy about this, and on our ar-



Figure 4. Newly installed air-conditioned trailer used for examinations at Rongelap.

rival in March it was gratifying that most of the people had put their money into savings accounts. The Utirik people seemed quite disappointed in not receiving any monetary compensation for their fallout exposure. In a lengthy meeting held with them on arrival of the team, the reason for this – the lack of radiation effects – was carefully explained, and it is believed they accepted the situation in good grace, judging by their cooperation in the examinations and cordiality to the medical team.

Findings

INTERVAL MEDICAL HISTORY

Table 3 shows the populations examined in 1965 and 1966. Table 4 shows the location of the populations under study, and Table 5 shows the percent distribution of the Rongelap population by age and sex.

Illnesses

During the past 2 years the Rongelap people have been generally in good health, and their nutritional status appears to have been satisfactory. An epidemic, believed to have been Asiatic influenza, with acute upper respiratory manifestations occurred in about half the population in the spring of 1964. Another influenza-like epidemic of similar proportions occurred in 1965. An outbreak of diarrhea, cause unknown, involved a large segment of the population during January and February 1965. About 15 cases of chicken pox occurred in the spring of 1965. On Ebeye Island, where about 100 Rongelap people live, an outbreak of conjunctivitis occurred during the year, but this responded to topical antibiotic treatment. Four children with varying degrees of paralysis, following a poliomyelitis epidemic in 1963, were treated and fitted with braces at Majuro. Surgery was performed on four cataract cases and one fistula *in ano*. There were 3 deaths in the exposed group and 3 in the unexposed people (see below). The most outstanding finding since the previous report has been the increasing number of cases of thyroid nodules. By July of 1966, 11 cases had been operated upon, the first 3 in 1964 at the Naval Hospital in Guam. The last 8 cases were brought to the Medical Research Center at Brookhaven National Laboratory for extensive studies



9-1048-65
Figure 5. Rongelap girl with toys following annual party, 1966.

Table 3

Numbers of People Examined
During the 11- and 12-Year Surveys

	1965	1966
Rongelap and Ailingnae exposed – adults (age >19)	48	49
Rongelap and Ailingnae exposed – children (age 11 or 12 to 19)	23	23
Rongelap unexposed – adults (age >19)	83	–
Rongelap unexposed – children (age <19)	108	–
Children born to exposed parent(s) (age <11)	48	–
Utirik exposed – adults (age >19)	–	70
Utirik exposed – children (age 12 to 19)	–	35

Table 4

Present Known Location of Rongelap and Utirik People Under Study

	Rongelap and Ailingnae exposed		Children of exposed parent(s)	Rongelap unexposed		Utirik exposed		Total
	Adults (age >19)	Children (age 12-19)		Adults (age >19)	Children (age <19)	Adults (age >19)	Children (age 12-19)	
Majuro	7	4	5	12	12	9	9	58
Ebeye	20	11	21	35	41	15	8	151
Rongelap	22	5	34	57	75	0	0	193
Utirik	0	0	0	0	0	56	26	82
Other atolls	3	1	0	11	8	5	5	33
Total	52	21*	60	115	136	85	48	517

*Two of the 23 children moved up to the adult group.

Table 5

Percent Distribution by Age and Sex of Rongelap Populations Under Study, 1966

Age group	Rongelap and Ailingnae exposed				Unexposed			
	Males		Females		Males		Females	
	No.	%	No.	%	No.	%	No.	%
> 12-20	15	20.6	14	19.2	20	12.5	24	15.0
21-30	3	4.1	9	12.3	13	8.1	12	7.5
31-40	5	6.8	4	5.5	16	10.0	15	9.4
41-50	4	5.5	6	8.2	12	7.5	8	5.0
51-60	5	6.8	1	1.4	7	4.4	9	5.6
61-70	2	2.7	3	4.1	10	6.3	5	3.1
> 70	1	1.4	1	1.4	5	3.1	4	2.5
Total	35	47.9	38	52.1	83	51.9	77	48.1
	Children of exposed parent(s)				Children of unexposed parents			
<12	28	21.1*	32	24.1*	61	23.1*	43	16.3*

*Percent in total population: $\frac{28 \times 100}{35 + 38 + 28 + 32} = \frac{28}{133} \times 100 = 21.1$; etc.

and later surgery in Boston: 3 in the summer of 1965 and 5 in the summer of 1966. These cases will be discussed in detail below.

Deaths

Table 6 lists the times and causes of death in both exposed and comparison populations. The deaths subsequent to the 1964 survey were as follows.

A 77-year-old woman in the Ailingnae group (No. 43) died of pneumonia. She had had arterio-

sclerotic heart disease, asthma, and marked peripheral sclerosis with occlusion of the right radial artery, and was quite senile. A 79-year-old exposed woman in the Ailingnae group (No. 28) died in December 1965 of cardiac decompensation. She had developed ankle edema, dyspnea, cough, and anorexia prior to death. A 77-year-old exposed man, also in the Ailingnae group (No. 29), died in February 1966, probably of asthma.

In the comparison population, a 61-year-old woman (No. 893) died of acute cellulitis of the



Figure 6. Medical history being taken by a Marshallese practitioner. 9-1049-65

Table 6

Mortality

Exposed				Unexposed			
Year	Subject No.	Age & sex	Probable cause	Year	Subject No.	Age & sex	Probable cause
1956	25	46 M	Hypertensive heart disease	1958	857	65 M	Cerebral thrombosis(?)
1957	38	78 M	Coronary heart disease, diabetes	1959	854	55 F	Infection urinary tract, diabetes
*1958	31	35 M	Acute varicella	1960	933	56 M	Pneumonia secondary to influenza
1959	62	60 F	Ovarian cancer	1960	927	65 M	Pneumonia secondary to influenza
1962	30	60 F	Cancer of cervix**	1960	861	68 F	Diabetes, cancer cervix(?)
1962	46	84 M	Arteriosclerotic heart disease	1962	953	48 M	Status asthmaticus
1962	26	21 M	Brain damage following fall from tree	1962	848	41 F	Neurosyphilis(?)
1962	56	75 F	Fractured vertebrae	1963	886	54 M	Asthma(?)
1963	52	55 F	Poliomyelitis, bulbar	1964	893	61 F	Diabetes
1963	57	107 F	"Old age" (?)	1964	862	91 M	Heart disease
*1964	43	77 F	Pneumonia, heart disease	1964	894	68 F	Pneumonia
*1965	28	79 F	Heart disease				
*1966	29	77 M	Asthma, heart failure				

*Ailingnae group.

**Not confirmed by autopsy or biopsy.

Table 7
Mortality, 1954-1966, by Age as of 1954

Group	Age: <10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	>80
Rongelap (175 rads)	0/19*	1/13	0/10	0/5	2/7	2/5	1/1	2/3	1/1
Ailingnae (69 rads)	0/6	0/1	0/1	1/5	0/1	0/1	3/3	-	-
Unexposed**	0/56	0/28	0/30	2/18	3/21	3/17	2/7	0/1	1/1

*Mortality/number in group.

**This group was not examined until 1957.

neck and face complicating diabetes, and a 68-year-old woman (No. 894) died of pneumonia complicating Asiatic influenza. A 91-year-old male (No. 862) died presumably of cardiovascular disease; he had been quite senile and bedridden for the past few years.

During the past 12 years, 13 deaths have occurred in the exposed group. This represents 13.0 deaths per 1000 per annum (11.7 for the more heavily exposed group and 18.3 for the smaller Ailingnae group) compared with 8.3 per 1000 for the Marshall Islands as a whole in 1960.

In Table 7 mortalities for the exposed Rongelap and Ailingnae groups as well as the unexposed comparison population are presented according to decade of death. A χ^2 test for significance,* comparing groups of exposed and unexposed people, showed that the mortality in the Ailingnae group alone was significantly greater than in the control group. The Rongelap exposed group combined with the Ailingnae exposed also showed a significant difference compared with the unexposed. These results should be interpreted with caution since the numbers of people involved are too small for a sensitive statistical test. None of the deaths in the exposed group can be related directly to radiation exposure. The causes of death are similar to those in the unexposed population. The slightly greater mortality in the exposed Rongelap people may be related in part, at least, to the larger percentage of older people originally in this group. No cases of leukemia have appeared in the exposed population. Two earlier deaths from cancer in exposed women and the recent development of a malignant thyroid nodule, to be described below, make it necessary to keep in mind the possibility of causal relationship with radiation exposure.

*We are grateful to Mr. Keith Thompson of Brookhaven National Laboratory for carrying out the statistical analyses.

Births

Twelve babies were born to exposed parents and 13 to unexposed parents during the period between March 1964 and March 1966. The birth rate per year is calculated from the number of births per women of childbearing age (15 to 45). There were 30 such women in the exposed group (including 3 unexposed women whose spouses are exposed males) and 32 in the unexposed group (see Table 8). From these data, there appears to be no difference in fertility between the two groups. All these babies appeared normal except for the two listed below, who were both offspring of unexposed parents.

Congenital Anomalies. Two abnormal babies were noted during the 1965 survey. One was a hydrocephalic (No. 1061) and the other a mongoloid (No. 1055).

Sex Ratio. Table 9 lists the births by sex in relation to the exposure of the parents. There appears to be no radiation-induced sex ratio alteration.

Miscarriages and Stillbirths. A total of 5 miscarriages occurred over the past 2-year period, all in unexposed women, one of whom (No. 959) had 3.

As had been noted earlier, the exposed women had a somewhat greater incidence of miscarriages and stillbirths over the first 4 years post exposure (see Table 8). During 1954-58 the exposed women had 13 miscarriages of 32 total pregnancies (40.6% incidence), and the unexposed women had 8 miscarriages in 49 pregnancies (16.3% incidence) during the 4-year period 1956-60. A χ^2 test for significance* showed that total miscarriages and stillbirths were significantly greater (at the 5% level) in the exposed women compared with unexposed during the first 4 years, but there was no significant difference after this period. Since 1958 the exposed women have had 5 miscarriages in 48 pregnancies (10.4%) and the unexposed women 10 miscarriages in 62 pregnancies (16.1%).

Table 8
Births and Fetal Deaths^a by Year

Year	Women aged 15-45	Total pregnancies	Live births	Children		Miscarriages ^a	% Pregnancies terminating in miscarriage
				M	F		
<u>Exposed^b</u>							
1954 ^c	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	6	5	2	3	1	17
1960	24	10	9	5	4	1	10
1961	23	7	6	2	4	1	0
1962	24	4	4 ^e	1	3	1	25
1963	27	8	7	3	4	1	12
1964	26	6	6	1	5	0	0
1965	30	5	5	3	2	0	0
1966 ^d	30	2	2	2	0	0	0
<u>Unexposed</u>							
1956	29	9	7	6	1	2	22
1957	30	11	9	4	5	2	18
1958	30	9	8	5	3	1	11
1959	29	10	9	4	5	1	10
1960	29	10	8	5	3	2	20
1961	29	10	10	9	1	0	0
1962	30	6	5	4	1	1	17
1963	32	6	5	2	3	1	17
1964	32	12	10	8	2	2	17
1965	32	7	5	3	2	2	29
1966 ^d	32	1	0	0	0	1	100

^aIncludes stillbirths and neonatal deaths.

^bIncludes nonexposed females mated to exposed males.

^cIncludes only children conceived after March 1, 1954.

^dIncludes data only through March 1966.

^eIncludes twins.

Vital Statistics on the Utirik People

Since a new health aide was not able to locate medical records covering the past 3 years, vital statistics on the Utirik people were particularly difficult to obtain. It appeared that 9 people of this exposed group had died over the past 2 years. The causes of death could not be determined. This makes a total of 24 deaths in the 157 people during the 12-year period since exposure. No major epidemics of disease were reported. During the summer of 1964 a number of children developed diarrhea, and in about 5 cases bloody stools, fever, and vomiting were noted. No deaths occurred, and the cases improved on antibiotic treatment.

Table 9

Sex Distribution of Rongelap Children Born Since 1954
(including some stillborn)

Parents	Sex of children	
	Male	Female
Exposed male and exposed female	13	12
Unexposed male and exposed female	12	15
Exposed male and unexposed female	3	7
Exposed female and unknown male	2	2
Unexposed male and unexposed female	61	44

PHYSICAL EXAMINATIONS

Adult Examinations

The general health and nutritional status of the people of Rongelap and Utirik Atolls were satisfactory and about the same in the exposed as in the unexposed populations, with the exception of thyroid abnormalities in the exposed Rongelap population, which will be treated separately below because of the importance of this development. As noted in previous surveys, the variety and distribution of physical abnormalities (with the exception of thyroid nodules) did not appear to be significantly different in the exposed and unexposed populations. The varying incidence from year to year of such findings as cervicitis, prostatic hypertrophy, and arteriosclerosis probably to some extent reflects differences in clinical criteria of judgment among the examiners. There was a marked tendency for extensive dental caries in young adults. Special ophthalmological and slit-lamp observations for lens opacities were not carried out during the past 2 years. Physical abnormalities in the adults are listed in Table 10.



Figure 7. Part of a physical examination.

9-1050/65

Table 10

Physical Findings in Rongelap and Utirik Adults, 1965-1966

	Rongelap exposed (48 examined)		Rongelap unexposed (81 examined)		Utirik exposed (71 examined)	
	Subject Nos.	%	Subject Nos.	%	Subject Nos.	%
Anemia, anemic tendency*	12, 22, 55, 64, 70	11.0	826, 829, 841, 843, 860, 865, 947, 964	9.9	2128, 2246	2.8
Arteriosclerosis, peripheral, mild	58, 59, 78, 79, 82	11.0	856, 884, 910, 941, 948, 957, 961, 964, 970, 991	12.4	2146, 2148, 2194, 2244	5.6
Arteriosclerosis, peripheral, moderate to severe	1, 11, 16, 41, 60, 68	13.0	853, 859, 860, 878, 899, 908, 929, 947	9.9	2105, 2110, 2175, 2191, 2198, 2214	8.5
Asthma			916	1.2		
Auricular fibrillation with myocardial damage	80	2.1				
Bradycardia	27	2.1				
Cardiac enlargement**	1, 60, 76, 80	8.3	858, 859, 917, 942, 947	6.2		
Cervical erosion	63, 74	4.3			2162	1.4
Cervical lacerations	49	2.1	829	1.2	2166	1.4
Congenital defects						
Dislocation of hip	41	2.1				
Prominent head of ulna	14, 28	4.3	833, 858, 882, 915	4.9		
Bilateral shortening of 5th finger	1, 41, 75, 78	8.3	832, 836, 910, 914			
Polydactylism			938	1.2		
Dislocated wrist					2139, 2161	2.8
Flexion deformity, fingers			826, 894	2.5		
Cyst, ovarian	39	2.1	832	1.2		
Cystocele	1, 18, 71, 74	8.3	835	1.2		

*RBC: F <3.9 million; M <4.3 million. Hemoglobin: F <11.0 g; M <13.0 g. Hematocrit: F <35%; M <38%.

**By x-ray and/or physical examinations.

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Table 10 (cont'd)

Physical Findings in Rongelap and Utirik Adults, 1965-1966

	Rongelap exposed (48 examined)		Rongelap unexposed (81 examined)		Utirik exposed (71 examined)	
	Subject Nos.	%	Subject Nos.	%	Subject Nos.	%
Deafness	1	2.1	853, 884, 916, 964	4.9		
Diabetes mellitus			853, 855, 898, 915, 917, 918, 936, 991	9.9	2101, 2111	2.8
Emphysema			853	1.2		
Epilepsy			875	1.2		
Gynecomastia	55	2.1	964	1.2		
Hallux valgus	50	2.1	860	1.2		
Hernia					2112, 2152	2.8
Hypertension (>140/90)	1, 11, 60	6.4	851, 853, 856, 859, 898, 908, 947, 961, 963, 982	12.4	2120, 2146, 2169, 2175, 2186, 2194	8.5
Kyphosis, scoliosis	13, 77	4.3	860, 864, 964	3.7	2169, 2186, 2198, 2202, 2223	7.1
Lenticular opacities	13, 55, 58, 60, 68, 82	13.0	856, 858, 859, 878, 884, 897, 899, 908, 915, 929, 935, 936, 941, 947, 961, 991	19.7	2101, 2112	2.8
Leprosy, arrested	77	2.1				
Leukoplakia			918, 941	2.5		
Liver palpable	66	2.1	844, 853, 899, 936, 941	6.2	2135	1.4
Myocardial damage or insufficiency (EKG)	60	2.1	844, 851, 858, 878, 884, 917, 947, 956, 957, 969, 970	13.6		
Obesity	1, 49, 74, 78	8.3	849, 851, 853, 880, 895, 898, 908, 951, 982, 1041, 1042	13.6	2128, 2129, 2150, 2157, 2158, 2172, 2215, 2216, 2244, 2246	14.1
Osteoarthritis	13, 29, 50, 55, 60	11.0	858, 859, 860, 878, 884, 894, 896, 898, 915, 922, 928, 935, 936, 947, 961, 964	19.7	2140, 2169, 2181, 2186, 2191, 2193, 2198, 2202	11.3
Pinguecula	18, 34, 45, 60, 79	11.0	842, 843, 844, 895, 899, 917, 941, 951, 966, 991	12.4	2169	1.4
Pregnancies	12, 22, 24, 64, 72	11.0	826, 841, 865, 896	4.9	2164, 2249	2.8
Prostatic hypertrophy	7, 55, 68	8.3	853, 884, 899, 915, 948, 961, 1007	8.6	2169	1.4
Proteinuria (>100 mg)			855	1.2	2118, 2240	2.8
Pterygium	4, 7, 10, 11, 12, 34, 41, 45, 64, 66, 70, 78, 82	27.0	823, 840, 844, 851, 852, 853, 858, 859, 864, 878, 880, 884, 897, 898, 908, 918, 920, 928, 929, 947, 948, 956, 957, 961, 969, 971, 982, 1007, 1041, 1042	37.0	2103, 2104, 2110, 2112, 2114, 2135, 2137, 2140, 2148, 2161, 2162, 2166, 2181, 2186, 2191, 2193, 2198, 2200, 2206, 2211, 2212, 2214, 2220, 2221, 2240	35.2
Rheumatic heart disease	76	2.1			2128	1.4
Syphilis(?), arrested	11, 59	4.3	846, 860, 899	3.7	2181	1.4
Tumor, benign	7, 10, 37, 41, 60	11.0	844, 859, 864, 880, 897, 922, 964, 1001, 1007	11.1	2109, 2140, 2162, 2169, 2212	7.1
URI			831, 853	2.5	2109	1.4

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As part of the cancer surveys, roentgenograms of the chest on all of the population under study are scheduled every 2 to 3 years. Chest plates on some 100 people were made during the past 2 years. Only 2 cases thought to be tuberculous were noted, and these appeared to be old fibrotic inactive disease in older people. Otherwise evidence for pulmonary and cardiac diseases was minimal. Papanicolaou smears of the vaginal region were obtained on 51 females (1965).* No evidence of malignancy was noted. A rather high incidence of inflammation was noted, possibly related to poor hygiene, and 14% of these women had trichomonas infections. With the exception of a malignancy of the thyroid to be described below, no malignant

lesions were detected, and only a few benign soft-tissue tumors were found during the past 2 years.

Hospitalization for further examinations, surgery, and other treatment was recommended for a number of people. Some of the recommendations included cervical dilatation and curettage; surgical correction of rectocele, anal fistula, and deformed toe; removal of ovarian cyst, Bartholin's cyst, and cyst on foot; skin biopsy; cataract removal; hip fusion; treatment of diabetes and inflammatory disease; poliomyelitis rehabilitation; neurosurgical consultation on hydrocephalic child; examinations for possible brain tumor in epileptic; and evaluation of a case of leprosy at a sanatorium. Limited treatment was carried out by the medical team during the survey.

Examination of residual "beta burns" in adults and children revealed few changes as compared

*We are grateful to Dr. Genevieve Bader at Memorial Hospital, New York, for these analyses.

Table 11
Residual "Beta Burns"*

Subject No.	Age	Sex	Data
2	13	M	Roughening and pigment variation on front of neck. Several pigmented macules ACF.** Perianal depigmentation.
3	12	M	Mottled pigmentation both axillae. Pigmented area behind left ear.
11	61	M	Pigment changes left ACF. Pigment variation with many moles in and beneath axillae.
12	29	F	Two pigmented moles on back of neck. Small keloid at site of mole removal on right shoulder.
17	14	F	Scarring and pigmentation left ACF.
20	18	M	Pigmented patch back of neck.
23	15	M	Pigmented macules left axilla, front of neck, and chest. Depigmented spots shaft of penis.
24	24	F	Slight pigment variation on front of neck. Several pigmented macules dorsum left foot.
34	56	F	Moles on front of neck, particularly on left side.
39	26	F	Slight roughening and pigmentation back of neck. Pigment variations and slight hyperpigmentation dorsum right foot.
49	26	F	Numerous pigmented macules on both sides of neck and a few on arms and ACF.
54	12	M	Mottled pigmentation and depigmentation on front of neck.
58	71	F	Moles over front and side of neck and on left side of face.
59	46	F	Mottled pigmentation and depigmentation on back of neck.
63	47	F	Slight rugosity and pigmented ridges on back of neck.
64	41	F	Mole back of neck. Slight pigment variation and a few macules front of neck.
65	12	F	Pigment variation and roughening front of neck.
67	25	F	Depigmented scars dorsum left foot.
74	23	F	Slight roughening and pigmentation back of neck.
75	28	F	Slight pigmented area dorsum right first toe.
78	48	F	Numerous pedunculated moles on sides and front of neck.
79	50	M	Pigmented and depigmented scar on posterior surface of the left ear.

*Includes appearance of moles which may or may not be related to radiation exposure in some cases.

**ACF = antecubital fossa.

with the 10-year survey. Table 11 lists the residual skin lesions. No further increase in benign nevi seems to have taken place, and some regression in pigmented areas and scarring may be occurring. No evidence was seen of chronic degenerative changes or cancer of the skin.

Pediatric Examinations

Pediatric studies including a brief interval history, routine clinical examination, a roentgenogram of the left hand and wrist, and physical

anthropometry were carried out on exposed and unexposed children in 1965 (23 exposed, 48 born to exposed parents, 108 unexposed; 179 total). In 1966 only the 23 children in the directly exposed group were studied. Special attention was directed during these 2 years to the palpatory findings in the neck and thyroid gland areas.

The abnormal findings on physical examination of the children are listed in Table 12. One child (No. 1061) born to nonexposed parents had hydrocephalus, developmental retardation, and hepa-

Table 12

Physical Findings, Pediatric Examination
(See Table 17 for thyroid abnormalities)

	1965 Survey			1966 Survey	
	Exposed children (23)*	Unexposed children born before 1 Jan. '55 (39)	Unexposed children born after 1 Jan. '55 (69)	Children born to exposed parents (48)	Exposed children (23)
Hypertension	0/21	0/29	0/18	-	0/23
Vitiligo	1	1	-	-	-
Café-au-lait spots	2	-	3	2	-
Acute skin infection	2	3	5	2	1
Thrush	-	-	-	1	-
Umbilical hernia	-	-	1	-	-
Papilloma	1	-	-	-	-
Otitis media					
Acute	3	5	7	7	-
Chronic	-	1	1	2	-
Eczema	-	-	-	1	-
Adenopathy	-	-	4	8	3
Palpable spleen	-	1	-	-	-
Palpable liver	-	-	2	3	-
Malformation, toes (No. 112)	-	-	-	1	-
Systolic murmur	9	14	20	16	-
Keloids	1	-	-	-	-
Conjunctivitis	1	1	2	2	1
Bullous lesion, lips	1	-	-	-	-
Hypoparathyroidism (No. 21)	1	-	-	-	-
Unilateral breast development (No. 65)	1	-	-	-	-
Tonsils, hypertrophy	2	7	12	6	1
Molluscum	-	-	2	4	-
Dermatitis, nonspecific	-	-	1	-	-
Nevi	2	5	3	1	-
Polio. residual	-	5	3	1	-
Cheilosis	-	-	1	1	-
Nystagmus	-	-	1	-	-
Thoractomy scar	-	1	-	-	-
Mongolism (No. 1055)	-	-	1	-	-
Hydrocephalus (No. 1061)	-	-	1	-	-
Deformed hand (burns)	-	-	1	-	-
Cystic mass, sole of foot (No. 807)	-	-	1	-	-
Congenital heart disease (Nos. 1032, 1058)	-	-	2	-	-
Tracheostomy scar	1	-	-	-	-
Warts	2	-	-	2	-
Pilonidal sinus	-	-	-	-	1
Pelvic peritonitis	-	-	-	-	1

*Number examined.

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Table 13

Assay for Human Growth Hormone (HGH)
in Exposed Marshallese Children

Subject No.	Time after insulin, min	Blood glucose, mg/100 ml	HGH, ng/ml*
2	25	31.6	4.0
3	30	89.6	<2.0
6	25	26.9	<2.0
8	25	29.0	5.0
33	25	34.0	<2.0
54	15	67.1	<2.0
54	30	35.6	3.0
65	15	33.6	4.0
65	27	22.6	2.0
83	30	38.1	3.0

*Assayed by Dr. J. Roth, National Institutes of Health, Bethesda, Maryland. The normal fasting level is 0 to 3.0 nanograms/ml, with at least a 3- to 5-fold rise 60 min after insulin injection.

tosplenomegaly. Another child (No. 1055), also born to nonexposed parents, had diagnostic stigmata of mongolism. The results of examinations of the neck and thyroid gland are summarized elsewhere in this report. With the exception of the thyroid nodules, no correlation between the development of abnormalities in children and exposure to radiation could be suggested.

In 1965 assays for human growth hormone levels were done on blood specimens obtained from 8 exposed Marshallese children. After an overnight fast, crystalline insulin was injected intravenously at a dose of 0.1 mg/kg body weight. Blood was drawn at 15 and 30 min. The existing circumstances in the field precluded the continuation of the hypoglycemic state beyond 30 min. The hormone values were determined by the method of Glick et al.¹⁹ The results are summarized in Table 13. Even though a higher level of growth hormone secretion might have occurred if hypoglycemia had been prolonged, measurable amounts of human growth hormone were found in all children tested. The 2 markedly physically retarded boys were among the 8 studied. When it is noted that TSH secretion is also adequate in these children (see section on thyroid gland), the results qualitatively document the existence of anterior pituitary gland function.

Goodenough "Draw a Man" tests²⁰ were taken by a number of children. Some of the drawings are

shown in Figure 8. Because of language barriers, such tests are difficult to carry out. Cultural, social, and educational variables also complicate the scoring. The figures are being analyzed with the hope that numerical scores may provide some measure of mental development in the growth-retarded children.

A detailed analysis of the growth and development data on the Marshallese children during the period 1958 through 1963 has been published.²¹ The analysis has been extended to include the growth measurements for 1964 and 1965. In 1966 the unexposed (control) children were not examined. The trends revealed previously have continued. Among the girls, there is no significant difference between exposed and unexposed children in either the statural or weight curves (Figures 9 and 10). There is no significant difference in body weight between exposed and unexposed boys (Figure 11). Among the boys only, the statural growth of the exposed lags below that of the unexposed subjects (Figures 12 and 13). This difference in stature is better delineated when age at exposure is considered (Figure 13). Thus, boys exposed at ages >6 years show no difference in statural growth from that of unexposed boys. Boys exposed at ages 2 years and younger show the most prominent retardation. Analysis this year indicates that boys exposed at 3 to 5 years of age are also showing some lag in statural growth. Children born to exposed parents have demonstrated no significant difference in statural and weight growth as compared with children born to unexposed parents (Figures 14 to 17).

Skeletal age assessments by the method of Greulich and Pyle²² have been plotted against chronological age in boys (Figure 18) and in girls (Figure 19). The lines represent the best fitting linear relationships by the least-squares method. While the curves for the exposed group (both boys and girls) fall to the right of the curves for the unexposed, the differences are not statistically significant. The points representing the markedly growth-retarded boys are immediately apparent from inspection. The graphs also indicate that the skeletal age assessments at given chronological ages for the Marshallese boys and girls are lower (by about 6 to 12 months) than the Greulich-Pyle standards for American children.

Since September 1965 the exposed children have been given courses of thyroid hormone.

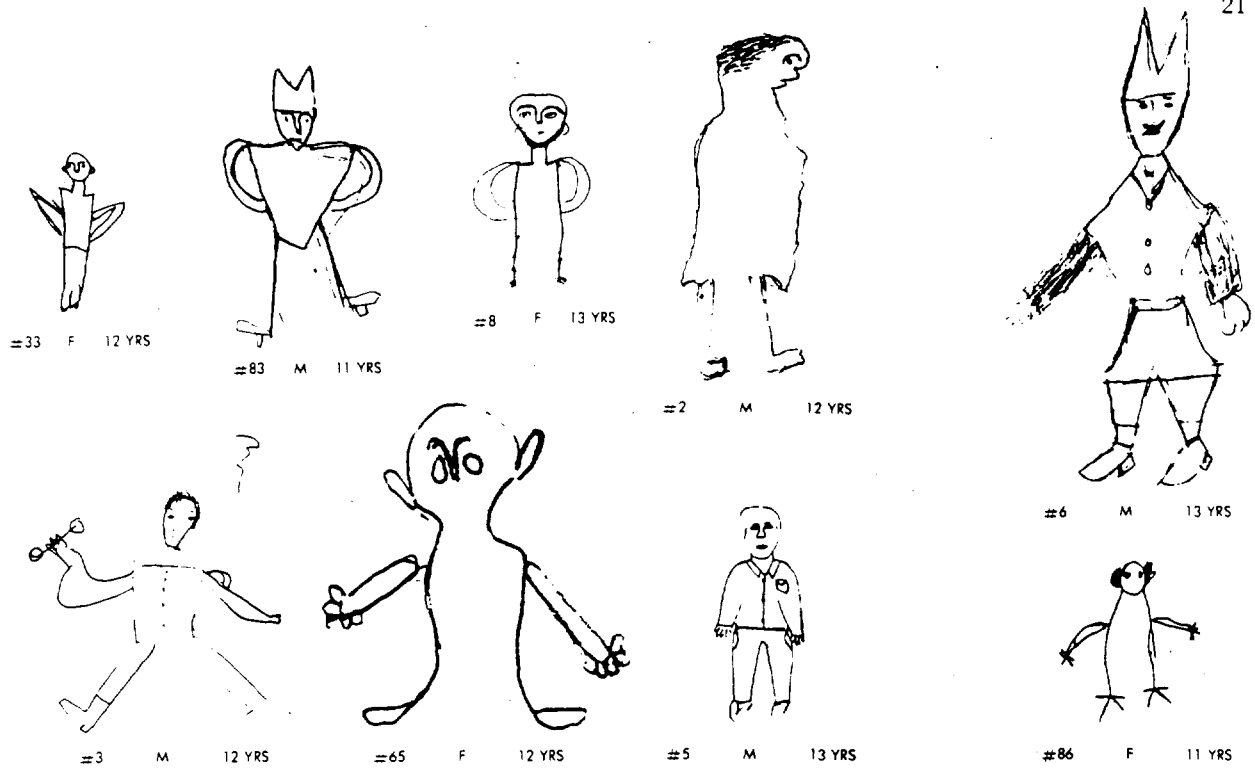


Figure 8. Drawings by Rongelap children for Goodenough "Draw a Man" test.

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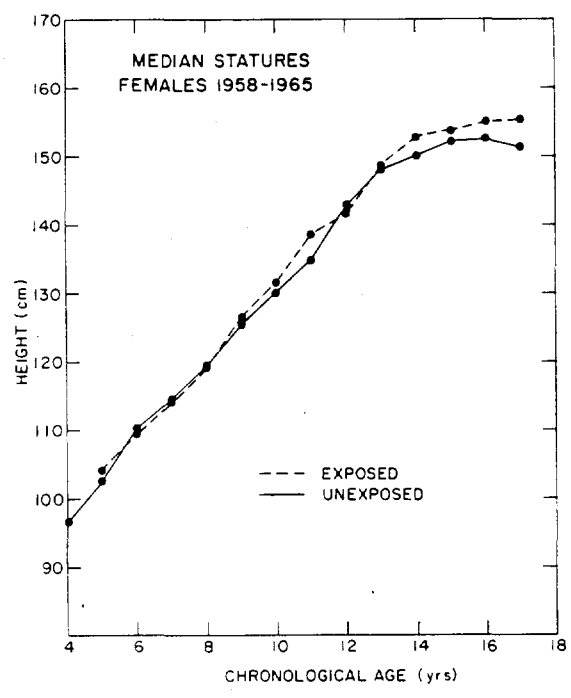


Figure 9.

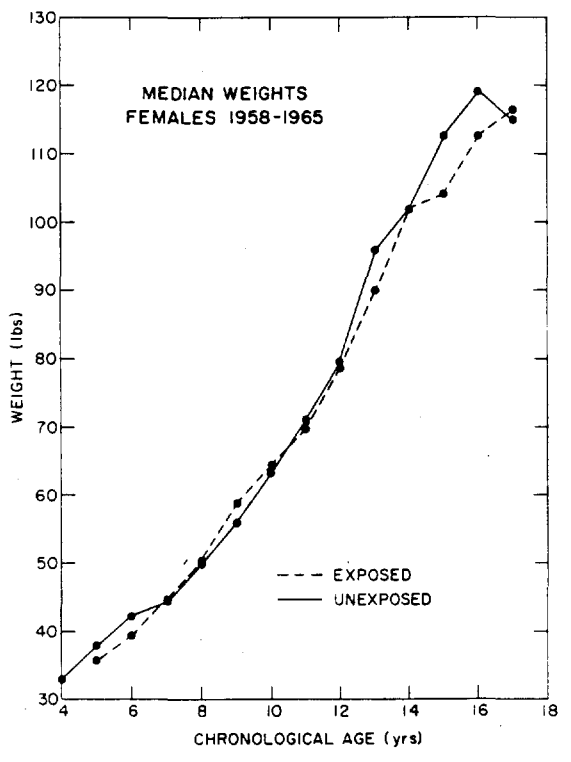


Figure 10.

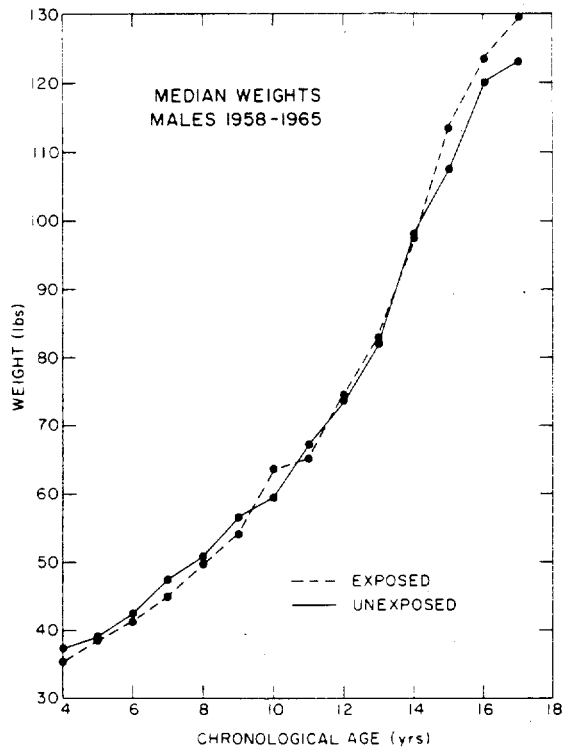


Figure 11.

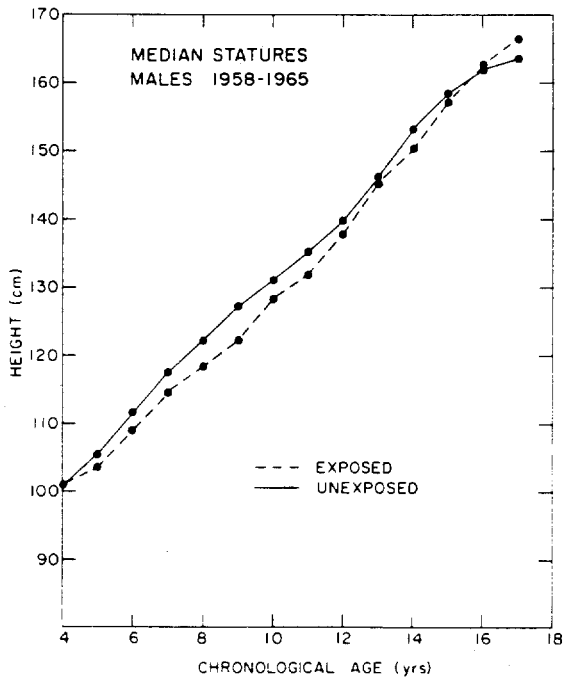


Figure 12.

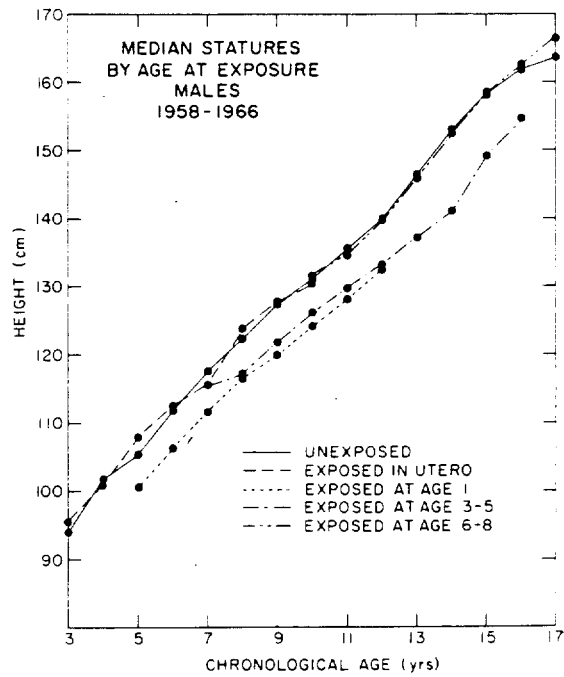


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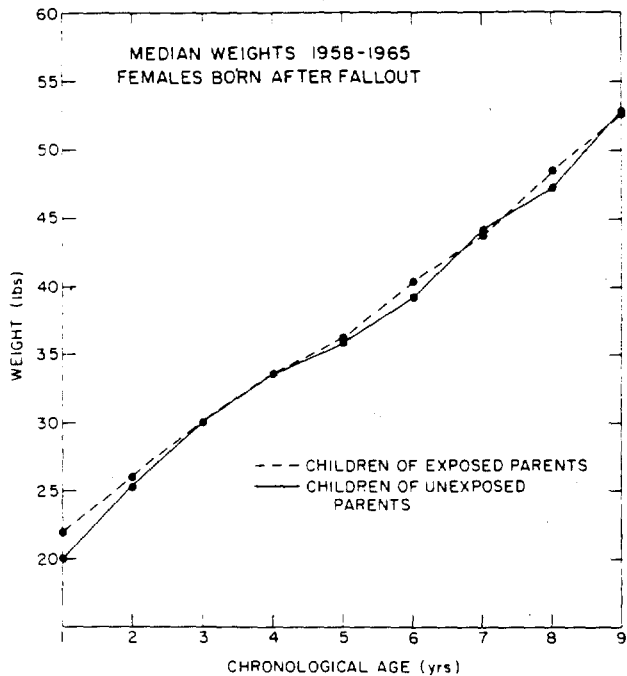


Figure 14.

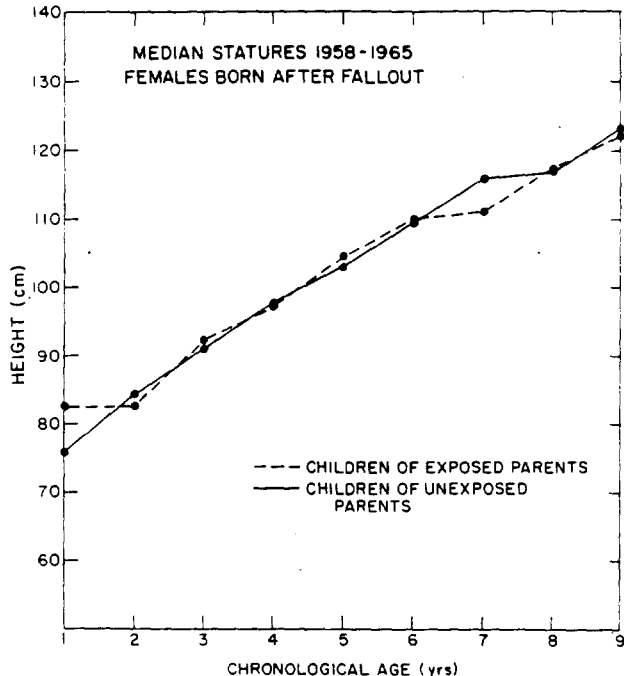


Figure 15.

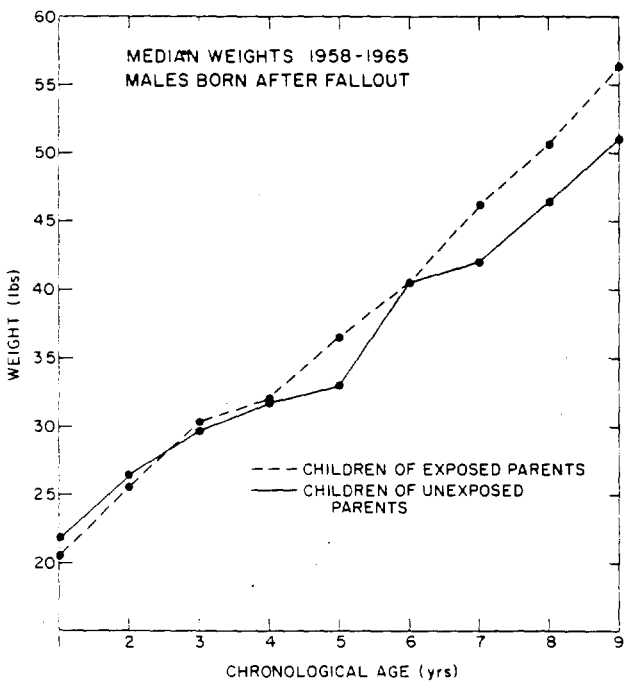


Figure 16.

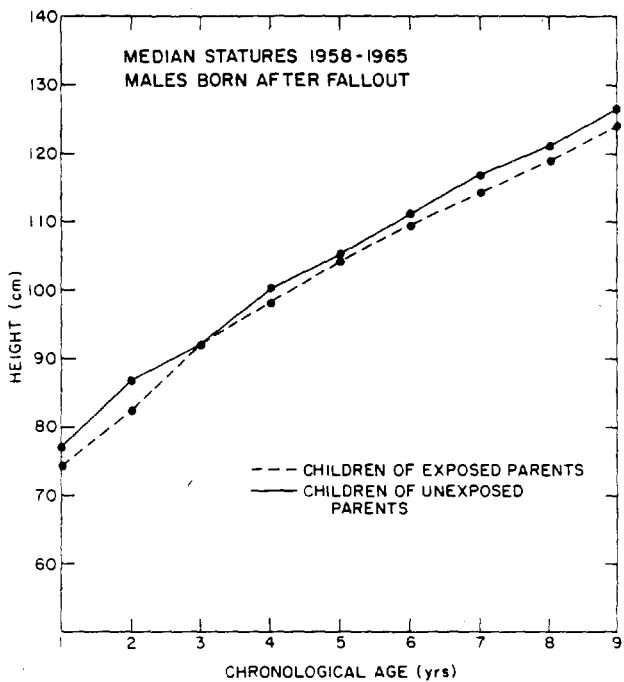


Figure 17.

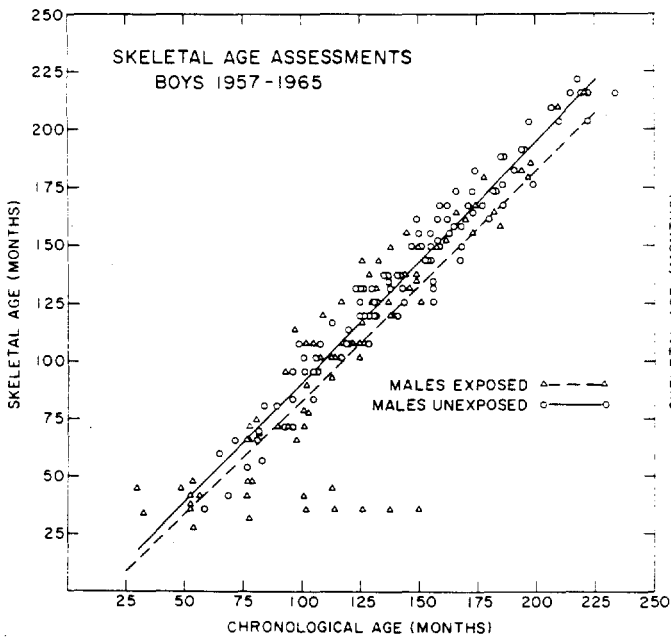


Figure 18.

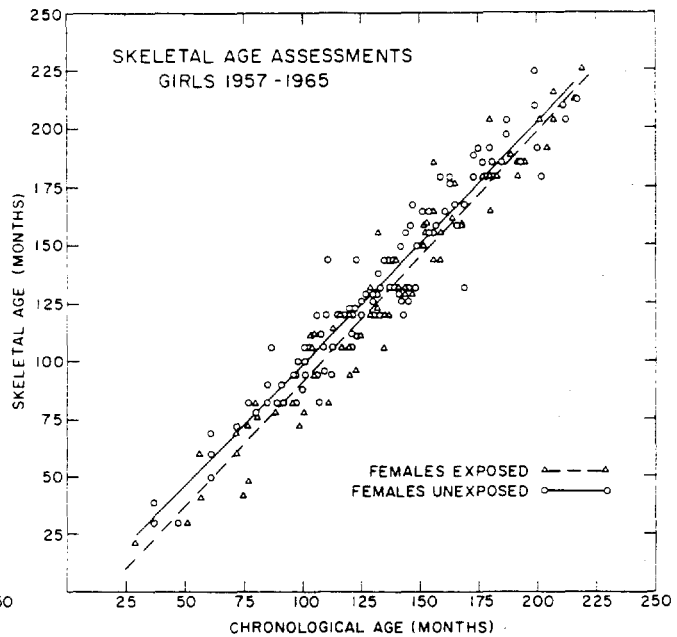


Figure 19.

THYROID FINDINGS

During the past 3 years, the development of thyroid abnormalities in a significant number of the people exposed on Rongelap, and in one from the Ailingnae group, has resulted in extensive thyroid studies and surgical intervention in some cases. The examination and therapy of the first 6 cases of nodules of the thyroid gland have been described.^{10,23,24} Since then, the number of cases of nodules and hypothyroidism has increased to 18, and the new cases are described below.

The Radiation Dose to the Thyroid Glands

The dose to the thyroid gland from radioactive iodine is determined by its uptake by the gland, its half-life in the gland, the size of the gland, and the relative proportion of the several radioisotopes of iodine involved. The relative distribution of radioiodines in fallout depends on the type of explosion but in general is well known. In addition to ¹³¹I, the isotopes ¹³³I, ¹³⁵I, and to a less extent ¹³²I contributed significantly to the thyroid dose. The only direct data available on the Rongelap people are radiochemical analyses of pooled urine samples taken 15 days and longer after the fallout. Three separate estimates have been made of the dose from radioiodines to the thyroid glands of adults

exposed on Rongelap: 150 rads (from direct measurement of urinary ¹³¹I),²⁵ 100 rads (by indirect measurements on pigs removed from Rongelap plus Marshallese urinary excretion data),²⁶ and 160 rads (based on recent recalculations of early data²⁷ - see Appendix 2). The last recalculations were based on analysis of pooled urine samples mainly from adult Rongelap people taken 15 days after the detonation; an estimate of the one-day thyroid content of ¹³¹I was 11.2 μ Ci (5.6 to 22.4 μ Ci), assuming that 0.1% (0.005 to 0.2%) of the maximum thyroid burden (not corrected for physical decay) was excreted in the urine on the 15th day. The dose of 160 rads to the adult thyroid was calculated from oral intake and inhalation of the various iodine isotopes, considering their fission yield, the average energy deposited in the thyroid gland per disintegration, and the time of absorption. The dose to the thyroid glands of children <4 years old was then calculated by means of these factors with consideration of pulmonary function and the thyroid size of a child that age.²⁷ The main source of iodine ingestion was considered to be water, and since it was being rationed at the time of the fallout, it was assumed that the children drank the same amount of water as adults and therefore had the same thyroid burden of radioiodines. The small size of the childhood thy-

roid then resulted in a substantially larger dose. The total estimated dose from the various iodine isotopes to the child's gland was about 1000 rads, with a range of 700 to 1400. The glands received an additional 175 rads from external gamma radiation. Details of these calculations have been given by James and Ng and are presented in Appendix 2. Although the skin overlying the thyroid gland was frequently the site of "beta burns" as shown in Figure 20, the deposit of radioactive materials in this area probably did not add significantly to the thyroid dose, since most of the beta irradiations were too weak to have penetrated to the depth of the gland.

Previous Thyroid Studies

Until 1963 no thyroid abnormality was detected in either the exposed or the comparison population, except for one case of asymptomatic diffuse thyroid enlargement seen in an unexposed woman. It has not been possible to perform basal metabolism rate determinations, but careful physical examination of the thyroid and a variety of tests of thyroid function have been performed during the previous surveys.

Protein-Bound Iodine.²⁸ The serum protein-bound iodine has been determined by the methods of Foss et al.²⁹ at Brookhaven National Laboratory, the Boston Medical Laboratories, and Bio-Science Laboratories, Van Nuys, California. In addition, estimation of the butanol-extractable iodine of serum was done at Bio-Science Laboratories, and also column chromatography of the serum iodine by a modification of the method of Galton and Pitt-Rivers.³⁰ In several instances the capacity of thyroxine-binding alpha globulin (TBG) was measured at NIH by a method described previously.³⁷

The results of analyses for iodine in serum are shown in Table 14. (See Appendix 3 for complete protein-bound iodine data.) It is apparent that on several occasions and with several different methods the average serum protein-bound iodine in the inhabitants of both Rongelap and Utirik is higher than normal, and that from 16 to 64% of the natives on Rongelap and 90% on Utirik show values that are above the normal range by American standards. No significant differences in the PBI levels have been noted between the group that had been exposed to radiation and the unexposed group. The first results showing an elevated PBI were obtained in 1958, and since that time

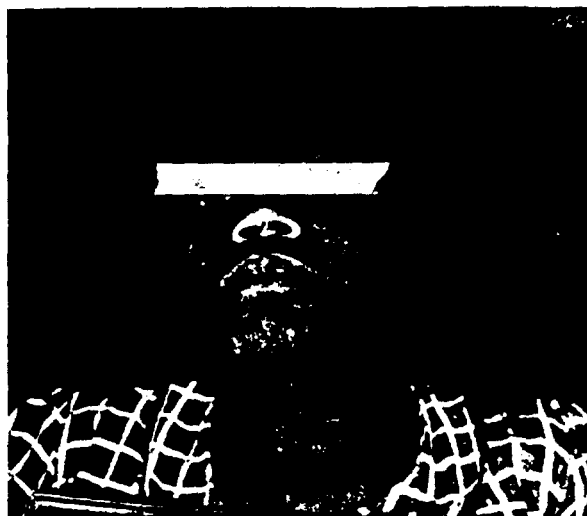


Figure 20. "Beta burns" of neck (subject No. 39, March 1954). The area over the thyroid was a frequent site of burns.

care has been taken to ensure that glassware and syringes were not contaminated with iodine. This can be seen by the fact that the total iodine is not markedly greater than the PBI and by the normal values for PBI obtained in 1964 on members of the medical team, whose blood was obtained at the same time and under the same conditions as that of the natives. The elevation in PBI could be due to a general increase in serum PBI in all the Rongelap population, or it could be due to the occurrence of some genetic difference, so that a substantial fraction of the population shows abnormally high PBIs and the remainder of the population is normal. In the first case, a plot of the level of PBI versus frequency of occurrence at that level would show a normal distribution, except that the whole curve would be displaced about 2 $\mu\text{g}\%$ upwards. In the second case, the distribution curve would be bimodal, and a family tree would show familial clustering, the precise type depending on the manner of inheritance. Figure 21, a distribution curve of PBI level versus incidence at that level, shows no evidence for a bimodal distribution. The low number of PBI values between 7.75 and 8.0 $\mu\text{g}\%$ seems to be due to statistical fluctuation because of the small numbers of cases. Furthermore, the elevated values (defined as those above 8.0 $\mu\text{g}\%$) did not show a familial pattern of distribution. It appears, therefore, that the eleva-

Table 14

Serum Iodine

Serum Protein-Bound Iodine					
Date	Group	Average. $\mu\text{g}\%$	Range. $\mu\text{g}\%$	No. samples	Percent over 8 $\mu\text{g}\%$
1959	Rongelap	6.2	4.1 - 9.2	12	16
1962	Rongelap	8.6	4.6 - 12.0	14	64
1963	Rongelap	8.1	1.9 - 12.0	29	66
1964	Medical team	4.9	2.5 - 6.9	10	-
1964	Rangelap	7.1	2.0 - 10.2	11	36
1965	Rongelap exposed	7.6	4.1 - 11.9	31	42
1965	Rongelap unexposed	7.0	3.9 - 10.7	19	28
1966	Rongelap exposed	6.1	3.1 - 11.8	19	16
1966	Utirik exposed	11.8	6.9 - 28.7	25	92
Serum Butanol-Extractable Iodine					
1959	Rongelap	4.9	2.7 - 8.7	12	
Serum Iodine Chromatography					
	Group	Total iodine. Av $\mu\text{g}\%$	Iodoprotein. Av $\mu\text{g}\%$	$T_4 + T_3$. Av $\mu\text{g}\%$	No. samples
1959-1964	Rongelap	6.98	2.22	4.53	19
1965-1966	Rongelap	7.40	2.80	4.21	18
1966	Utirik	18.10*	9.20	4.00	5
1964-1966	Americans	5.09	0.80	3.76	25

*One Utirik sample was quite high in total iodine (32.0 $\mu\text{g}\%$) suggesting contamination of this sample. The remaining 4 samples showed total iodine levels very nearly equal to the combined iodoprotein and thyroxine values.

tion in PBI is a general phenomenon which affects the entire population.

The difference between PBI and BEI in 12 cases was 1.5 $\mu\text{g}\%$, which is somewhat greater than an average value of 0.6 $\mu\text{g}\%$ in the United States.^{31,32} This was suggestive evidence for the presence of iodoprotein in serum, and the results obtained by column chromatography substantiate this, as an average iodoprotein level of 2.22 $\mu\text{g}\%$ was found. The average value for the amount of thyroxine iodine plus triiodothyronine iodine in these sera was 4.53 $\mu\text{g}\%$. Five plasma samples from Utirik people assayed in 1966 showed an unusually high average of 9.2 $\mu\text{g}\%$ of iodoprotein. (See Table 14.) These data may be compared with results obtained on 25 normal North American controls residing temporarily in Washington, D.C., who showed an average serum iodoprotein level of 0.8 $\mu\text{g}\%$ and an average $T_4 + T_3$ iodine level of 3.76 $\mu\text{g}\%$.

Dietary Iodine and Urinary Excretion of Iodine.

The supply of iodine in the Marshallese diet has generally been considered adequate in view of the

large consumption of seafood. Analysis of 24-hr urine samples from 28 people at the Boston Medical Laboratory in 1965 gave an average value of 105 μg iodine per day (range: 19.5 to 279), which is within the range of values found in an unselected population living in the Eastern United States.³³ A complete table of urinary iodine excretion data is presented in Appendix 4.

Thyroid Uptake of Radioiodine. Uptake of radioiodine by the thyroid was tested in 1965 on 21 Marshallese (12 exposed and 9 comparison adults) with no known thyroid abnormalities.

Studies with ^{132}I were performed with a collimated 1-in. NaI crystal 25 cm from the patient's neck. The ^{132}I was milked daily from tellurium-132 bound to a resin by elution with 0.1 M NH_4OH , and it was calibrated against a ^{137}Cs standard. The ^{132}I (5 to 10 μCi) was administered by mouth before breakfast, and counts were obtained over the neck at $\approx 1/2$, 1, 2, 3, and 4 hr; a single 3-hr urine was assayed for ^{132}I . A predose count of the neck was performed, since a small amount of what was presumed to be ^{137}Cs in-

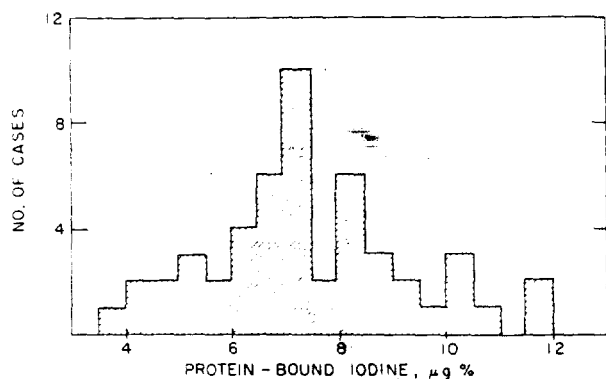


Figure 21.

creased the background slightly. Mathematical analysis* of these data was done on an IBM 7094 computer with the program of Berman et al.³⁴ No experimental correction was made for extrathyroidal radioactivity "seen" by the counter, since the computer program adjusted the readings over the neck for this factor. A least-squares best fit, assuming exponential thyroid uptake and renal excretion of iodide, produced a "best" value for this factor, termed σ_{31} .

The results of the studies with ^{132}I are shown in Table 15, where they are compared with values obtained from normal individuals residing in the United States. It can be seen that the rate of thyroid uptake and the rate of urinary excretion are both decreased. Since they are decreased, more or less proportionately, the calculated asymptotic uptake is normal or slightly elevated. One may use these data plus the urine iodide values to calculate the average daily secretion of thyroid hormone, assuming steady state conditions, using the formula

$$S = \frac{EU}{1 - U},$$

where

S = amount of iodine secreted by the thyroid ($\mu\text{g}/\text{day}$),

U = fractional thyroid uptake of iodine, and

E = urinary iodine ($\mu\text{g}/\text{day}$).

With $E = 105 \mu\text{g}/\text{day}$ and $U = 0.42$, the value for S is calculated to be $76 \mu\text{g}$ iodine/day. This value is

*We are indebted to Dr. Mones Berman, National Institutes of Health, Bethesda, for this analysis.

Table 15

Kinetic Studies With ^{132}I

Group	λ_{21} *	λ_{81} **	Theoretical uptake	σ_{31} †	No. cases
Marshallese	0.72	0.97	42%	0.08	21
U.S. normals	1.0	2.0	33%	-	-

*Fraction of extrathyroidal iodide transferred to the thyroid per day.

**Fraction of extrathyroidal iodide excreted in the urine per day.

†Value derived by the computer for the fraction of extrathyroidal iodide "seen" by the counter.

somewhat higher than similar ones calculated for other groups but is not extraordinarily high.³⁵⁻³⁷

Serum Cholesterol. Serum cholesterol determinations on most of the exposed and an equal number of the unexposed population were carried out in 1957 and 1959. The results are tabulated in Appendix 5. Although the cholesterol levels in the exposed group are about 17% below the levels in the unexposed group, the difference is not quite significant at the 5% level. Individual values were not indicative of thyroid disease.

Discussion of Previous Thyroid Studies. Until the recent development of hypothyroidism in two boys, it had been the consensus of all physicians who examined these people that they were euthyroid. A conceivable explanation for the high PBI could be an elevation of thyroxine-binding proteins in serum which, as in the congenital elevation of thyroxine-binding globulin described by Beierwaltes and Robbins,³⁸ causes an increase in the serum PBI without hyperthyroidism. The levels of the TBG in the Marshallese serum measured by Robbins, however, were within normal limits. The discrepancy between PBI and BEI suggested the presence of an iodoprotein in serum. The chromatography of serum iodine showing an iodoprotein level in the Rongelap people of $2.2 \mu\text{g}\%$ (and higher in the Utirik people) seems to implicate the iodoprotein as the cause for the elevated PBI.

No adequate data are available on the calorogenic potency of serum iodoproteins, but there are some results which show that most of the iodinated amino acids in this protein are monoiodotyrosine and diiodotyrosine.^{39,40} These iodoamino acids are

devoid of physiological activity. Hence an iodoprotein containing only these iodoamino acids is likely to be also physiologically inactive. The reason these individuals have such an iodoprotein in the blood is not clear. The data on normal controls from the Eastern United States, who showed 0.80 $\mu\text{g}\%$ iodoprotein iodine in their serum, suggest that it is a normal, albeit minor, constituent. The method of chromatography employed is such that well under 5% (or 0.2 $\mu\text{g}\%$) of serum thyroxine iodine appears in the unretarded or iodoprotein fraction. Therefore, the finding of iodoprotein does not appear to be a methodologic artifact.

More recently, with the development of thyroid abnormalities in the exposed Marshallese (to be described), it was possible to examine serum iodoprotein levels in cases with thyroid hypofunction. These data are presented in Table 16. It seems likely that the source of the iodoprotein was largely extrathyroidal, since the levels of iodoprotein were

near the normally high Marshallese values (a) in cases which had been on suppressive thyroxine therapy, (b) in cases with atrophic glands due to radiation (subjects No. 3 and No. 5), and (c) in thyroidectomized cases. The source of the iodoprotein is not known. The previously reported finding of high plasma proteins,⁵ particularly gamma globulins, in the Marshallese is of interest but may be an unrelated phenomenon. It will be important to see in future studies whether the iodoprotein can be labeled with radioiodine.

The data on urine iodine show values in the normal range. In general, it had been expected that individuals living close to the sea and eating seafood and fish would show relatively higher iodine intake. The inhabitants of the Marshall Islands have fish as one of their main sources of animal protein. Furthermore, these people are constantly exposed to sea spray, since the island at its widest is about $\frac{1}{4}$ mile across, and its highest point is ≈ 20 ft above high tide.

The data on urinary iodine were used with the results obtained with ^{132}I studies to calculate the amount of iodine secreted daily by the thyroid. The value 76 $\mu\text{g}/\text{day}$ is somewhat higher than the 57 $\mu\text{g}/\text{day}$ found by Stanbury et al.³⁵ or 58 $\mu\text{g}/\text{day}$ found by Freinkel and Ingbar³⁶ but closer to the value 70 $\mu\text{g}/\text{day}$ proposed by Riggs.³⁷ Unfortunately, nothing is known about the rate of turnover of the serum iodoprotein. If it has roughly the same rate of degradation and the same volume of distribution as thyroxine and it is assumed to have its origin in the thyroid gland, then one would expect the Marshallese thyroids to secrete organic iodine proportional to the level of organic iodine in their serum. Addition of iodothyroxine values to iodoprotein iodine levels for both Marshallese and Americans and multiplication of the ratio by the best value for iodine secreted by normal Americans results in

$$\frac{2.22 + 4.53}{0.80 + 3.76} \times 58 = 86 \mu\text{g}/\text{day}.$$

This agrees fairly well with the 76 $\mu\text{g}/\text{day}$ calculated independently from urine and radioiodine studies and is compatible with the clinical picture of a euthyroid status in spite of an elevated PBI and elevated thyroid iodine secretion rate. However, as noted above, it seems somewhat more likely that the serum iodoprotein is not of thyroidal origin.

Table 16

Serum Iodoprotein Levels in Relation to Thyroid Function				
Subject No.	Total iodine, $\mu\text{g}\%$	PBI, $\mu\text{g}\%$	T_4 iodine, $\mu\text{g}\%$	Iodoprotein iodine,* $\mu\text{g}\%$
<u>Hypothyroid</u>				
3		3.2	1.0	(2.2)
5		3.1	1.8	(1.3)
65		3.1	1.9	(1.2)
<u>Thyroidectomized**</u>				
17		1.8	<0.5	(>1.3)
21		1.3	<0.5	(>0.8)
64		5.0	2.0	2.9
69		5.7	1.7	(4.0)
<u>L-Thyroxine Treated†</u>				
34	10.8 [9.1]		6.5 [4.6]	3.2 [4.5]
59	8.6 [8.2]		4.8 [5.3]	3.8 [2.9]
68	11.8 [7.1]		5.8 [4.6]	6.0 [2.5]

*Iodoprotein levels in parentheses represent the difference between PBI and T_4 levels. The others were measured directly by the column method.

**L-Thyroxine stopped several weeks before sampling.

†Treated with L-thyroxine, 0.2 mg per day, for 6 months. Values in brackets are determination made prior to starting thyroxine treatment (1965).

The depressed thyroidal iodine uptake rate and renal excretion rate are puzzling, and no explanation for them is available at this time.

Development of Thyroid Abnormalities

During the past 3 years, beginning at 9 years after exposure, a total of 18 cases of abnormalities of the thyroid gland have been detected. Nodules of the thyroid gland were found in 16 cases, and 2

cases have hypothyroidism with no nodules. All occurred in the more heavily exposed Rongelap people except for one woman in the less exposed (Ailingnae) group. A thyroid nodule was first noted in 1963 in a 12-year-old girl in the exposed group, and in 1964 two additional cases with nodules were found in exposed girls 13 and 14 years of age.^{10,23} In March 1965 three additional cases in exposed people were noted in boys

Table 17

Thyroid Abnormalities in Exposed Rongelap People, 1966

Subject No. and sex	Present age, yr	Age at exposure, yr	Year	Age, yr	Findings
3 M	13	1	1965	12	Hypothyroid, PBI < 2 µg% March 1965; retardation of growth preceded these findings by a number of years. 3/66 growth spurt and improved appearance on thyroxine.
5 M	13	1	1965	12	Hypothyroid, PBI < 2 µg% March 1965; retardation of growth preceded these findings by a number of years. 3/66 growth spurt and improved appearance on thyroxine.
17 F	15	3	1963	12	Adenomatous goiter; total thyroidectomy, 1964. No recurrence.
21 F	15	3	1964	13	Adenomatous goiter; total thyroidectomy, parathyroidectomy, 1964. No recurrence.
69 F	16	4	1964	14	Adenomatous goiter, partial thyroidectomy, 1964. No recurrence.
2 M	13	1	1965	12	Adenomatous goiter, partial thyroidectomy, 1965. No recurrence.
20 M	19	7	1965	18	Adenomatous goiter, partial thyroidectomy, 1965. No recurrence.
64 F	42	30	1965	41	Mixed papillary and follicular carcinoma, total thyroidectomy-surgical and therapeutic radioiodine, 1965. No recurrence.
72 F	18	6	1965	17	Three-mm nodule left lobe. 9/66 nodule not palpable.
42 F	15	3	1965	14	Two-mm nodule right lower lobe. 3/66 nodular enlargement (~1½ × normal) entire gland; firm 5-mm nodule right lobe. 7/66 subtotal thyroidectomy: adenomatous goiter.
61 F	20	8	1965	19	Six to 8-mm smooth nodule left lower pole. 3/66 1-cm nodule left lobe. 7/66 subtotal thyroidectomy: adenomatous goiter.
40 M	41	29	1965	40	Two-mm nodule right lower pole. 3/66 no nodules detected.
59* F	46	34	1965	45	Five-mm nodule midline. 3/66 same. 7/66 subtotal thyroidectomy: adenomatous goiter.
54 M	13	1	1966	13	Nodular enlargement (~1½ × normal) left lobe and isthmus with 2-mm firm nodule.
19 M	17	5	1966	17	Multinodular soft enlargement entire gland (~1½ × normal). 1-cm nodule right lower pole.
36 M	19	7	1966	19	Slight nodular enlargement, entire gland. 1-cm nodule, not clearly demarcated, at right lower pole. Many tiny nodules over surface of gland.
33 F	13	1	1966	13	9/65 questionable irregular gland. 3/66 definite 5-mm nodule left lobe. 7/66 subtotal thyroidectomy: adenomatous goiter, Hurthle cell adenoma.
65 F	13	1	1966	13	9/65 questionable small nodule. 3/66 5-mm nodule right lobe. 7/66 right subtotal thyroidectomy: adenomatous goiter.

*Exposed to only 69 rads whole-body radiation and presumably proportionately less thyroid dose.

Table 18

Rongelap Thyroid Abnormalities, Age Specific Incidence (1966)*

Age at exposure	No. exposed	Nodules		Hypothyroidism		Total abnormalities	
		No.	%	No.	%	No.	% of Group
1-5	13	9	69.2	2	15.4	11	84.6
6-10	6	4	66.7	-	-	4	66.7
11-15	7	0	0	-	-	0	0
16-20	5	0	0	-	-	0	0
>20	24	2	8.3	-	-	2	8.3

*In 55 living of original 64 Rongelap people in heavily exposed group. One nodule in woman in less exposed group (not included in table).

Table 19

Thyroid Abnormalities (Nodules and Hypothyroidism) in Marshallese Populations Examined 1964-1966

Island group	Age (1954) <10 yr			Age (1954) 10-19 yr			
	No. examined	Estimated thyroid dose**	% Abnormalities	No. examined	Estimated thyroid dose**	% Abnormalities	
Rongelap	19	I 700-1400 γ 175	78.9	12	I 350-600 γ 175	0.0	
Ailingnae†	6	I 275-550 γ 69	0.0	1	I 175-300 γ 69	0.0	
Utirik†	40	I 55-110 γ 14	0.0	16	I 25-55 γ 14	0.0	
Unexposed (Rongelap and Utirik)	61	-	0.0	36	-	0.0	
Island group	Age (1954) 20-40 yr			Age (1954) >40 yr*		All ages	
	No. examined	Estimated thyroid dose**	% Abnormalities	No. examined	% Abnormalities	No. examined	% Abnormalities
Rongelap	14	I 160 γ 175	14.3	10	0.0	55	30.9
Ailingnae†	4	I 54 γ 69	25.0	3	0.0	14	7.1
Utirik†	22	I 16 γ 14	4.5	21	4.8	99	2.0
Unexposed (Rongelap and Utirik)	48	-	0.0	49	6.1	194	1.5

*Dosage in this group was the same as in the 20 to 40-year group.

**I = Dose from β and γ radionuclides; γ = dose from external γ radiation.

†In estimating the thyroid doses to the Ailingnae and Utirik exposed group it was assumed that such doses were proportional to the thyroid doses of the Rongelap exposed group, based on relative whole-body gamma dose received.

13 and 18 years of age and in a 41-year-old woman, the first adult case. Two cases of hypothyroidism in growth-retarded boys were also noted at this time. In September 1965 a further examination was carried out by two of us (R.A.C. and J.E.R.), and 5 more exposed people were found with nodules varying in size from 2 to 8 mm. One of these was a 45-year-old woman in the group that received only an estimated 69 rads. The cases up to this point were recently reviewed.²⁴ In March 1966 nodules of the thyroid gland were detected in 5 additional exposed children. Table 17 gives some pertinent data on all these cases. Table 18 shows the distribution of the thyroid abnormalities (nodules and hypothyroidism) by age. In Table 19 results are presented on the various populations studied along with the estimated radiation dose to the thyroid gland. It is noteworthy that the preponderance of thyroid abnormalities have occurred in children exposed at <10 years of age and only in the more heavily exposed group (15 of 19 children, 78.9%). No cases with thyroid abnormalities were detected in the children in the lower exposure groups of the same age range (6 Ailingnae children, 40 Utirik children) or in the 61 unexposed Rongelap children. Two adults with thyroid nodules were noted in the more heavily exposed Rongelap group and one in the Ailingnae group. In the Utirik and unexposed populations a low incidence of thyroid nodules was found, and these occurred only in the older age group.

In view of the potential seriousness of the thyroid abnormalities in the Marshallese, it was decided that the exposed people should receive thyroid hormone treatment for the remainder of their lives in order to suppress TSH secretion. Such treatment might prevent further development of nodules and possibly induce regression of existing nodules. The treatment might also stimulate growth in retarded children. Accordingly, at the time of the September 1965 survey, the 55 people in the more heavily exposed group were started on L-thyroxine at a daily dose of 0.3 mg to all people <50 years of age and 0.2 mg to all people >50. The new cases with thyroid nodules were left in the Islands under hormone treatment, with the idea that, if at the time of the next survey (March 1966) the nodules had not regressed or further nodules had developed, consideration would be given to bringing them to the United States for study and possible surgery. In March 1966 it was

decided that 5 such cases should be brought to the United States. These cases had not shown reduction in nodule size, though presumably they had been on the hormone therapy for the 6-month period. Four children with thyroid nodules who were not considered to have had an adequate trial therapy with thyroid hormone were left in the Islands to be re-evaluated later. In one 40-year-old man a nodule had disappeared, presumably as a result of therapy.

Surgical Cases

Of the 16 cases with nodules, a total of 11 have had surgery. In 1964, the first 3 cases in teen-age girls were operated upon* at the U.S. Naval Hospital, Guam.^{10,23} In July 1965, 3 cases were brought to the Medical Research Center at Brookhaven National Laboratory (Figure 22) and later taken to the New England Baptist Hospital, Boston, for surgery.** In May 1966, 5 additional cases were brought to Brookhaven for further examinations (Figure 23) and were later taken to the New England Deaconess Hospital in Boston for surgery.** At Brookhaven, detailed thyroid studies were carried out, including ¹³²I and ^{99m}Tc uptake studies and scans before and after TSH administration† and measurement of basal metabolism rate, serum protein-bound iodine, serum thyroxine (*T₄*) level, and serum antithyroglobulin antibody titer. Brief hospital summary reports on these cases are presented in Appendix 6.

Gross Appearance. In all 9 children operated upon, the glands were found at surgery to be multinodular, although in some cases the nodules had appeared clinically to be solitary. The nodules varied in diameter from a few mm to several cm, in consistency from fluctuant to relatively hard, and in color from pale grey to pink or red. Cyst formation was present in many, and some had hemorrhagic areas. Figure 24 shows the gross appearance in some cases with benign nodules at surgery. In one adult (No. 59), there was a solitary nodule, and the surrounding tissue appeared normal. In the other adult (No. 64), the gland also did not show multiple nodularity, but contained two firm, yellow, malignant nodules about 1 cm in diameter (Figure 26).

*Surgery was performed by Captain C.A. Broaddus, MC, USN.

**Surgery was performed by Dr. B.P. Colcock of the Lahey Clinic.

†We are grateful to Dr. H.L. Atkins for these analyses.



Figure 22. Rongelap thyroid cases at Brookhaven (July 1965) for studies and later surgery. The two boys had benign adenomatous nodules. The woman had a thyroid malignancy removed.



Figure 23. Marshallese thyroid cases at Brookhaven (June 1966) with Dr Conard. The Marshallese man on the right is an interpreter. All these cases had benign adenomatous nodules removed.

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Figure 24. Benign thyroid nodules at surgery. Top: exposed thyroid with arrows pointing to nodules. Bottom: sectioned gland from another case. Note multinodular, cystic, and hemorrhagic nature of gland.

Microscopic Appearance. The microscopic appearance of all the benign nodular glands in the children was characteristic of adenomatous goiter and varied mainly in the degree of change. The architecture of the gland was disrupted by the nodules of widely varying sizes. Some of the nodules contained microfollicular elements with and without colloid, others were atrophic, some contained large cysts with colloid, some with hemorrhage, and still others showed extensive proliferation of the epithelial layers with marked infolding, giving an "arboreal" appearance. Figure 25 indi-

cates some of the changes characteristic of these benign nodules. In one case (No. 33), in addition to adenomatoid nodules there also was present a Hurthle's cell adenoma. In another case (No. 61), one pathologist remarked that "some observers might regard the large nodule as a follicular adenoma." In subject No. 59, a 46-year-old woman who had received less than half the radiation dose of the other cases with nodules, there was a solitary adenomatous nodule, well circumscribed, and the surrounding thyroid tissue was normal in appearance. This was in contrast to the other cases in whom the glands were almost entirely abnormal in appearance. A 41-year-old woman (No. 64) had a mixed papillary and follicular carcinoma of the thyroid with localized metastasis to a blood vessel and lymph node (Figure 26). Following complete thyroidectomy by surgery and therapeutic ^{131}I no recurrence or further metastasis has been noted. In September 1966, at Tripler General Hospital, Hawaii, thyroid uptake studies following TSH stimulation (10 units daily for 2 days), thyroid scans, and skeletal surveys for metastasis showed absence of the thyroid and no detectable metastasis.*

Thyroid Function Tests Related to Thyroid Abnormalities and Growth Retardation

In Table 20 the cases with thyroid abnormalities are listed along with the growth status of children and results of thyroid function tests. The results of kinetic analysis of ^{132}I tests are given in Table 21. Two 12-year-old boys (No. 5 and No. 3) who had been exposed at 15 and 18 months of age respectively have had the greatest retardation of growth and development. Subject No. 3 had shown no change in bone maturation since 1961 and until recently had the bone age of a 3-year old child. The bone age of No. 5 has shown continuing slow growth and in 1965 was 5½ years. Both these boys in 1965 had the height of normal 7-year old Marshallese boys. Their dwarfism was particularly evident in comparison with younger siblings who were taller than they. In 1965 it was found that in both cases the levels of protein-bound iodine had dropped below 2 $\mu\text{g}\%$. Before that time, they had levels considered to be in the normal range, and there was no reason to relate their dwarfism to hypothyroidism. With the development of the low PBIs they showed definite

*We are grateful to Major Ronald Moore, MC, USA, for carrying out these examinations.

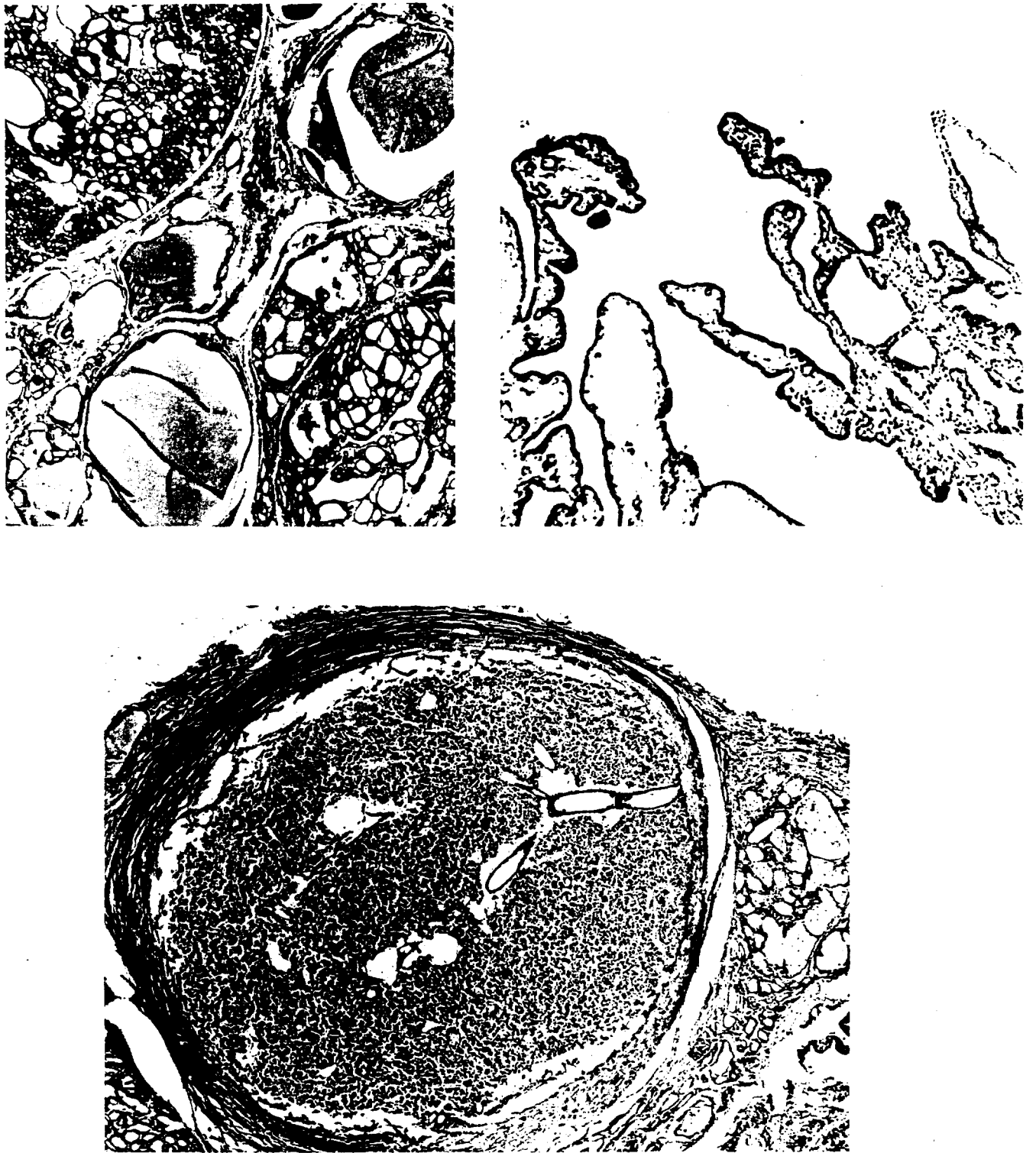


Figure 25. Microscopic sections of adenomatous nodules. Top left (21 \times): characteristic appearance of thyroid glands with wide variation in size of follicles. Some nodules consist of microfollicular tissue, others of colloid cysts; still others show hyperplasia. Top right (150 \times): hyperplasia with papillary infolding of epithelium. Bottom (25 \times): Hurthle cell adenoma (subject No. 33).

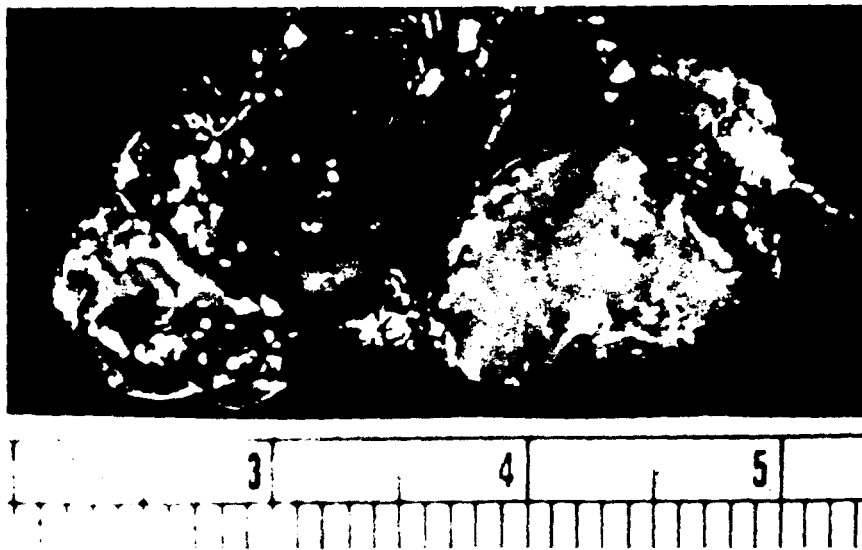


Figure 26. Top: mixed papillary and follicular carcinoma at surgery (subject No. 64). White nodule is malignant tissue.
 Bottom left (21 \times): microscopic section showing relatively normal thyroid tissue beneath the malignant tissue.
 Bottom right (10 \times): local lymph node invasion by the tumor.

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Table 20

Growth and Thyroid Studies in Subjects With Thyroid Abnormalities

Subject No. and sex	Thyroid abnormality	Year tested	Chronological age, yr	Growth		Neck ¹³² I at 4 hr, ^a % of dose		Serum iodine, µg%		Serum TSH, ^c ng/ml
				Skeletal ranking, yr	Statural ranking, ^b percentile	Pre TSH	Post TSH	PBI	T, I	
3 M	Atrophy	1965 1966	13	3	<10			1.4 3.2	0.8 1.0	>120 159
5 M	Atrophy	1965 1966	13	5½ ₂	<10		8	1.9 3.1	0.8 1.8	119 248
17 F	Nodular goiter	1965 1966 ^d	15	13½ ₂	90			6.8('64) 1.8	<0.5	372
21 F	Nodular goiter	1965 1966 ^d	15	15	10-25			8.1('64) 1.3	<0.5	440
69 F	Nodular goiter	1965 ^d 1966 ^d	16	16	>90		6.5	7.1 5.7	1.7	125
2 M	Nodular goiter	1965 1966 ^d	13	11½ ₂	25-50	(29)	(23)	7.9 5.2	4.2 2.6	9.6 26.6
20 M	Nodular goiter	1965	19	Adult	25-50	(31)	(31)	6.5	4.2	
72 F	Nodular goiter	1965	18	17½ ₂	>90			5.3		
42 F	Nodular goiter	1965 1966	15	13½ ₂	10	(15)	(24)	5.7	4.6	<17
61 F	Nodular goiter	1965 1966	20	Adult	50-75	12*(10)	(24)	7.9	4.9	
54 M	Nodular goiter	1965 1966	13	13	90	19		8.3 5.0		<3 <17
19 M	Nodular goiter	1965 1966 ^d	17	15½ ₂	10			3.9	3.3	<17
36 M	Nodular goiter	1965 1966	19	18	75			4.1 4.2	4.3	<17
33 F	Nodular goiter	1965 1966	13	13½ ₂	>90	(16)	(24)	7.0 5.9	3.8	7.3 197
65 F	Nodular goiter	1965 1966	13	10½ ₂	<10	17 (9)	(14)	4.9 3.1	2.1 1.9	6.6 23.5
59 F	Solitary nodule	1965 1966	35			7*(20)	(38)	8.2 8.6	5.3 4.8	
64 F	Carcinoma	1965 1966 ^d	31			(19)	(28)	8.6 4.9	3.4 2.0	
40 M	Solitary nodule	1965	40					10.3		

^aValues in parentheses obtained at BNL.

^bRelative statural growth expressed as percentile ranking compared with growth curves of unexposed Marshallese children.

^cAssays in 1965 could not detect levels below 3 nanograms/ml. Seven unexposed children in the same age group had <3 ng/ml. Assays in 1966 could not detect levels below 17 ng/ml. Three exposed children without thyroid disease (Nos. 6, 8, and 32) had <17 ng/ml. Two children exposed *in utero* (Nos. 84 and 86) and two unexposed children had <17 ng/ml. The 1966 levels were obtained about 3 weeks after cessation of thyroxine therapy, the 1965 levels, before therapy was instituted. We are grateful to Dr. William Odell at the National Institutes of Health for carrying out these assays.

^dPost partial thyroidectomy.

*While taking thyroxine.

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Table 21
Kinetic Analysis of ^{132}I Studies

Location and year	Subject No.	Urine, ^a per day	Thyroid, ^b per day	Thyroid fraction ^c	Iodide space, liters
Marshalls, March 1965	Exposed ^d	1.10 (0.34-2.57)	0.67 (0.33-1.27)	0.40 (0.25-0.65)	
	Unexposed ^e	0.81 (0.17-1.99)	0.79 (0.23-1.47)	0.52 (0.26-0.77)	
BNL, June 1965	2 pre TSH	1.20	1.71	0.41	
	post TSH	0.80	1.19	0.60	
	20 pre TSH	2.12	2.30	0.49	
	post TSH	1.10	1.81	0.62	
	64 pre TSH	1.88	1.08	0.36	
	post TSH	0.81	1.56	0.66	
Marshalls, March 1966	3	0.52	0.005	0.01	
	5	0.86	0.14	0.14	
	69 (partial thyroidectomy)	1.13	0.15	0.12	33.3
	2 (partial thyroidectomy)	1.53	0.92	0.37	20.0
	61 (on thyroxine)	1.26	0.58	0.31	29.0
	54	0.50	0.69	0.58	
	65	1.08	0.72	0.40	20.4
	59 (on thyroxine)	1.65	0.10	0.06	16.9
BNL, June 1966	42 pre TSH	1.87	1.29	0.41	
	post TSH	0.69	1.30	0.65	
	61 pre TSH	0.13	0.48	0.78	
	post TSH	0.50	1.67	0.77	
	33 pre TSH	0.20	0.97	0.82	
	post TSH	0.45	1.57	0.78	
	65 pre TSH	1.83	0.82	0.31	
	post TSH	2.35	1.14	0.33	
59 pre TSH	3.53	1.51	0.30		
post TSH	1.29	3.09	0.71		

^aFraction of extrathyroidal iodide excreted in the urine per day (λ_{61}).

^bFraction of extrathyroidal iodide transferred to the thyroid per day (λ_{21}).

^cTheoretical thyroid uptake, $\lambda_{21}/(\lambda_{21} + \lambda_{61})$.

^dMean and range of 12 subjects.

^eMean and range of 9 subjects.

signs of hypothyroidism: coarse facial features, dry skin, and Achilles' reflexes with typical sluggish return. There was no palpable thyroid tissue. They did not show apparent mental retardation. TSH levels (Table 20) were markedly elevated in both boys, corroborating the presence of primary hypothyroidism. Figure 28 shows bone age retardation

in one of these boys. Typical bone dysgenesis associated with hypothyroidism⁴³ was noted in 1965 in these boys. Figure 29 shows such changes in the heads of the humeri in one case (No. 5).

From Table 20 indications can be seen that several other children with thyroid abnormalities (subject Nos. 2, 20, 33, and 65), some of whom

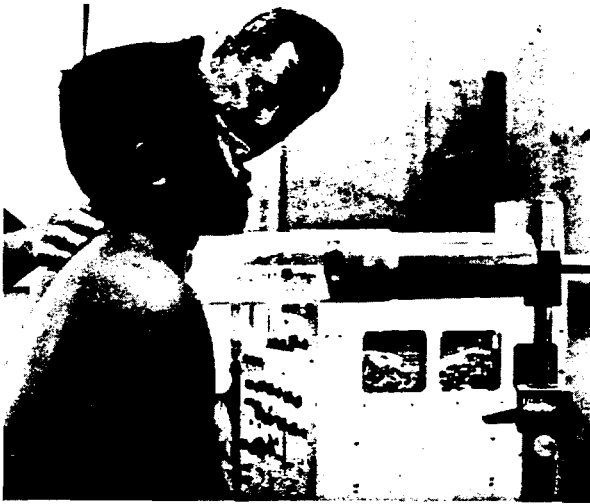


Figure 27. Thyroid uptake study with ^{132}I , Rongelap Island.

may have some degree of growth retardation, also show evidence of hypofunctioning glands or glands that are functioning at maximum capacity, based on results of thyroid function studies (elevated serum TSH levels or little or no response in ^{132}I uptake following TSH stimulation). One of these (No. 65) had a low serum thyroxine level. The adults with thyroid nodules (Nos. 59 and 64) and two of the children (Nos. 42 and 61) had normal responses to TSH. Several of the studies listed in Table 20 were performed after subtotal thyroidectomy (Nos. 17, 21, 69, 2, and 20). The results indicate inadequate function of the thyroid remnant, even in Nos. 17, 21, and 69, who had been operated upon in June 1964 and had no thyroxine replacement before September 1965. The serum iodoprotein levels (PBI less T_4 I) ranged from 1.5 to 2.9 and did not differ significantly from values in unexposed Marshallese.



Figure 28. Wrist x rays showing marked retardation of skeletal maturation in dwarfed boy with hypothyroidism (right, subject No. 3, chronological age $10\frac{1}{2}$ yr) compared with younger brother with normal osseous development (left, subject No. 83, chronological age $8\frac{1}{2}$ yr).

During the March 1966 survey, ^{132}I studies were done in 8 subjects with thyroid abnormalities. In addition to urine and neck radioactivity measurements, as described above from the 1965 survey, 6 subjects were given 500 mg KClO_4 by mouth ≈ 4 hr after the ^{132}I dose. Neck measurements were continued for 45 min longer. The neck uptake curves in these subjects are shown in Figure 30. In 3 subjects (Nos. 3 and 5, who had severe growth retardation, and No. 69, who had a subtotal thyroidectomy in 1964) the neck uptake was almost entirely due to iodide circulating in the blood, no correction having been made for this factor. In 3 other patients (Nos. 2, 54, and 65) there was a brisk uptake to about 18% of the dose. Following KClO_4 , there was no loss of iodine from the neck. If the thyroid gland had contained iodine which had been trapped as iodide but not organified, this should have been discharged by the KClO_4 . A phenomenon of this kind has been seen in radiation-damaged thyroid glands after treatment of hyperthyroidism with radioiodine^{41,42} but was not observed in the Marshallese subjects.

Computer analysis of the ^{132}I data obtained from all the patients so studied is presented in

Table 21. This includes data obtained in the Marshall Islands in March 1966 and preoperatively at BNL in June 1965 and June 1966. Computer analysis of the data obtained at BNL in June 1966 was evaluated in several ways: with or without the corrected neck counts using a lead shield, with or without inclusion of urine data. None of these made an important difference in the value for thyroid accumulation rate, but the uncorrected data gave somewhat greater reliability. The very low urine excretion rates in some cases are probably due to incomplete urine collection, and result in comparable errors, in the opposite sense, in the computed thyroid fraction. In Table 21, uncorrected neck counts are used except for the data at BNL, June 1965. In the Marshall Islands in March 1966, blood ^{132}I was measured at 2 and 4 hr in order to calculate the iodide space. The data obtained in March 1965 on Marshallese without thyroid abnormality are included for comparison. The two cases with severe growth retardation (Nos. 3 and 5) had markedly diminished thyroid accumulation of ^{132}I as did one subject (No. 59) who was on thyroxine therapy and one (No. 69) after partial thyroidectomy. Two subjects (Nos. 2

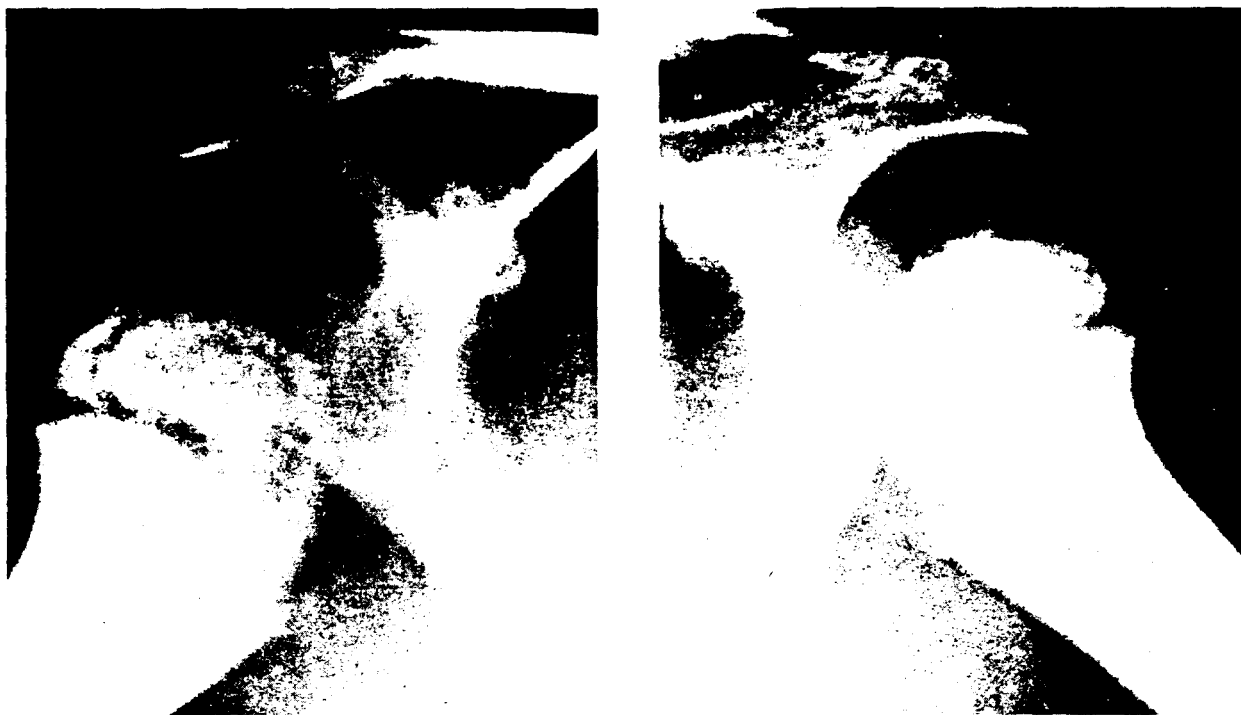


Figure 29. Bone dysgenesis of heads of humeri in subject No. 5, typical of hypothyroid disease.

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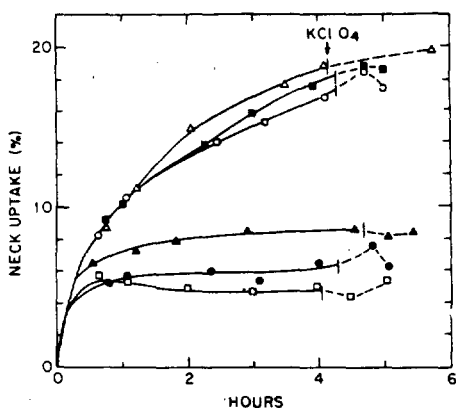


Figure 30. Neck accumulation of ^{132}I in subjects with thyroid abnormality. Values are gross neck counts as % of dose, uncorrected for blood background following oral administration of ^{132}I . Computer analysis of these data indicates that 7% of the extrathyroidal iodide pool is "seen" by the neck counter. At the vertical lines, 500 mg KClO_4 was given by mouth. Δ , Subject No. 54; \blacksquare , subject No. 2 (partial thyroidectomy); \circ , subject No. 65; \bullet , subject No. 5; \square , subject No. 3; \blacktriangle , subject No. 69 (partial thyroidectomy).

and 20) had little or no response to TSH. The other patients had apparently normal thyroid accumulation rates, and several (Nos. 33, 42, 59, 61, 64, and 65) responded to TSH. Urine excretion rates were variable and, in some instances, very low, probably because of incomplete urine collection. Following TSH, in many instances the urine excretion rate was lower than the control. The reason for this is unknown. The theoretical thyroid fraction following TSH was sometimes elevated despite a fall in thyroid accumulation rate because of a relatively greater fall in urine excretion rate. Incomplete urine collection probably accounts for the very high thyroid fraction in some cases. The thyroid accumulation rate, on the other hand, is relatively unaffected by inaccurate urine collection.

From the data in Tables 20 and 21 it is evident that several of the children who developed thyroid nodules but were not clinically hypothyroid had evidence that their thyroid glands were stimulated by elevated endogenous TSH production, and two of these children were unable to respond further to exogenous TSH.

Results of Thyroid Hormone Therapy

Although it is too soon to evaluate completely the results of thyroid hormone treatment, there are definite indications of beneficial effects. In one

adult a nodule could not be palpated 6 months after treatment was begun. In September 1966, thyroid examinations showed that 4 children with nodules (Nos. 19, 36, 54, and 72) noted previously had evidence of slight regression of the nodules, and it was decided to re-evaluate their cases in March 1967, after another 6 months of therapy. The curves for stature and bone age before and after thyroid hormone administration, plotted for the two most retarded boys (Nos. 3 and 5) in Figure 31, show a definite spurt in growth subsequent to treatment. This acceleration is very prominent for bone development in subject No. 3. Figure 32A shows the remarkable change in appearance of No. 3 after thyroid hormone therapy for 6 months. Figure 32B shows the improvement in bone maturation in wrist bones of No. 3 after 1 year of therapy - compare with Figure 28. These findings indicate that the growth retardation noted among the boys was attributable to functional hypothyroidism. Supporting this assumption is the appearance of epiphyseal dysgenesis in one of these children. Figure 29 shows this dysgenesis in the heads of the humeri. Until 1965, the serum protein-bound iodine (PBI) determinations had yielded results in the euthyroid range. It is possible, however, that the PBI levels actually represented disproportionately high amounts of physiologically inactive serum iodoprotein and inadequate amounts of active hormone. The forthcoming survey in 1967 will be important in assessing further growth stimulation from thyroid hormone treatment.

Discussion of Thyroid Findings

The development of abnormalities of the thyroid glands in the exposed Marshallese people beginning 9 years after fallout is consistent with the known etiological relationship of irradiation of the thyroid gland with the development of such abnormalities. Though the exact mechanism involved in the pathogenesis of such lesions is not clear, it is known that adenomas and cancers of the thyroid gland can be produced in laboratory animals by a variety of agents or regimens which interfere with the ability of the gland to synthesize thyroid hormone. Both benign and malignant neoplasms have been shown to be produced by iodine deficiency,⁴⁴ agents that chemically inhibit thyroxine synthesis such as thiouracil,⁴⁵⁻⁴⁹ irradiation of the gland,⁵⁰⁻⁵² and irradiation of the gland with ^{131}I .⁵³⁻⁵⁵ Furthermore, subtotal thyroid-

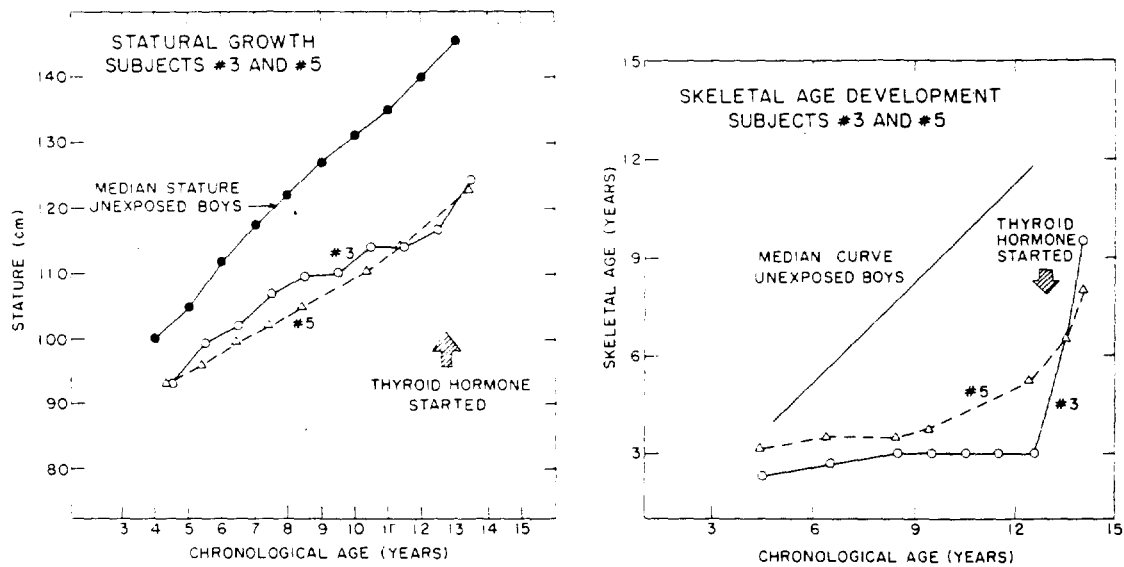


Figure 31.



Figure 32. *A*: Dwarfed boy (subject No. 3) shown at left before thyroid hormone therapy and at right after 6 months therapy with 0.3 mg thyroxine. *B*: Roentgenograph of wrist of subject No. 3 showing marked improvement in skeletal maturation after 1 year hormone treatment. Compare with Figure 28, before treatment.

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ectomy in the rat has been reported to cause adenoma formation in the remaining thyroid tissue 2 years later.^{56,57} All these procedures produce hypothyroidism which serves as an effective stimulant to the secretion of TSH by the pituitary gland. The thyroid gland under the influence of TSH first undergoes diffuse hyperplasia and hypertrophy and adenoma formation. In the cases of iodine deficiency and partial thyroidectomy, it is probable that no drug or carcinogenic agent is involved. The incidence of carcinomas after a combination of carcinogenic agents (such as radiation or acetylaminofluorene) and any factor that causes hypothyroidism is much higher.⁵⁸ Radiation by either x rays or ¹³¹I is particularly effective, since it simultaneously acts as a carcinogen and, by impairing the functional capacity of the gland, induces TSH secretion and thyroid stimulation.

The high incidence of adenomatous goiters and hypothyroidism in the more heavily irradiated children exposed at <10 years of age, compared with no abnormalities in some 100 children in the same age range in the less exposed and unexposed groups, leaves little doubt about the etiological relationship of these lesions with irradiation exposure. The higher incidence in children may be related to increased sensitivity of the child's thyroid to irradiation but is probably related more directly to the relative magnitudes of the radiation doses received, the small child's gland probably having received 5 to 10 times the radiation dose received by the adult's gland. Since most of these lesions appeared in teen-age children, puberty may have been a contributing factor. The incidence of the lesions in female children is only slightly greater than in male children (1.27 to 1). A greater prevalence of thyroid abnormalities in that sex is correctly noted. The increased incidence of thyroid abnormalities, including one case of malignancy, in the adults of the more heavily exposed Rongelap population makes it necessary to consider seriously radiation exposure as the etiological factor. In Appendix 7 statistics on thyroid malignancies in the Marshall Islands and the Trust Territory are presented. A low incidence of solitary nodules was noted in older (>50 years of age) unexposed Marshallese of both Rongelap and Utirik Islands. The single nodule case in the less exposed 46-year-old Ailingnae woman, on the other hand, may well fall into the category of the sporadic type, since the nodule was well encapsulated, and the remainder of the thyroid gland appeared normal.

Urine analyses indicate that iodine intake is adequate. The incidence of nodules and sporadic cases of goiter seen at the Majuro Hospital* is low, and the Marshallese population shows no evidence for goitrogenic factors in their environment.

The sensitivity of children's thyroid glands to the development of neoplastic changes from radiation exposure has been amply demonstrated. A series of retrospective and prospective studies have clearly shown the causal relation of irradiation of the neck region in infants and later development of thyroid cancer.⁵⁹⁻⁶² Although the calculation of the dosage in the Marshallese is subject to large uncertainties, the greater incidence of pathological changes in the glands of the Marshallese may be related to a greater dose of radiation received by their thyroid glands. Lesions similar to those seen in the Marshallese have been reported in children 5 to 11 years old after treatment with ¹³¹I for thyrotoxicosis.^{63,64} Doniach⁶⁵ points out that cell division in the growing thyroid gland of the child may be a factor in the increased sensitivity to irradiation. There appears to be an increased incidence of thyroid carcinoma in inhabitants of Hiroshima and Nagasaki exposed to radiation from the atomic bomb explosions.⁶⁶ These people were exposed to varying doses of external radiation to the thyroid gland but not to internal exposure from radioiodine.

It has been assumed that the slight growth retardation previously noted in some of the exposed children was due to radiation, but the mechanism has been obscure. The growth hormone studies suggested that pituitary function was normal. X rays of the sella turcica showed no evidence of pituitary abnormality. With the recent development of hypothyroidism in two of the most growth-retarded boys and evidence of mild hypothyroidism in several other children with thyroid nodules and growth retardation, a hypothyroid etiology seems most likely. Elevated TSH levels indicate that the hypothyroidism is primary. Minimal hypofunction of the gland may have been missed in the past, since the apparently normal PBI levels may have been spuriously high, the true thyroxine level being masked by the elevated iodoprotein component characteristic of the Marshallese.

*In March 1966, two cases of goiter with large, nodular glands were seen at the Majuro Hospital. One may have had mild hypothyroidism. These cases were not part of the population under study.

The results of institution of thyroid hormone therapy in September 1965 are of interest. The rationale for its possible value in inhibiting the development of thyroid nodules appears to be on a firm basis. Bielschowsky⁵⁸ and Astwood and Cassidy⁵⁷ have reviewed the favorable effects of thyroid treatment of patients with nodules of the thyroid gland. The only experimental evidence found directly applicable to the Marshallese situation, however, is in a paper by Nichols et al.⁶⁸ and unpublished data by Godwin⁶⁹ demonstrating a reduced incidence of ¹³¹I-induced adenomas in rats treated with thyroid hormone.

Although the children with thyroid nodules have thus far shown no evidence of thyroid carcinoma, experimental evidence strongly supports the belief that thyroid carcinoma may develop in this exposed population. This likelihood led to the performance of thyroidectomy on all children with thyroid nodules which had failed to disappear on suppressive therapy, even though earlier operative specimens had revealed only adenomatous changes. Since total thyroidectomy was not performed except in a few cases, even the operated patients will have to be followed closely for the possible development of new nodules, and the suppressive effect of thyroid hormone replacement therapy may be important even in them.

The results of the treatment of children with thyroid hormone will also test the thesis of hypothyroid etiology of growth retardation. Early indications are that the treatment is successful, since there has been a spurt in growth in the two most dwarfed boys in the year following institution of the treatment.

AGING STUDIES

During physical examinations, aging effects are usually referred to in a general qualitative sense. In order to evaluate possible aging effects better, a more quantitative approach was indicated. A large number of criteria have at one time or another been regarded as being age dependent. These studies represent an effort to select criteria which could be used under the conditions of these examinations. The over-all objective has been to combine the scores of the various criteria into one "average age score" for each individual and to compare scores in the exposed and unexposed groups of similar age. An earlier attempt to quan-

tify these aging criteria has been presented in previous publications.^{7,70} In this report several new criteria have been added, and further statistical treatment of the data has been undertaken.

Methods

The aging criteria to be presented were recorded only in adults (20 years of age and older). Data were recorded on 91 adults, 36 in the Rongelap and Ailingnae exposed groups and 55 in the larger comparison population. The ages were reasonably well distributed except for the small number of people >60 years of age. The study was hampered by the small number of people involved, lack of vital statistics on the Marshallese people, the language barrier, and uncertainty as to the exact ages in some of the older people. In selecting the criteria to be used, these difficulties limited the extent and usefulness of those tests which require motivation and cooperation on the part of the subject.*

In this report 14 criteria of aging are presented: 4 involving the special sense organs (visual acuity, accommodation, arcus senilis, and hearing loss); 4 involving neurological or neuromuscular function (vibratory sense, reaction time, rapidity of movement, and hand strength); 3 involving the integument (skin looseness, skin elasticity, and hair graying); one cardiovascular test (systolic blood pressure); and 2 miscellaneous tests (serum cholesterol and body potassium). Two of the tests required subjective evaluation on the part of the examiner (hair grayness and arcus senilis); 7 required varying degrees of motivation and cooperation on the part of the subject (visual acuity, accommodation, hearing loss, vibratory sense, reaction time, neuromuscular function, and strength); and 5 tests involved direct measurements (skin looseness, skin elasticity, systolic blood pressure, body potassium, and serum cholesterol).

Integument. A special caliper with constant spring tension was devised for skin examinations.^{7,70,71} (1) *Skin Looseness.* The skin fold at the junction of the chin and neck was measured in millimeters as described previously. (2) *Skin Elasticity.* This was measured on the back of the hand by allowing the caliper to pinch a fold of skin for

*Several tests were tried and discarded for these reasons, including vital capacity and cardiovascular response to two-step test. Also not included were several tests that were difficult to quantify such as baldness and retinal and peripheral arteriosclerosis. Other tests, such as serum folic acid and vitamin B₁₂ levels, were eliminated because of poor correlation with aging.

1 min and measuring the time for the skin fold to retract to the surrounding skin surface. The exact end point was sometimes difficult to measure in older people, and if the fold had not retracted completely in 90 sec, this time measurement was used at the maximum. (3) *Graying of the Hair*. The degree of graying was expressed on a 0 to 4+ scale as follows: 0, no graying; 1+, slight "salt and pepper" graying; 2+, moderate "salt and pepper" graying; 3+, nearly complete graying; 4+, complete graying.

Special Senses.* (1) *Eyes*. All ophthalmological values were obtained during the 1964 survey. (a) *Accommodation* was measured in diopters by use of the Prince refracting rule. The average reading of the two eyes was used. (b) *Visual acuity* was measured by Snellen's test. It was found that by taking the square root of the average visual acuity (denominator) of the two eyes the scale was more linear. Thus the best vision, 20/10, was represented as 3.2 (the square root of 10), the worst reading, 20/200, was represented as 14.1 (the square root of 200), and intermediate readings were similarly recorded. (c) *Arcus senilis* was estimated on a 0 to 4+ scale. Only slight limbic clouding was considered as 1+ with increasing clouding as 2+ or 3+ and marked clouding as 4+.

(2) *Ears (Hearing)*. Audiometric examinations were carried out in a special cubicle lined with acoustic tile. A rugged screening type of audiometer was used.** Impairment of hearing was averaged for the two ears as follows: the decibel loss for each of the 6 frequencies (200, 500, 1000, 2000, 4000, 7000) for each ear was averaged, and a mean frequency loss in decibels for the two ears was obtained.

Cardiovascular Changes. *Systolic Blood Pressure*. Two readings were obtained with the standard aeronoid cuff-type sphygmomanometer, and the average of the two was used. There was no basic or

*With regard to the reliability of determinations using the Prince refracting rule, Snellen's test, and hearing acuity, it should be pointed out that these 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 have the test repeated by more than one examiner. The data from these tests are believed to be sufficiently reproducible to be of relative value, although not so accurate perhaps as might be obtained under more desirable conditions.

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



Figure 33. Marshallese subject taking light-extinguishing test of neuromuscular and mental ability as part of the aging study.

adjusted level of physical activity such as resting for a standard period of time prior to the readings. Pressures were taken from the left arm with the subject supine during the course of the physical examination.

Neurological and Neuromuscular Function. (1) *Vibratory Sense*. Vibratory perception was measured over the head of the left tibia by an electric vibrometer set at a standard frequency of 120/sec at 20 V. The end point was the voltage intensity required for perception of the vibration. (2) *Neuromuscular Function*. This 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 (1964 data). The total number of depressions represented the score. (3) *Light Extinction Time.** A battery of lights were connected in random series, and the subject extinguished

*We are grateful to Dr. J.W. Hollingsworth of Yale University for information on the construction and use of this instrument.

each light as it appeared. The total time necessary to extinguish all the lights was measured. This test involved both manual dexterity and mental reaction time⁷² (see Figure 33). (4) *Hand Strength*. The spring tension of the hand grip was measured in kilograms with a Smedly hand dynamometer.* The maximum squeeze strength in the dominant hand in 3 tries was recorded.

Miscellaneous Tests. (1) *Body Potassium Levels* (⁴⁰K). Since loss of muscle mass occurs with aging and total body potassium is closely related to muscle mass, it has been shown that body levels of potassium decrease with age.^{73,74} Whole-body spectrographic analyses were carried out in the Marshallese in a lead-shielded structure. In this way ⁴⁰K levels were obtained and presented as K/kg body weight. The accuracy of these data could have been improved by obtaining more precise determinations of lean body mass, such as by measurement of body water.⁷⁴ The values listed are averages of the 1961 and 1965 determinations. (2) *Blood Serum Cholesterol Levels*. These were obtained in 1957 on sera sent to the United States.**

*S.H. Stoelting Co., Chicago, Ill.

**The analyses were done at the National Institutes of Health by Dr. J.H. Bragdon and Mr. J.C. Lauter.

Correlation of Data. In order to place all the data on a common basis for comparison and combination, the data were converted to a percentage basis for each criterion. In the case of the measured data, values associated with least aging were taken as 0% (sometimes the highest reading, as with hand strength; sometimes the lowest, as with hearing loss), and conversely the values showing most aging were taken as 100%. The estimated criteria with values of 0, 1+, 2+, 3+, and 4+ were presented as 0, 25, 50, 75, and 100%, respectively. The data were examined on an individual basis as well as on a population basis, and comparisons were made largely by using means for each decade age group. Except where indicated, the data used were collected during the 11- and 12-year surveys.

An analysis of variance was used to determine differences among the factors of sex, age, and radiation levels.* These data were programmed and analyzed on a high speed digital computer.** For each criterion, the combined score over sex and radiation level was correlated with age. A curve of

*Mr. Keith Thompson of Brookhaven National Laboratory did the statistical analyses of these data.

**The Control Data Corporation 6600, at Brookhaven National Laboratory.

Table 22

Correlation of Criteria With Age and Radiation Exposure

	Correlation with age (<i>r</i> value)	Correlation with radiation	
		Percent	Significance* (<i>p</i>)
Grayness	0.87	+17.0	N.S. (0.70)
Arcus senilis	0.83	0.0	N.S. (1.00)
Accommodation	0.81	-14.1	N.S. (0.11)
Skin retraction	0.74	+ 7.3	N.S. (0.68)
Skin looseness	0.70	+ 1.6	N.S. (0.82)
Vibratory sense (M + F)	0.70**	- 1.4(M), +24.6(F)	N.S. (0.90, 0.20)
Visual acuity	0.69	+14.0	N.S. (0.59)
Hearing loss	0.67	+ 7.9	N.S. (0.40)
Hand grip (M + F)	0.67**	+13.8(M), +13.8(F)	N.S. (0.15, 0.18)
Reaction time (M + F) (light extinction test)	0.64**	- 2.0(M), -10.5(F)	N.S. (0.88, 0.55)
Systolic blood pressure	0.55	-11.5	N.S. (0.30)
Potassium (M + F)	0.41**	-14.6(M), +10.6(F)	N.S. (0.17, 0.22)
Cholesterol	0.39	-17.2	N.S. (0.05)
Neuromuscular function (M + F) (hand tally)	0.36**	+ 3.2(M), +1.1(F)	N.S. (0.85, 0.95)
Combined score†	0.99	+ 7.0	N.S. (0.27)

*N.S. - not significant at 5% level.

***r* values for males and females averaged.

†Weighted according to *r* value.

second degree in age was used when there was a significant departure from linearity. The criteria vibratory sense, hand grip, light extinction time, hand-tally count, and body potassium showed significant sex differences, and were therefore scaled for each sex (0 to 100%).

An attempt to correlate a combined "physiologic" age score with chronological age was done by obtaining a weighted average score for combined criteria for each age group (see Table 22). The absolute values of the correlation coefficients were used as the weighting factor. This has intuitive appeal, since the influence on the final combined score of any particular aging criterion is proportional to the absolute value of its correlation with age.

Results of Aging Studies

The results of these tests are presented graphically in Figures 34 to 48 and in Table 22. The mean values for each decade starting at age 20 are plotted at the midpoint of that decade for exposed and unexposed groups separately and combined, along with the standard errors of the means. The number beside each point is the number of people tested. The curve or straight line (whichever best represents the particular data) is drawn and its equation presented. The coefficient of correlation with age and significance is also presented. Most of the criteria show good correlation with aging. It is apparent that most of the criteria show the least change with age in the younger age groups, from 20 to 40 years of age, particularly systolic blood pressure, hearing, visual acuity, neuromuscular function, and skin retraction. Above about 40 years of age the criteria show the greatest change. With a few criteria, changes in the older age group tend to level off or be less steep (accommodation, visual acuity, skin looseness, and cholesterol).

In Table 22 age and radiation dependence of these criteria are presented. The criteria are arranged in descending order of correlation with aging as shown by the correlation coefficients. For those criteria in which sex differences were apparent, the r values for the two sexes were averaged to determine the over-all correlation with aging. Though there were isolated significant differences between the exposed and unexposed age groups for some criteria, no meaningful pattern emerged. The table shows that the correlation with radiation ef-

fects, in comparing exposed and unexposed groups, is not significant at the 5% level for any of the criteria. The over-all 7% increase in aging effects in the exposed group is not significant ($p = 0.27$). In Figure 48 the composite weighted biological or physiologic age scores are plotted against chronological age (means per decade). Combined mean values for exposed and unexposed groups are also presented with standard errors of the means. The over-all correlation of the biological age scores with chronological age is very good. The biological "age curve" (Figure 48) shows that there is less change in the younger age groups than there is after about age 40. Mean values for exposed and unexposed are not significantly different.

Discussion of Aging Studies

Though the criteria presented in this report show changes which are definitely correlated with chronological age on a group basis, such correlation is much less accurate on an individual basis. These tests of aging are least useful in the younger age groups (20 to 40 years of age), since most of the criteria either are not present until later or if present show slight increase during this age period. Therefore, more sensitive tests are needed to show aging in these groups.

Effects of radiation on aging as measured by these parameters were not detected in this population. Perhaps the tests were not sensitive enough to detect such effects at the level of radiation exposure sustained. It is not known if any of the "aging parameters" ordinarily associated with senescence are necessarily associated with irradiation aging, since the latter is very poorly defined or understood. Even if they were, this would not necessarily mean that they are related to mortality, which is a more pertinent correlation. Such criteria as hair graying, arcus senilis, neuromuscular function, etc., would not seem to have any obvious relation to mortality. On the other hand, loss of vigor and organ disfunction could well predispose to the development of old age diseases which would enhance mortality. Increase in mortality and life shortening in the exposed Marshallese is difficult to assay in view of the small numbers of people involved and the slightly greater proportion of older people originally in the exposed group compared with the unexposed group.

One radiation effect which might be classified under aging is the inducement of malignant disease

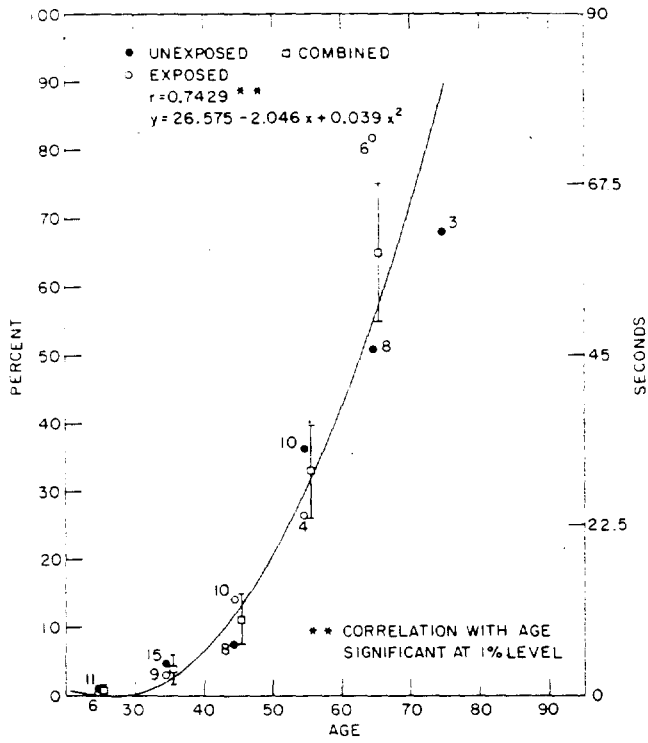


Figure 34. Skin retraction.

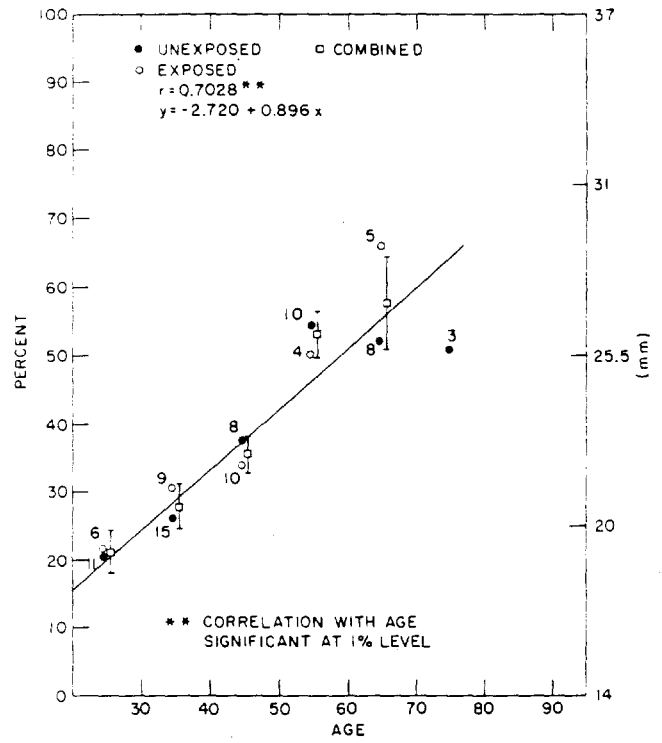


Figure 35. Skin looseness.

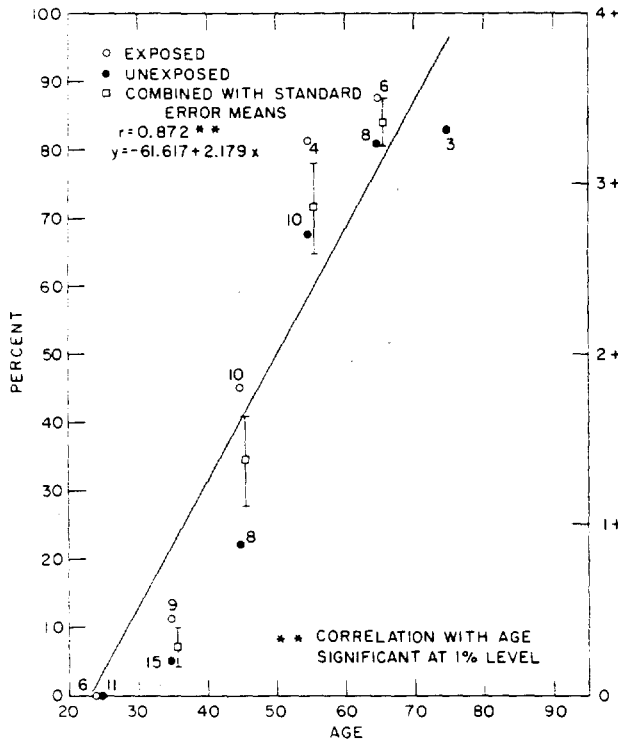


Figure 36. Hair grayness.

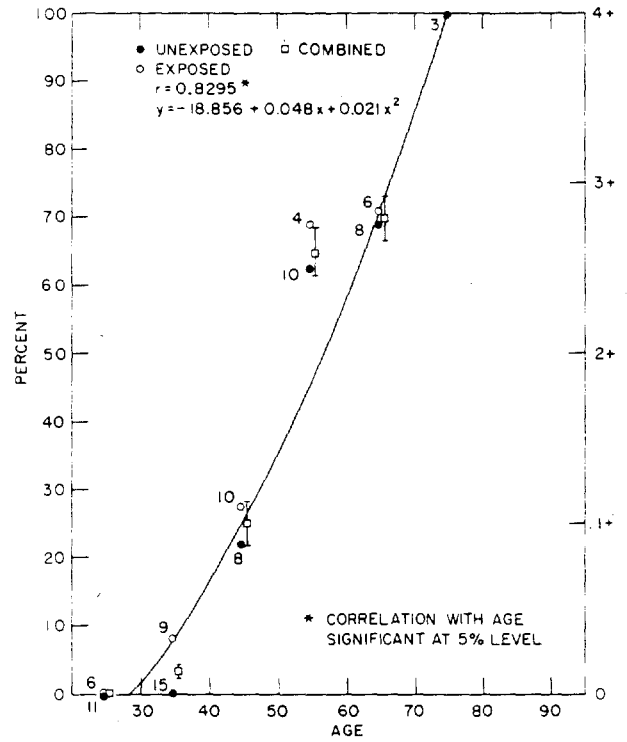


Figure 37. Arcus senilis (1964).

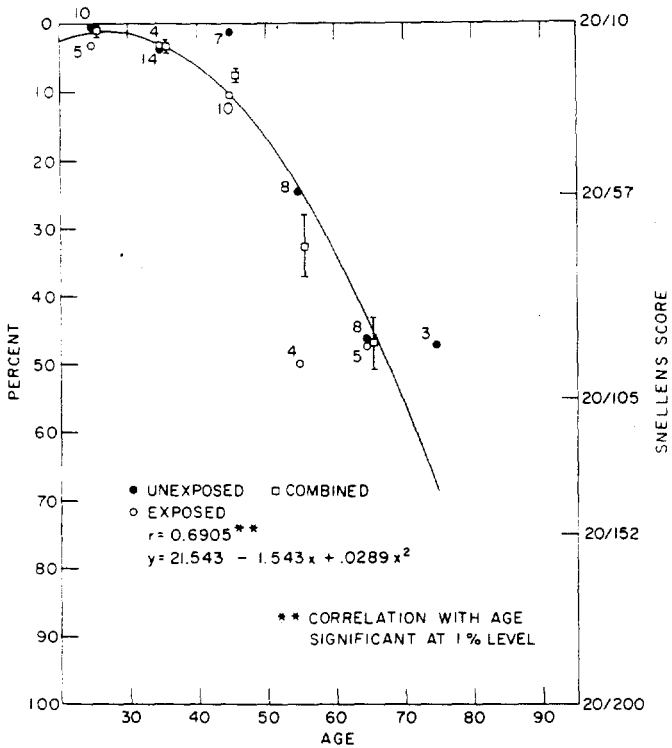


Figure 38. Visual acuity (1964).

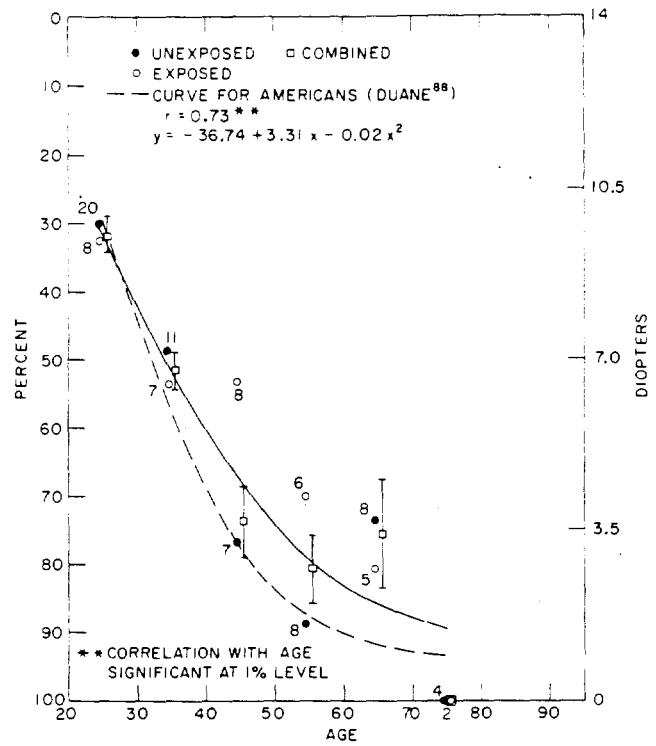


Figure 39. Accommodation (1964).

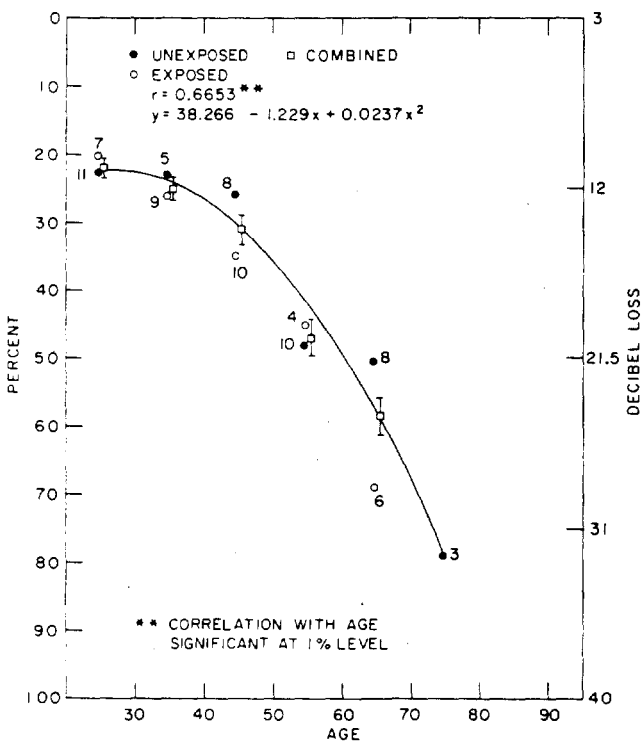


Figure 40. Hearing (average decibel loss).

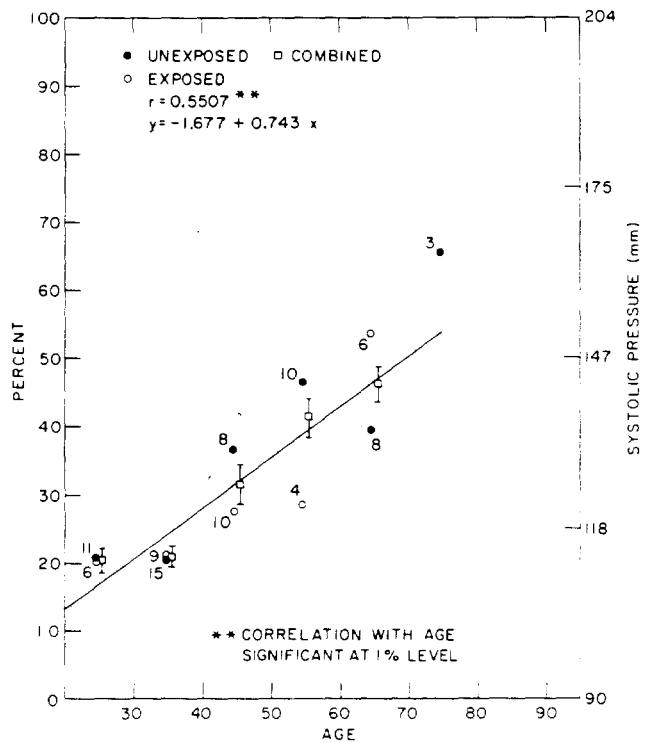


Figure 41. Systolic blood pressure.

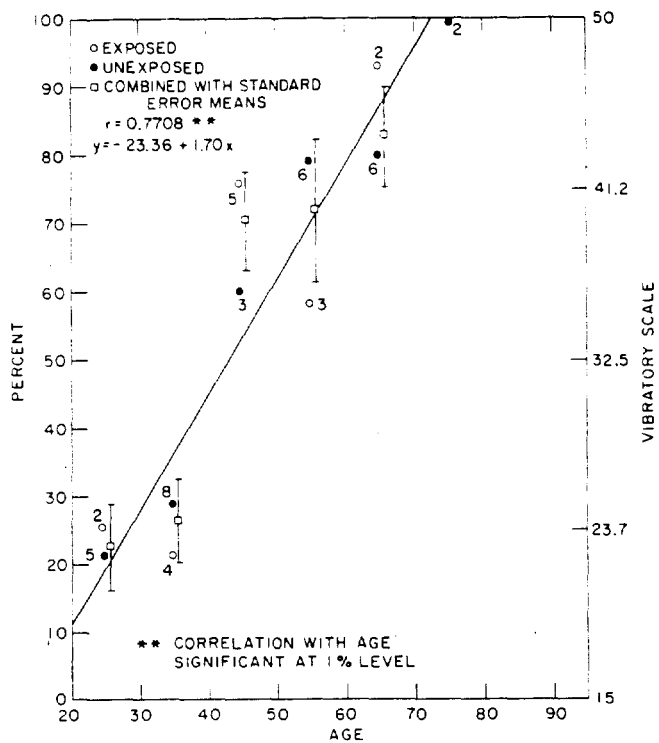


Figure 42. Vibratory sense in tibia, males.

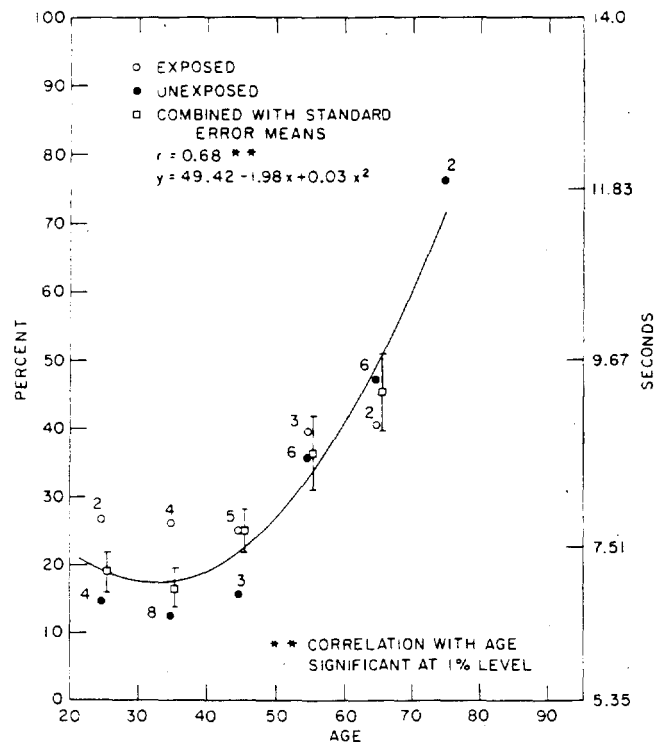


Figure 43. Reaction time (light extinction test), males.

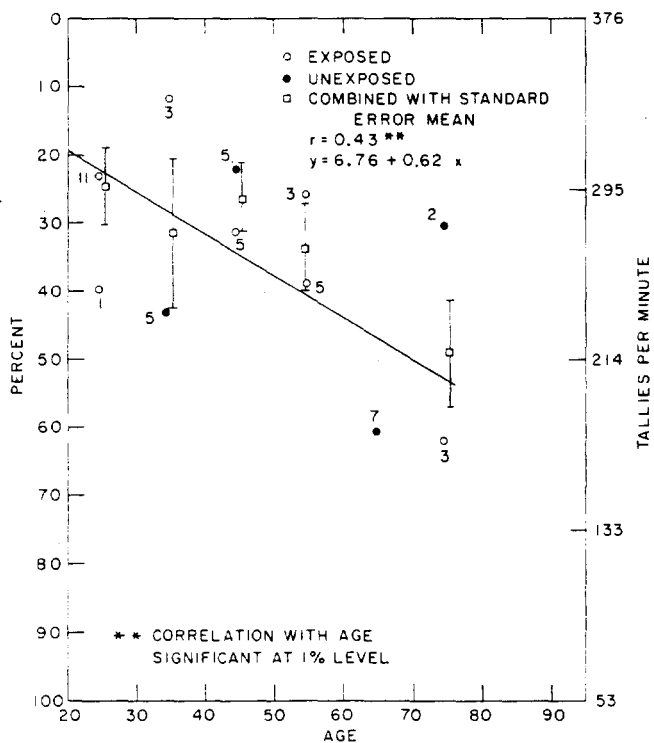


Figure 44. Neuromuscular function (hand tally test), males (1964).

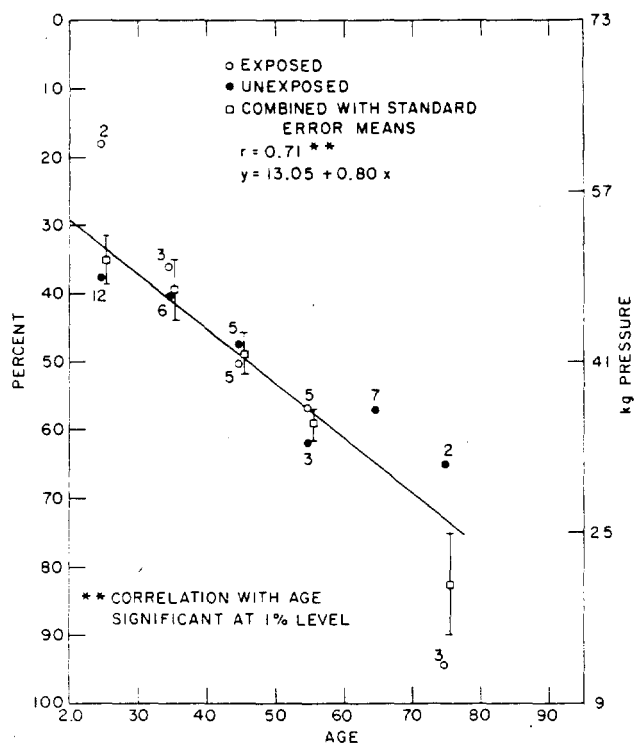


Figure 45. Hand grip, males.

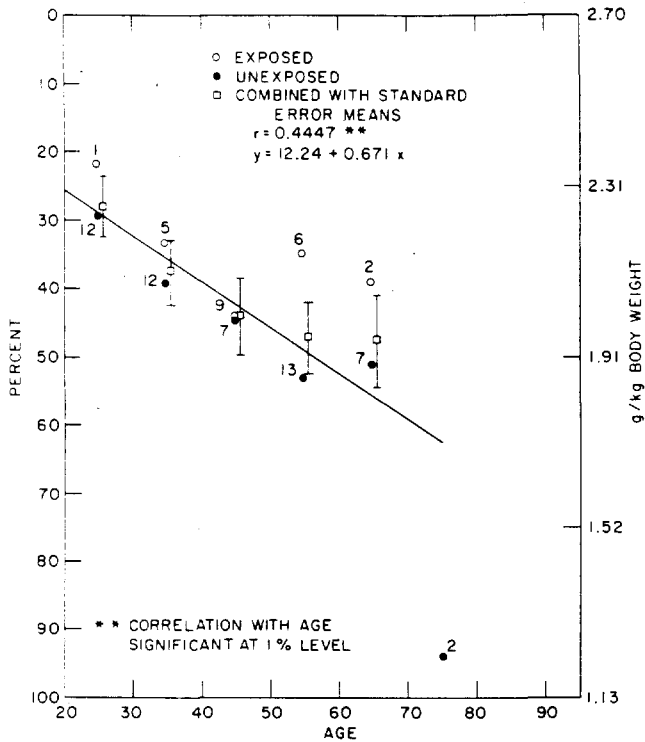


Figure 46. Potassium, males (1961 and 1965).

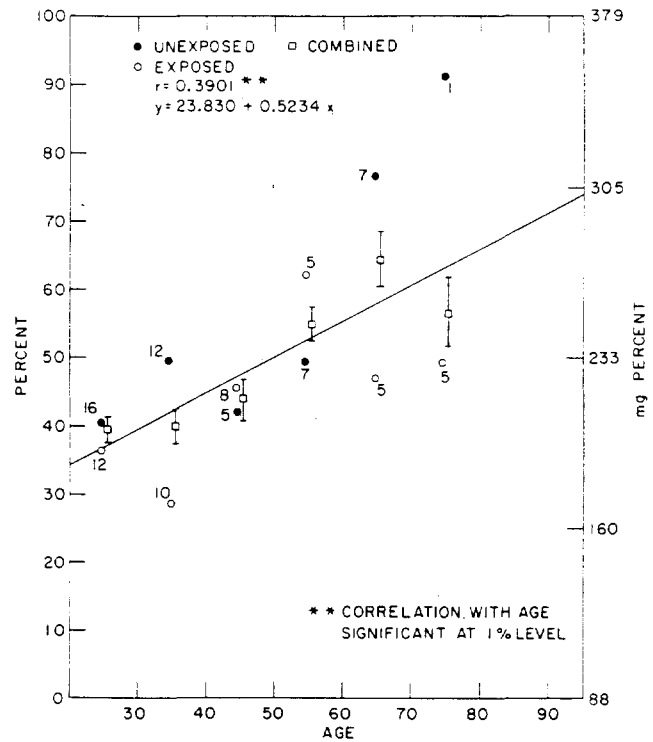


Figure 47. Cholesterol (1957).

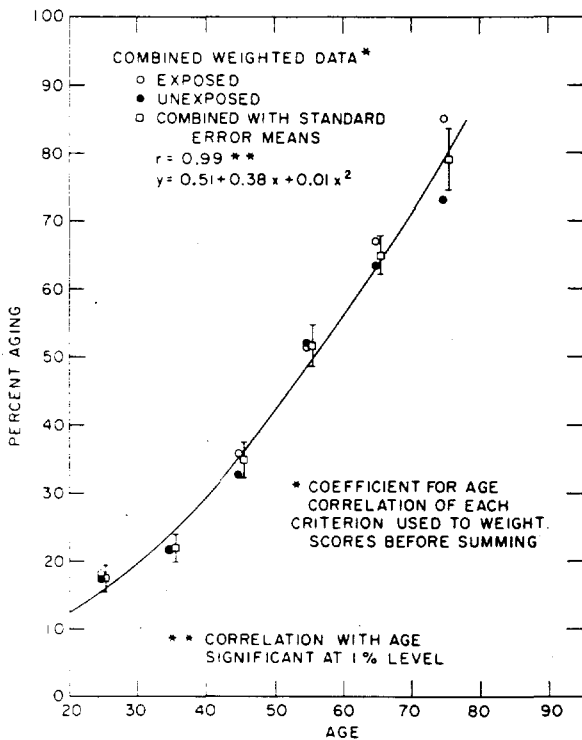


Figure 48. Biological age scores.

at an earlier time than it would ordinarily be manifest. One aspect of this phenomenon may be related to rapidity of cell proliferation (generation cycle) in a particular tissue, i.e., with more rapidly proliferating cells of the bone marrow, leukemia occurs relatively soon after irradiation, while malignant lesions of the skin would not become manifest until later in this more slowly proliferating tissue. In the Marshallese the high incidence of adenomatoid goiter in the more rapidly proliferating thyroid glands of the children may possibly represent premature appearance of lesions which are known to occur later in life and thus in a sense represent an aging process. On the other hand, the fact that no tumors (except benign nevi) have been noted in the heavily irradiated skin of these people might indicate a longer induction period due to slower cell turnover of the skin.

In considering future aging studies in the Marshallese it is hoped that more sensitive tests of vigor and organ function suitable to the conditions of study may be used. However, it must be borne in mind that such subtle aging effects may not become detectable by ordinary clinical means

Table 23

Mean Levels of Peripheral Blood Elements of Populations Under Study

	Plat. ($\times 10^{-3}$)		WBC ($\times 10^{-3}$)		Neut. ($\times 10^{-3}$)	
	1965	1966	1965	1966	1965	1966
Exposed	274 \pm 75*(56)**	—	7.39 \pm 1.7 (56)	6.82 \pm 1.8(55)	3.69 \pm 1.2 (56)	3.57 \pm 1.4(54)
Unexposed	342 \pm 95 (16)	—	7.12 \pm 1.4 (16)	6.24 \pm 2.3(15)	3.77 \pm 1.5 (16)	3.30 \pm 1.9(15)
Exposed children of parents	282 \pm 71(124)	—	7.34 \pm 2.2(124)	8.05 \pm 2.3(99)	3.86 \pm 1.8(125)	4.45 \pm 1.8(98)
Unexposed children of parents	416 \pm 130 (48)	—	10.17 \pm 2.5 (48)	—	4.41 \pm 2.0 (48)	—
Exposed children of parents	366 \pm 96 (69)	—	9.91 \pm 2.6 (69)	—	4.52 \pm 1.9 (69)	—
	Lymph. ($\times 10^{-3}$)		Hct. (%)		RBC ($\times 10^{-6}$)	
	1965	1966	1965	1966	1965	1966
Exposed	2.99 \pm 0.8 (56)	2.45 \pm 0.8(54)	40.2 \pm 4.8 (56)	39.7 \pm 4.3(48)	427 \pm 57 (50)	—
Unexposed	2.73 \pm 0.8 (16)	2.31 \pm 0.9(15)	39.6 \pm 5.0 (16)	39.7 \pm 4.6(15)	438 \pm 53 (16)	—
Exposed children of parents	2.82 \pm 1.0(124)	3.00 \pm 1.0(98)	40.5 \pm 5.0(124)	41.3 \pm 7.0(88)	443 \pm 53(111)	—
Unexposed children of parents	4.78 \pm 1.9 (48)	—	37.1 \pm 3.5 (48)	—	441 \pm 41 (44)	—
Exposed children of parents	4.55 \pm 1.5 (69)	—	36.4 \pm 2.4 (69)	—	439 \pm 34 (64)	—

Abbreviation. **Number of people in group.

or tissue shows advanced morbidity. In longitudinal studies of aging Rongelap individuals and groups may differ as time goes by.

HEMATOLOGICAL STUDIES

The hematological data are summarized in tables and the raw data on individuals are in the appendices. The more heavily exposed group who received 175 rads is designated "Rongelap exposed," the Rongelap who received a smaller exposure of 100 rads is designated "Rongelap unexposed," and the larger unexposed population of Rongelap as "unexposed." Because of the small number of people in the unexposed group, their data were not included for the Rongelap groups, but are summarized in a separate paragraph. The data are summarized separately in the appendices. The differences noted in age between the exposed and the unexposed groups in the comparisons of mean

levels for entire groups, comparisons are also made of age and sex groups. Ages 12 to 15, >15 to 40, and >40 years for each sex are compared.

The hematological data are summarized in Table 23 and in Figures 49 through 65. In Appendices 8, 9, and 10 are presented summaries of the mean blood counts of the exposed populations and of the various comparison populations since exposure in March 1954. In Appendices 11 and 12 are listed the individual blood counts for 1965 and 1966. In Appendix 13 basophil counts are presented. In 1966, hematological studies (excluding platelets) were done only on the exposed populations of Rongelap, Ailingnae, and Utirik.

Rongelap Population

Leukocytes. The mean leukocyte levels were slightly lower at 11 years than at 10 years post exposure in both exposed and unexposed Rongelap people. At 12 years the exposed (unexposed not done) showed slightly lower levels than the unexposed in 1965 (see Appendix 8). There was no apparent difference in the exposed and unexposed

counts at 11 years (7.4×10^3 in the exposed and 7.3×10^3 in the unexposed).

Differential counts showed neutrophils and lymphocytes to have very nearly the same mean level for exposed and unexposed Rongelapese at 11 years. Graphic treatment of these elements is presented in Figures 49 through 54. The similarity of counts between the two groups is probably best seen in the cumulative percent distribution curves (Figure 55). The levels of eosinophils, monocytes, and basophils were not remarkably different between the exposed and unexposed groups and were similar to the levels in past surveys.

Platelet counts at 11 years were about the same in the exposed and unexposed in both males and females (Figures 55 through 58). The closeness of counts in the two groups is best seen in Figure 55.

Erythropoietic function appeared to be about the same in exposed and unexposed groups based on

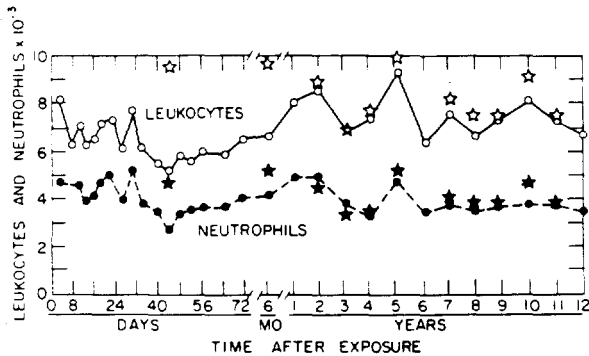


Figure 49. Mean neutrophil and white blood counts of Rongelap exposed people from time of exposure through 12 years post exposure. Stars represent mean values of comparison population.

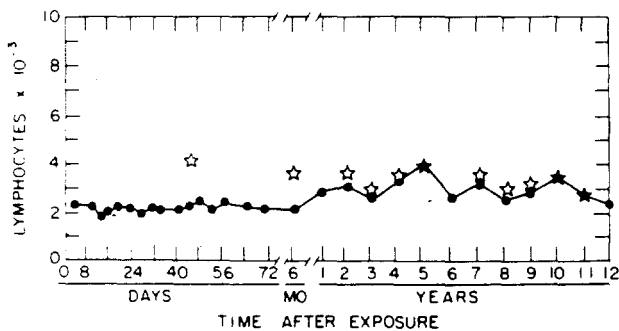


Figure 50. Mean lymphocyte counts of Rongelap exposed people from time of exposure through 12 years post exposure. Stars represent mean values of comparison population.

red cell counts and hematocrits at 11 years (see Figures 59 through 64).

During the 12-year survey, counts were done only on the exposed group, and no comparison with unexposed was done because of annual variability in counts. However, the hematological findings at 12 years in the exposed group are not

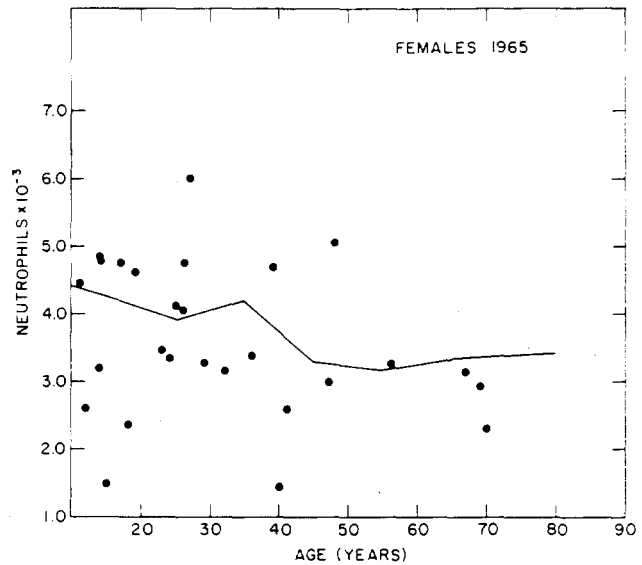


Figure 51. Neutrophil counts of Rongelap exposed females plotted against age. Solid line represents mean level of unexposed female population, 1965.

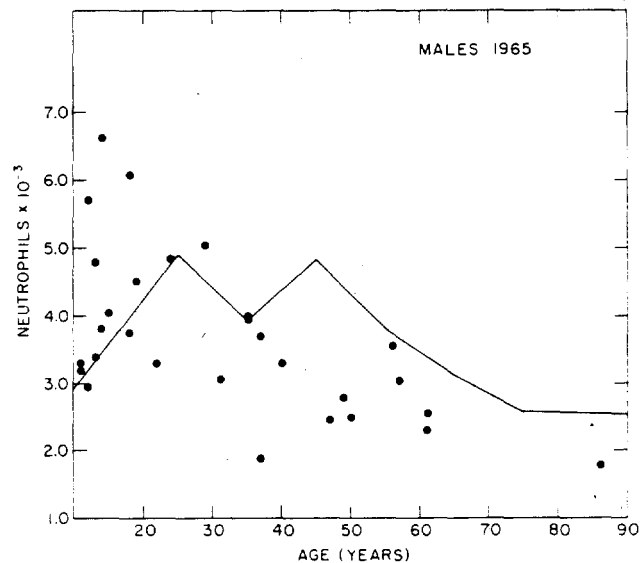


Figure 52. Neutrophil counts of Rongelap exposed males plotted against age. Solid line represents mean level of unexposed male population, 1965.

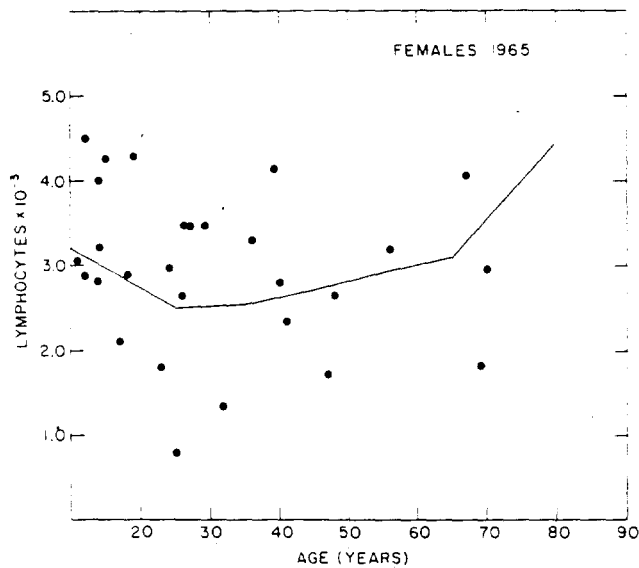


Figure 53. Lymphocyte counts of Rongelap exposed females plotted against age. Solid line represents mean level of unexposed female population, 1965.

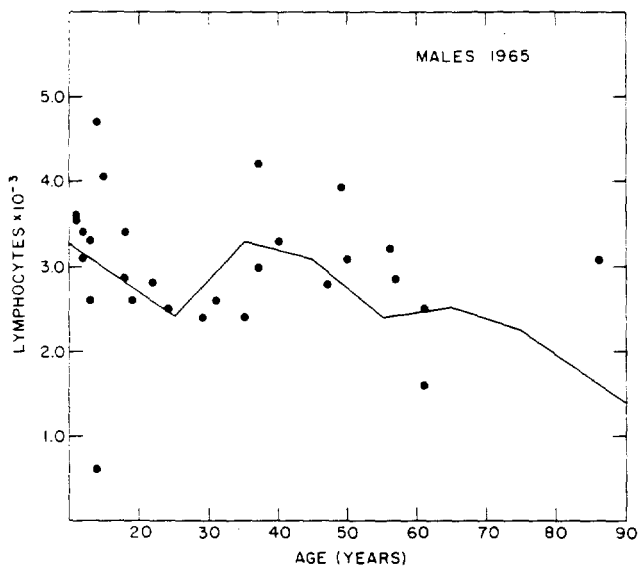


Figure 54. Lymphocyte counts of Rongelap exposed males plotted against age. Solid line represents mean level of unexposed male population, 1965.

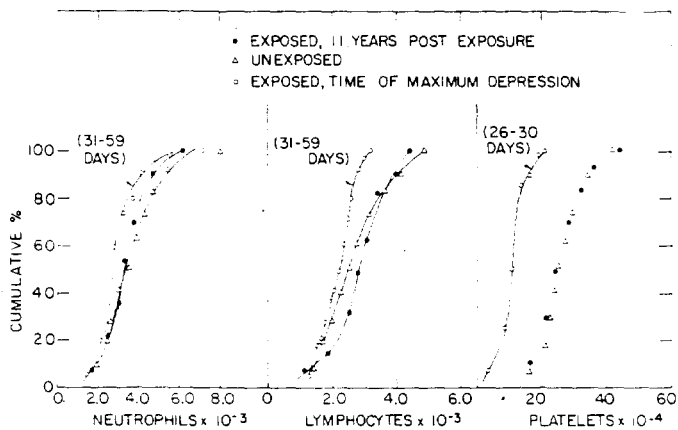


Figure 55. Cumulative percent distribution curves for neutrophils, lymphocytes, and platelets, 1965.

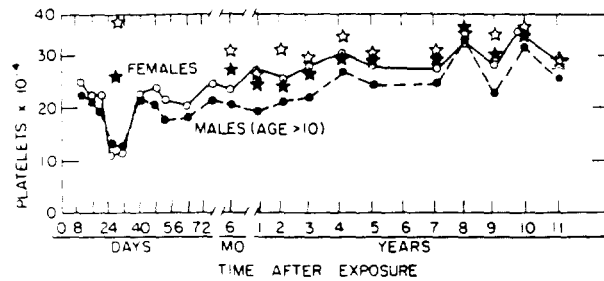


Figure 56. Mean platelet values of Rongelap exposed people from time of exposure through 11 years post exposure. Stars represent mean values of unexposed comparison population.

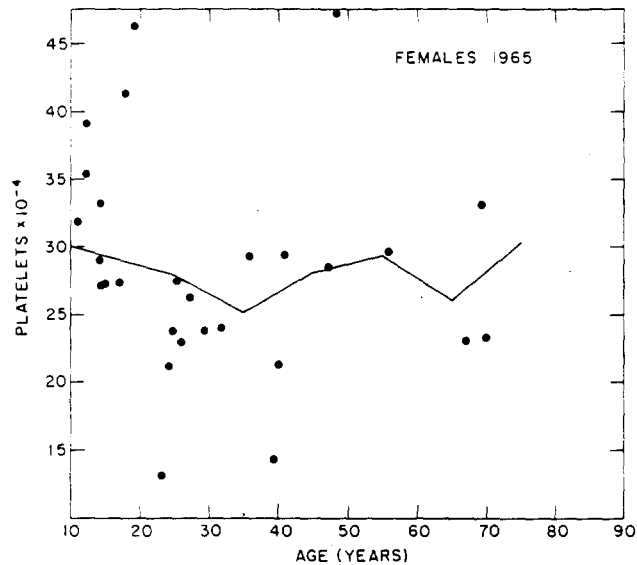


Figure 57. Platelet counts of Rongelap exposed females plotted against age. Solid line represents mean level of unexposed female population, 1965.

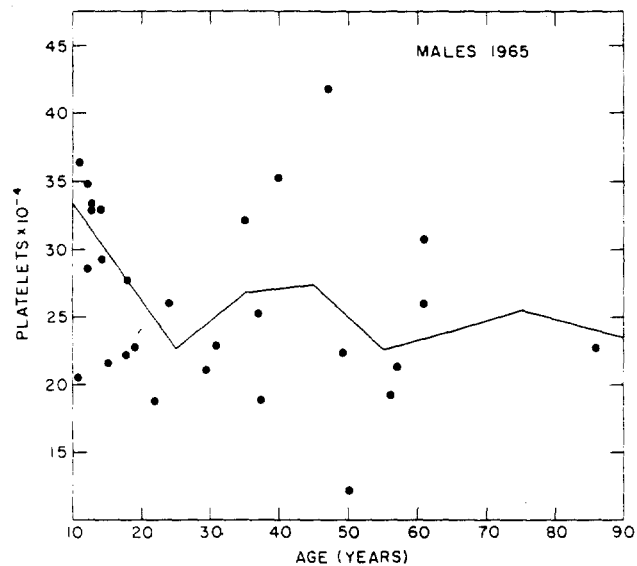


Figure 58. Platelet counts of Rongelap exposed males plotted against age. Solid line represents mean level of unexposed male population, 1965.

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platelet counts were done. The hematocrit levels were slightly increased over levels of 3 years ago, possibly indicating a better state of nutrition. None of the mean counts were very different from those of the unexposed comparison population (1965 counts) except for leukocytes, which were somewhat higher in the Utirik people. Appendix 10 shows levels of blood elements at various times since exposure.

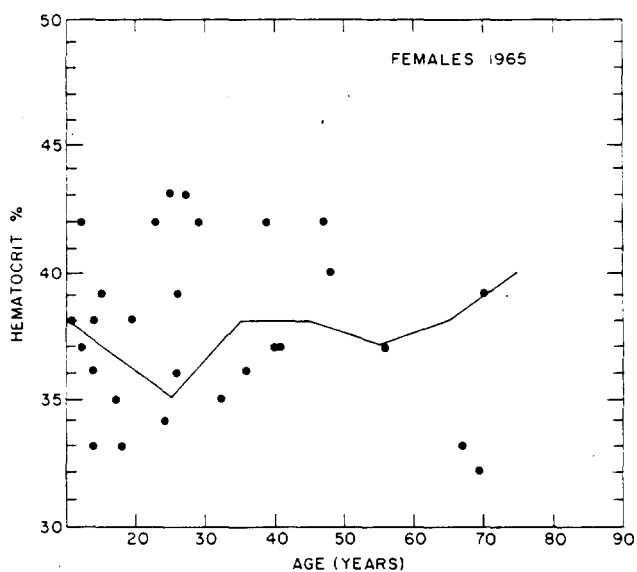


Figure 63. Hematocrit values of Rongelap exposed females plotted against age. Solid line represents mean level of unexposed female population, 1965.

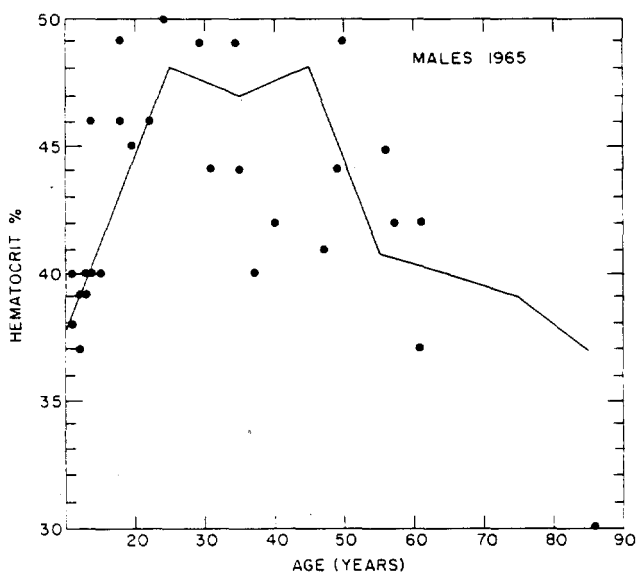


Figure 64. Hematocrit values of Rongelap exposed males plotted against age. Solid line represents mean level of unexposed male population, 1965.

Children of Exposed Parents

Children of exposed parents born since the fall-out showed levels of peripheral blood elements about the same as those of children of unexposed parents of the same age range.

Leukemia Survey

No evidence of preleukemia or leukemic state was detected in any of the people examined, either from the blood picture or from physical examination. Counts of 4000 white cells for percent basophils revealed no abnormally high levels (see Appendix 13). Subject No. 3, a 13-year-old boy, continues to show about twice the number of basophils (1.13%) noted in other Rongelapese. He has developed marked hypothyroidism during the past 2 years but shows no evidence of incipient leukemia.

Statistical Analysis of Rongelap Blood Data

In the last report¹⁰ a factorial analysis of variance of Rongelap blood data over the previous 4 years indicated that the slight depression of blood elements (leukocytes and platelets) of the exposed population over this period was highly significant. However, a similar analysis of the 1965 hematological data shows that at this time there was no significant difference (at the 5% level) of platelets, leukocytes, neutrophils, or lymphocytes between the exposed and unexposed Rongelap population.* This was also true for the Ailingnae population. The significantly greater platelet level in this group is unexplained. There was evidence that the leukocyte and platelet levels of the youngest age group in all populations were higher than in adults. No sex differences could be demonstrated in any of the 1965 data. The percent difference in blood levels between exposed and unexposed groups is presented in Table 24 and Figure 65. It can be seen that at 11 years there is no substantial difference between the groups.

CHROMOSOME STUDIES

Since chromosome aberrations have been demonstrated in cultured blood cells from various groups of people exposed to radiation, even years after exposure, it was of interest to see whether any such aberrations were present in the people of

*We are grateful to Mr. Keith Thompson of Brookhaven National Laboratory for the statistical analysis of these data.

Table 24

Percent Differences of Various Hematological Determinations Comparing Exposed Groups With Unexposed Group

	Plats.	Leuks.	Neuts.	Lymphs.
Rongelap				
1959*	- 5.9	- 5.5	- 8.2	- 0.5
1961	-14.9 ($p < 0.01$)	- 5.9	- 6.0	- 5.5
1962	- 8.0 ($p < 0.1$)	-11.1 ($p < 0.01$)	-13.6 ($p < 0.05$)	-13.0 ($p < 0.01$)
1963	-16.0 ($p < 0.01$)	- 3.5	- 4.4	- 3.9
1964	- 4.9	-10.5 ($p < 0.05$)	-23.5 ($p < 0.01$)	- 1.1
1965	+ 0.1	+ 1.4	- 5.1	+ 5.7
Ailingnae				
1959	- 4.4	- 4.8	- 6.3	- 8.4
1961	-12.6 ($p < 0.1$)	-	-13.6	+ 8.1
1962	- 6.5	-15.1 ($p < 0.05$)	-17.7 ($p < 0.1$)	-11.0
1963	-23.4 ($p < 0.01$)	- 6.7	+ 3.9	-20.8 ($p < 0.01$)
1964	+ 4.8	-22.1 ($p < 0.02$)	-26.4 ($p < 0.1$)	-11.1
1965	+24.8 ($p < 0.01$)	- 2.7	- 2.6	- 3.6
Utirik				
1963	+22.7 ($p < 0.01$)	-	+ 2.0	- 3.2

*Earlier data will be analyzed in the near future.

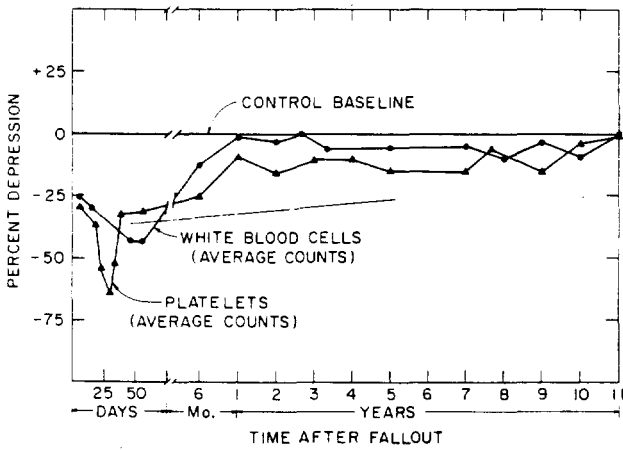


Figure 65. Depression of blood elements in Rongelap exposed people compared with average counts of unexposed people.



Figure 66. Two-hit chromosome aberrations in exposed Marshallese. Top: arrow points to dicentric form; bottom; arrow points to ring form.

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Rongelap as a result of their fallout radiation exposure. Attempts at 8 and 9 years post exposure to obtain suitable blood cultures had not been entirely successful. However, during the 10-year survey a slight modification of the Moorehead technique⁷⁵ resulted in a series of satisfactory cultures on 51 people: 30 in the higher exposure group (175 rads), 13 in the lower exposure group (69 rads) and 8 from the unexposed Rongelapese who were on another island at the time of the accident. The detailed results of these studies are presented in Appendix 14.*

Table 25 summarizes the results. A higher incidence of aneuploid cells was noted in the exposed group, but the difference was not great enough to be significant. Unexpectedly, the lower exposure group showed more aberrations than did the more heavily exposed group, and the latter group showed even less aberrations than the unexposed. However, the incidence of 2-hit aberrations was significantly higher ($p < 0.004$) in the exposed groups and did appear to be radiation induced. Figure 66 shows a dicentric and a ring form noted in chromosome spreads from two exposed individuals.

OTHER LABORATORY STUDIES

Total Blood Volume and Red Cell Volume

Previous studies (1961, 1962) with ⁵¹Cr-labeled erythrocytes on Marshallese subjects living in their native environment have shown reduced red cell

*We are grateful to Dr. Shields Warren and his group at the Cancer Research Institute in Boston for carrying out the chromosome analyses.

mass and/or total blood volume with total body weight used as a base line. During the 1963 survey, similar studies were performed on 21 Marshall Islanders, but these data were related to total body water as determined by tritiated water.¹⁰ Results showed that in all instances but one the values for red cell mass and total blood volume fell below normal levels for persons living in temperate zones of the United States.

The present study was undertaken during the surveys in 1965 and 1966. A total of 19 Caucasian Americans (3 females and 16 males) living in the Marshall Islands for periods of 3 months to 9 years were examined by the same techniques.* The results of these studies on each individual are presented in Appendix 15, along with data on the 21 Marshallese in whom these studies were carried out in 1963. The data were programmed and analyzed by a high speed digital computer. Regression lines obtained for the Caucasians and the Marshallese are presented in Figure 67 along with regression lines of Moore⁷⁶ and Siri⁷⁷ for Americans.

The Marshallese regression lines for both blood volume and red cell volume have very nearly the same slopes as the lines of the Siri and Moore groups, but they are significantly below the latter (significant at the 1% level). The Caucasians living in the Marshall Islands also show regression lines for blood volume and red blood cell volume with slopes similar to those of the Marshallese and the Siri and Moore groups. Comparison of the regression lines shows no significant difference between

*We are grateful to Dr. W.E. Siri, University of California, for carrying out the tritium-water analyses.

Table 25

Summary of Chromosome Findings

Group	No. of persons	No. of cells scored	Percent of cells with $2n \neq 46$	No. of persons with aberrations	Chromosome aberrations					Total cells with aberrations	Chromatid breaks	Isochromatid gaps
					Frag-ments	Dicen-trics	Rings	Ex-changes	Total aberrations			
Exposed 175 rads	30	1500	10	12 (40%)	11	6	-	5	22 (1.46%)	20 (1.33%)	43	15
Exposed 69 rads	13	650	8	11 (84.6%)	10	2	1	8	21 (3.23%)	18 (2.77%)	31	4
Unexposed	8	400	5.5	5 (62.5%)	9	-	-	-	9 (2.25%)	8 (2.0%)	6	5

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the blood volumes and the red cell volumes of the Caucasians living in the Islands and the Caucasians of the Siri and Moore groups; furthermore, duration of residency in the Islands has no significant effect. Earlier data had suggested that Caucasians living in the Marshall Islands might have reduced blood volumes and red blood cell volumes. Though this may be true for certain individuals, it does not seem to hold true for the group as a whole.

Test for Australia Antigen

The Australia antigen, a serum protein first detected in the serum of the Australian aborigines, was searched for in the Rongelap population.* Details of these studies are presented in Appendix 16. Samples of sera from 250 Rongelap people were examined between 1958 and 1965. Of these, 237 were consistently negative, 11 were consistently positive (4.4%), and 2 were inconsistent. Family studies indicated that positive subjects were homozygous for the genes. This antigen has been found to be relatively common in some forms of leukemia.

*These studies were carried out by Dr. B.S. Blumberg, Institute for Cancer Research, Philadelphia, Pa.

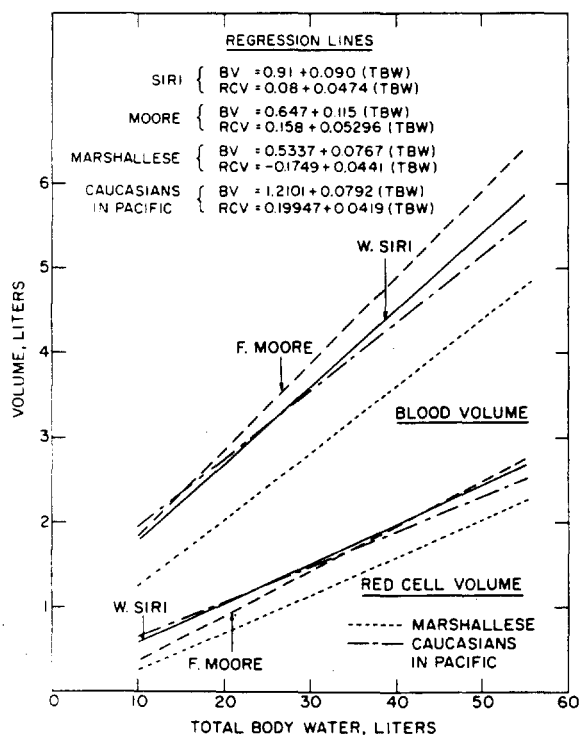


Figure 67.

Since the Rongelap people will be medically examined for many years, it will be interesting to see whether the presence of this antigen is related substantially to disease, particularly leukemia.

ESTIMATION OF INTERNAL BODY BURDENS OF RADIONUCLIDES

In the 1965 survey, the body burdens of radionuclides were determined by use of a portable shadow-shield type of whole-body counter and by radiochemical analysis of 24-hr urine specimens.

Whole-Body Counting

The use of the shadow-shield type of whole-body counter represents a departure from previous surveys, in which a 21-ton steel room had been transported to Rongelap and used for this purpose. Correlations between the two techniques were established by standardizations using the permanent steel room and a duplicate of the shadow shield at Brookhaven National Laboratory. The body ^{40}K values of the Rongelapese provide another means of correlation.

The shadow-shield counter (Figure 68) is very similar to the one described by Palmer and Roesch⁷⁸ and to the Hanford whole-body counter.⁷⁹ It was installed on Rongelap in one of the newly acquired air-conditioned trailers. The detector, an 11½-in.-diameter NaI (Tl) crystal 4 in. thick (Harshaw), is housed in a lead shielding supported by a steel plate about 14 in. above the bed. The subject to be counted lies on a foam rubber cushion in the trough between the two walls of lead bricks, and is moved to a position under the detector by a motor-driven worm-screw drive. The system was calibrated with a plastic phantom man, both in a stationary position beneath the counter and with movement equivalent to the length of the body during the count.

The signal from the detector was picked up by 7 photomultiplier tubes mounted on the crystal, and the gamma-ray spectrum was analyzed with a 400-channel pulse-height analyzer (RIDL). The gamma-ray spectral data were read out on rolls of adding-machine paper for immediate evaluation, and on punched paper tape for subsequent data processing which involved transfer of the data from the punched paper tape to magnetic tape and subsequent analysis in terms of radioisotopes by a spectral stripping program on an IBM-7094



Figure 68. Shadow-shield whole-body counter (gamma spectroscopy) in new trailer, with Marshallese subject being counted.

computer. In this program a library of spectra obtained by calibrations with the plastic phantom is used, but the gamma energy peaks extracted are not limited to those represented in the library. The program used was developed at Los Alamos⁸⁰ for an IBM-704 computer, and has been modified for use on the IBM-7094 and the CDC-6600 computers. Its use in the determination of body burdens of radionuclides has been described by Cohn and Gusmano.⁸¹

As in the previous surveys, both the population exposed to fallout in March 1954 and the unexposed population currently living on Rongelap were counted. An effort was made to count every resident. In addition, some Rongelapese currently living on another atoll, Ebeye, were brought back to Rongelap and included in the survey. Members of the AEC and Trust Territory medical teams were also counted. A total of 179 people were counted in the 1965 survey.

Radiochemical Analysis of Urine

In addition to the whole-body counts, information pertinent to the internal body burdens was obtained by radiochemical analysis of urine specimens for ⁹⁰Sr and ¹³⁷Cs, carried out by the Environment Studies Division, Health and Safety Laboratory, AEC, New York.* Twenty-four-hour urine specimens were collected from 23 residents of Rongelap Island and 5 residents of Ebeye Island. In addition, two pooled urine samples were obtained on Rongelap. The urines were analyzed first for ¹³⁷Cs by gamma-ray spectroscopy by counting the wet ashed sample on top of an 8×4-in. NaI (Tl) crystal. Then the residues were dissolved and analyzed radiochemically for ¹³⁷Cs and ⁹⁰Sr. Calcium was determined by the oxalate-permanganate titration method.

*We are grateful to Mr. Edward P. Hardy for carrying out these analyses.

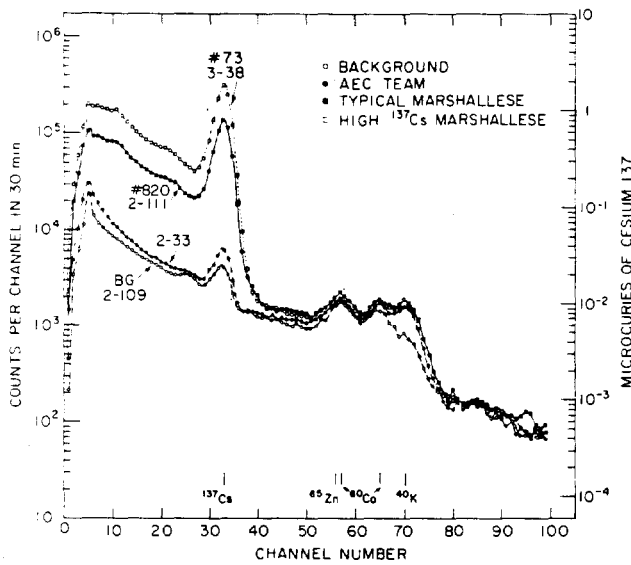


Figure 69. Unprocessed gamma-ray spectrographs obtained with an 11½-in. NaI (TI) crystal in a shadow-shield whole-body counter. The channel width was 20 keV. Traces of ¹³⁷Cs and ⁶⁰Co are apparent in the background spectrum. The right-hand scale was obtained by calibration with a "standard man" phantom and requires correction for other body sizes; it is adjusted for the ¹³⁷Cs peak.

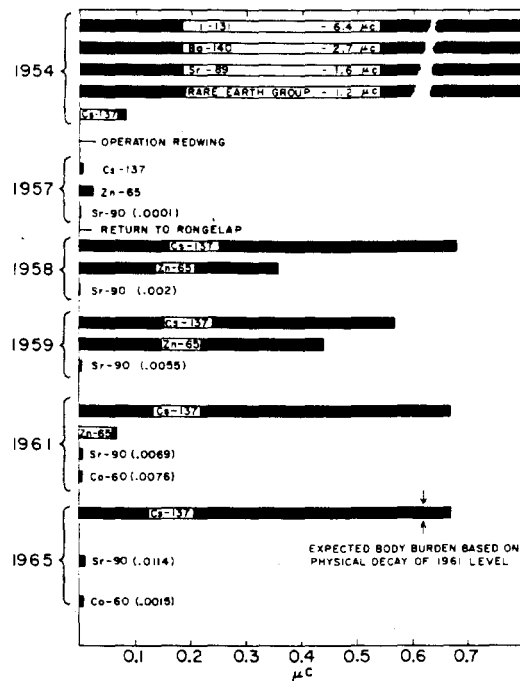


Figure 70. Estimated body burden of isotopes in Rongelap people at various times since 1954. Values obtained either by gamma spectroscopy or by radiochemical analysis of urine.

Table 26
Summary of Radionuclide Body Burdens of Rongelap People, 1965. Obtained by Whole-Body Counting

	People residing on Rongelap Atoll						Rongelapese residing at Ebeve	US AEC medical team	Trust Territory medical team
	Male			Female					
	Age <11	Age 11-15	Age >15	Age <11	Age 11-15	Age >15			
¹³⁷ Cs (nCi/kg body wt.)									
Exposed		14.0	17.1 ± 5.3		16.3	11.6 ± 5.0			
Unexposed	20.4 ± 7.2	19.9 ± 4.3*	14.7 ± 4.9	15.2 ± 5.4	13.9	11.0 ± 3.8			
Mean	20.4	18.1	15.5	15.2	16.1	11.2	0.89	0.16±0.11	0.45
⁶⁰ Co (pCi/kg body wt.)									
Exposed		26.5	40.7 ± 11.5		28.7	40.0 ± 31.3			
Unexposed	30.7 ± 17.6	31.5 ± 7.3	37.6 ± 13.8	27.0 ± 15.6	28.9	31.2 ± 11.2			
Mean	30.7	29.9	38.6	27.0	28.8	34.7	42.6	0.0	0.0
Potassium (g/kg body wt.)									
Exposed		2.19	1.97 ± 0.24		1.92	1.52 ± 0.39			
Unexposed	2.42 ± 0.42	2.09 ± 0.20	1.88 ± 0.32	2.42 ± 0.44	1.81	1.45 ± 0.22			
Mean	2.42	2.12	1.91	2.42	1.86	1.48	1.68	1.50 ± 0.18	1.61
Number of people									
Exposed		5	15		6	18	5		
Unexposed	21	11	30	21	7	27	2		
Total	21	16	45	21	13	45	7	9	2

*Standard deviation = $\sqrt{\frac{\sum(x^2)}{N} - \left(\frac{\sum x}{N}\right)^2}$

Table 27

Marshall Islands Radiochemical Urine Analysis, 1965

Subject No.*	Age	Sex	Volume, ml	g Ca/liter	pCi ⁹⁰ Sr/liter	nCi ¹³⁷ Cs/liter
<u>Rongelap Island</u>						
	Pool	M	5130	0.088	3.6	3.5
	Pool	F	6080	0.058	9.6	2.1
		Mean	5605	0.073	6.6	2.8
8	13	F	730	0.018	8.1	5.0
15	18	F	730	0.008	3.2	6.2
51	36	F	289	0.126	28.0	23.0
53	19	F	650	0.100	10.0	4.4
58	70	F	710	0.104	7.9	3.4
59	45	F	645	0.043	3.1	1.2
11	61	M	940	0.010	1.0	2.1
20	18	M	500	0.096	16.0	6.0
27	37	M	1320	0.103	5.8	5.1
40	40	M	560	0.192	11.0	4.7
41	55	M	890	0.084	6.2	2.2
50	45	M	1800	0.062	3.6	0.6
73	29	M	650	0.243	12.0	19.0
835	31	F	550	0.041	3.5	5.2
843	36	F	1340	0.087	5.3	1.5
928	52	F	1040	0.078	7.6	6.5
932	30	F	350	0.134	24.0	9.7
942	50	F	540	0.238	28.0	18.0
822	18	M	835	0.003	4.5	4.5
833	32	M	1000	0.143	10.8	5.1
840	35	M	800	0.037	6.4	6.0
853	60	M	1750	0.032	4.4	2.0
855	60	M	500	0.152	23.0	3.6
		Mean	831	0.093	10.1	6.3
<u>Ebeve Island</u>						
12	29	F	980	0.057	2.8	0.7
45	43	F	600	0.081	2.1	0.2
84	10	M	719	0.137	4.6	0.3
895	35	F	860	0.194	3.3	0.1
920	33	M	900	0.075	1.2	0.1
		Mean	812	0.109	2.8	0.3

*Subjects with numbers <84 are members of the exposed population; those with numbers >84, of the unexposed population.

Results and Discussion of Body Burden Estimation

Figure 69 shows some representative whole-body gamma-ray spectrographs obtained in the 1965 survey. The AEC team is seen to have a spectrum not markedly different from the background spectrum except for the natural ⁴⁰K peak and a trace of ¹³⁷Cs, whereas the Rongelapese have significantly higher ¹³⁷Cs peaks. Analysis of

the curves indicates that the ⁶⁰Co values are also detectable in many of the Rongelapese. As had been anticipated on the basis of its relatively short physical half-time, the ⁶⁵Zn seen in previous surveys had disappeared by 1965.

Table 26 summarizes the whole-body counting results, and the individual values are presented in Appendix 17. The results of the urine radiochemical analyses are displayed in Table 27. Inspection

of Table 26 shows that the body burdens of ^{137}Cs and ^{60}Co are not significantly different among the various subdivisions by age and sex of the Rongelap resident population. The values given for the children may be overestimates, because the calibrations were in terms of an adult-size phantom only and the counting geometry is such that a higher efficiency obtains for the children, especially the smaller ones.

Prior to 1962, the concentration of ^{90}Sr in the urine was used as a basis for estimating ^{90}Sr body burdens. Subsequently, the ratio of ^{90}Sr to Ca in the urine has been used. On this basis the 1965 mean concentrations of 10.1 pCi ^{90}Sr /liter and 0.093 g Ca/liter correspond to a body burden of 11.4 nCi. Data were not obtained on children in 1965. In previous years the urinary ^{90}Sr to Ca ratios in children have been about twice that for adults. Urines for ^{90}Sr analysis were not collected in 1966.

Comparison of the 1965 survey data with the results from previous surveys (Figure 70) shows that the mean levels of ^{137}Cs and ^{90}Sr have remained nearly constant since 1961, and that ^{137}Cs is at about the 1958 level. Further comparison with the 1954 and 1957 results indicates that the activities now being seen are almost entirely due to intake subsequent to the return of the Rongelap population to their home atoll in 1958.

If the ecological cycle were in equilibrium, it would be expected that, by virtue of physical decay, the specific activities of ^{137}Cs and ^{90}Sr in the food and the corresponding body burdens of these radionuclides would have decreased about 9% during the interim 1961 to 1965. Actually there may have been a small decrease that is masked by the unknown overestimation error in the children. Even with a correction for this, the sustained levels suggest that increasing amounts of the originally contaminated materials are getting into the food chain and thus maintaining the specific activity at the previous levels. This hypothesis is supported by data obtained from E. Held, University of Washington,⁸² to the effect that soil and plant surveys indicate that the ^{137}Cs levels in plants have remained about the same since 1961. There is no ready explanation of the mechanism for this. These findings are to be contrasted with those cited below for an Eskimo population.

Table 28 shows the relationship between ^{137}Cs body burden and its excretion rate for those resi-

Table 28
Comparison of Body Burdens and Excretion Rates

Subject No.	^{137}Cs body burden, nCi	^{137}Cs , nCi/liter	Urine volume, l	^{137}Cs , nCi/day	Fraction/day
8	508.9	5.0	0.730	3.65	0.00717
15	405.6	6.2	0.730	4.526	0.01115
51	484.8	23.0	0.289	6.647	0.01343
53	971.2	4.4	0.650	2.86	0.00294
58	575.0	3.4	0.710	2.414	0.00419
59	732.7	1.2	0.645	0.774	0.00105
11	525.5	2.1	0.940	1.974	0.00375
20	773.9	6.0	0.500	3.000	0.00387
27	1326.0	5.1	1.320	6.732	0.00507
40	1047.0	4.7	0.560	2.63	0.00251
41	1209.0	2.2	0.890	1.958	0.00161
50	728.9	0.6	1.800	1.08	0.00148
73	1861.0	19.0	0.650	12.35	0.00663
835	644.8	5.2	0.550	2.86	0.00443
843	419.8	1.5	1.340	2.01	0.00478
928	491.4	6.5	1.040	6.76	0.01375
932	549.6	9.7	0.350	3.395	0.00617
942	1058.0	18.0	0.540	9.72	0.00918
822	694.6	4.5	0.835	3.758	0.00540
833	636.9	5.1	1.000	5.10	0.00800
840	1453.0	6.0	0.800	4.80	0.00329
853	814.9	2.0	1.750	3.50	0.00429
855	606.2	3.6	0.500	1.80	0.00296
				Mean	0.00553

dents of Rongelap for whom individual urine specimens were analyzed. An average of 0.553% of the body burden is excreted per day, the range being 0.105% to 1.375%. These values correspond to turnover half-times of 120 days, 660 days, and 50 days, respectively. Except for some of the very slow turnover rates, these values fall within the ranges cited or reported for other populations, in which the means were 75, 74, 87, 115, and 135 days.⁸³ Some of the lowest turnover rates in the Rongelap population may be ascribed to uncertainties in the completeness of the urine collections. Thus it is to be expected that the ^{137}Cs value would fall to near-zero levels in a year of living in a noncontaminated environment. That this fall does occur is shown by the results with the Ebeye population, who have been absent from Rongelap for various times from 1 to 18 months.

Because it is not a gamma-ray emitter, ^{90}Sr is not detected by the whole-body counting method. (Theoretically it might be possible to estimate ^{90}Sr

from its bremsstrahlung spectrum, but in practice the complications caused by the presence of gamma-ray emitters and by the size of the human body render this method infeasible.)

Comparison of the excretion rates of ^{90}Sr and ^{137}Cs with the body burdens of ^{137}Cs measured by whole-body counting provides a clue to the ^{90}Sr body burden status. A quantitative value cannot be deduced, however, because the factors relating the behavior of the two nuclides are not sufficiently well established. The study by Hardy, Rivera, and Conard⁸⁴ summarized in Appendix 18 is pertinent to this problem. ^{137}Cs and ^{90}Sr retentions were followed for 190 days after ingestion of representative Rongelap food items by one of the investigators. The ^{137}Cs ingested was almost quantitatively absorbed from the gastrointestinal tract, but 50% of the ^{90}Sr was excreted via the feces in the first 10 days and may be regarded as not having been absorbed. By the end of the study almost all of the ^{137}Cs and about 75% of the ^{90}Sr had been excreted in the urine and feces. The exact figure for retention is dependent on the correction used for activities ingested in the normal diet, and these were not measured. In this study the biological half-time for ^{137}Cs was estimated as 74 days. For both ^{137}Cs and ^{90}Sr the urinary excretion rates were markedly elevated during the ingestion period and for a few days afterward. This confirms other results to the effect that the excretion rates found are strongly affected by the recent diet and, when there has been a recent intake of high activity food, the excretion rates do not provide accurate indices of the body burdens.

Comparison of ^{137}Cs Levels in Marshallese and Alaskan Eskimos

It is of interest to compare the ^{137}Cs body burden findings in the Rongelapese with those reported for certain Alaskan Eskimos. The findings in the Eskimo population in August 1965 are shown in Table 29.⁸⁵ It may be noted that the results for adult Eskimos are equal, within statistical limits, to those for the adult male Rongelapese. For the Eskimo population the 1965 data run about 30% lower than the comparable 1964 data, and this trend parallels the findings in the local caribou, one of the principal components of their diet.⁸⁵ The *Federal Radiation Council Radiation Protection Guides*⁸⁶ is cited as recommending a limit of 3000 nCi ^{137}Cs in individual adults for this population,

Table 29

Cesium-137 Body Burdens
for Residents of Anaktuvuk Pass, Alaska⁸⁵

Age, yr	No. persons	Body burden, nCi	nCi per kg body wt.
>21	23	920±58	15.7±1.0
15-20	5	490±42	9.5±1.0
3-14	22	170±17	6.2±0.3
>21*	16	900±60	16.0±1.3

*"Controls" - members of a group who were first counted in 1963 and are re-examined periodically.

and opinions are expressed to the effect that the current body burdens do not constitute a radiological health hazard.⁸⁵ These opinions are consistent with those that have governed the policies applicable to the Rongelapese.

Radiochemical Analysis of Coconut Crabs

A food item that has been of special interest throughout the Rongelap medical surveys is the coconut crab (*Birgus latro*). Because of its high ^{90}Sr content, it has been banned as a food. The following concentrations of ^{90}Sr and ^{137}Cs were found in a crab taken from Rongelap Island in 1965 (radiochemical analysis by the Health and Safety Laboratory): ^{90}Sr , 66,600 pCi/kg original matter; ^{137}Cs , 12,700 pCi/kg original matter; stable Ca, 92.1 g/kg original matter.

Previous analyses of crabs taken from Rongelap Island have been reported in the 7, 8, and 9-10 year reports.⁸⁻¹⁰ For ^{90}Sr the results have run: at 7 years 1140 pCi ^{90}Sr /g Ca; 8 years 1317, 1086, 1113, and 1378 pCi ^{90}Sr /g Ca; and 9-10 years 865, 628, and 780 pCi ^{90}Sr /g Ca, and 39,292, 45,318, and 66,234 pCi ^{137}Cs /kg. The 1965 results for ^{90}Sr are slightly below the average for the previous survey and for ^{137}Cs are reduced to about 1/4 the previous results. All these results are markedly lower than those that have been obtained with crabs from the more heavily contaminated islets of Eniatok and Kabelle (Figure 71).

The crab data may be compared with the data on Rongelap subject No. 73, who had the highest ^{137}Cs body burden (25,400 pCi/kg) and was excreting activity in concentrations of 78,000 pCi ^{137}Cs /g Ca and 50 pCi ^{90}Sr /g Ca. It seems clear from this that because of its relatively high ^{90}Sr content the

very different from those of the 11-year survey, and no untoward changes are apparent.

Ailingnae Population

The mean blood counts in the 14 Ailingnae people at 11 years were about the same as in the unexposed comparison population, except that platelet levels were higher. At 12 years the counts

in this group were similar to those in the exposed Rongelap group. Appendix 9 summarizes the Ailingnae counts since 1954.

Utirik Population

The Utirik population, examined in 1966, had mean leukocyte counts and differential counts not very different from those of 3 years previously. No

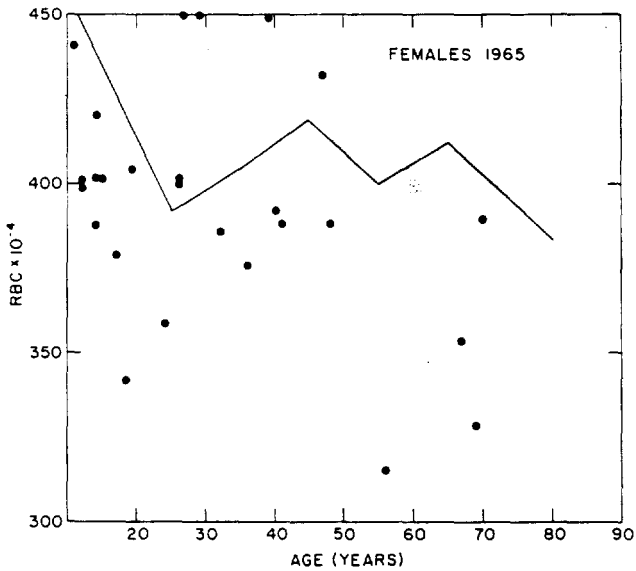


Figure 59. RBC values of Rongelap exposed females plotted against age. Solid line represents mean level of unexposed female population, 1965.

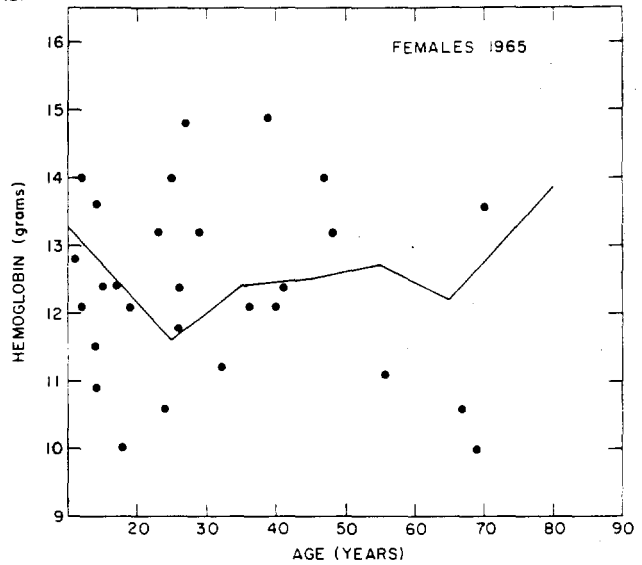


Figure 61. Hemoglobin values of Rongelap exposed females plotted against age. Solid line represents mean level of unexposed female population, 1965.

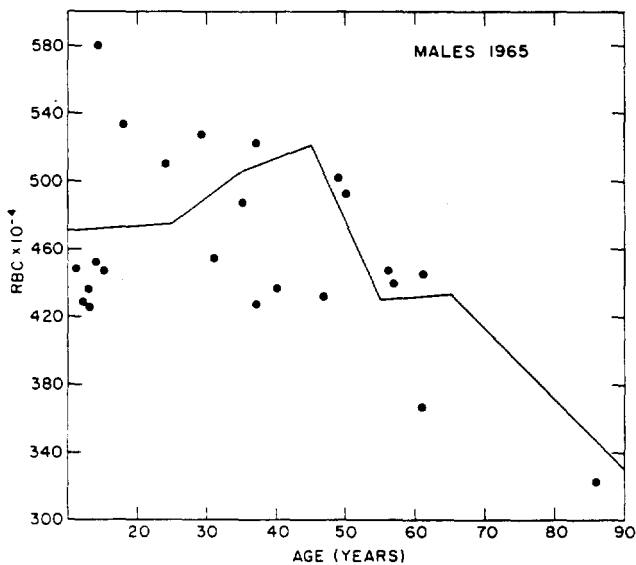


Figure 60. RBC values of Rongelap exposed males plotted against age. Solid line represents mean level of unexposed male population, 1965.

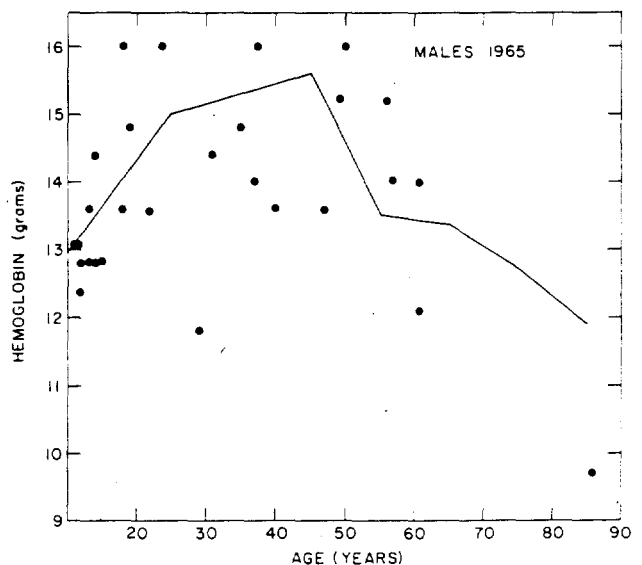


Figure 62. Hemoglobin values of Rongelap exposed males plotted against age. Solid line represents mean level of unexposed male population, 1965.

use of crab meat as a food item should continue to be avoided. Other foods, however, are responsible for the ^{137}Cs levels found.

Summary

Medical surveys were carried out on the exposed people of Rongelap Island in March 1965 and March 1966. In 1965 the comparison population was also examined, and in 1966 the exposed population of Utirik Island was examined.

INTERVAL MEDICAL HISTORY

During the past 2 years the Rongelap people have been generally in good health with satisfactory nutritional status. No unusual epidemics of disease occurred. Over the 2-year period 2 deaths had occurred in the Ailingnae group (originally exposed to 69 rads). No autopsy was obtained, but the deaths occurred in older people with no obvious connection with radiation exposure. One death occurred in an older woman of the comparison population. The death rate has been higher in the exposed population than in the unexposed population, which may in part be related to the larger percentage of older people originally in the exposed group. Birth rate has been about equal in the exposed and unexposed groups. No miscarriages or stillbirths were noted in the exposed

women during the past 2 years, but 5 miscarriages occurred in unexposed women. No abnormal babies were born in the exposed group, but two (hydrocephalic and mongoloid) were born to two unexposed women.

The people of Utirik Island appeared to have been in good health and reported few serious medical happenings over the past 3 years. The birth and death rates in this group appeared to be similar to those in the Rongelap group.

PHYSICAL FINDINGS

As noted in previous surveys, the variety and distribution of physical abnormalities (with the exception of thyroid pathology) did not appear to be significantly different in the exposed people as compared with the unexposed population. The trends toward growth retardation in the exposed children, previously noted, have continued. No leukemia or cancer (except one thyroid cancer) was detected.

THYROID FINDINGS

The dosimetric calculation for radiation dose to the thyroid from internal absorption of radioactive iodines from the fallout was reviewed. Early thyroid studies including protein-bound iodine determinations, iodoprotein levels, dietary iodine and urinary excretion of iodine, thyroid uptake of radioiodine, and serum cholesterol levels were reviewed.

Thyroid abnormalities during the past 3 years have increased to 18 cases, 16 with nodules and 2 with hypothyroidism. It is noteworthy that in the higher exposure group thyroid abnormalities occurred in 79% of the children exposed at <10 years of age, as compared with no cases in the children of the Ailingnae, Utirik, or unexposed groups. Only one adult in the Ailingnae group developed a thyroid nodule. Several nodules were noted in the thyroid glands of older Utirik and unexposed adults of Rongelap. Surgery was performed in 11 cases: 9 children and 1 adult were found to have adenomatous goiters, and 1 adult a mixed papillary and follicular carcinoma of the thyroid gland with localized metastasis. The gross and microscopic appearance of these lesions was described and depicted. Correlation of growth retardation in exposed children with thyroid pathology was indicated by the recent finding of definite

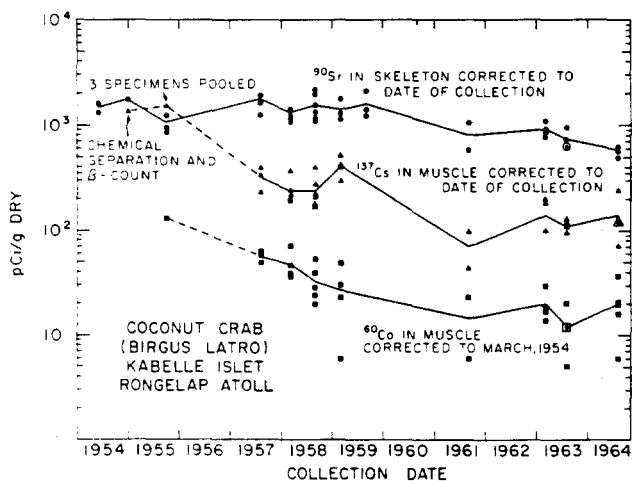


Figure 71. Analyses for ^{90}Sr , ^{137}Cs , and ^{60}Co in coconut crabs of northern Rongelap Atoll since 1954. (Courtesy of E. Held, University of Washington.)

hypothyroidism in the two most growth-retarded boys with subsequent growth response to thyroid hormone treatment. Thyroid function tests in cases with thyroid abnormalities also indicated reduced thyroid function in some other children. Preliminary indications are that thyroid hormone treatment instituted in the exposed population in September 1965 is having a beneficial effect in reducing thyroid nodules and stimulating growth of children. The importance of these thyroid developments in the Marshallese is discussed.

OTHER STUDIES

Hematological studies show that the leukocyte and platelet levels of the peripheral blood of the exposed Rongelap people seem to have recovered to levels equal to those of the unexposed population. The blood levels of the Utirik population showed no obvious abnormalities and were similar to those previously noted.

Chromosome studies of white cells of 51 peripheral blood cultures showed a low incidence of 2-hit aberrations in the exposed people which was quite significant in view of the absence of any such aberrations in the comparison population.

Blood volume and red blood cell volume studies showed that people of Rongelap have significantly reduced levels as compared with Americans. Caucasians living in the Marshall Islands did not generally show this phenomenon.

Aging studies have been repeated with the addition of several new parameters (vibratory sense, light extinction test, body potassium levels, and blood serum cholesterol) and with more refined statistical analysis. Curves of response for each aging parameter and an over-all biological age score curve are presented. No significant increase in aging effects in the exposed population compared with the unexposed population could be detected. The over-all correlation of the biological age scores with chronological age was good. It was concluded that the tests employed may not have been sensitive enough to detect subtle effects of aging at this exposure level.

ESTIMATION OF INTERNAL BODY BURDENS OF RADIONUCLIDES

In the 1965 survey whole-body counting was done on 179 people with an 11½-in.-diameter

NaI (Tl) crystal in a portable shadow-shield type of counter. The Rongelap residents exposed in 1954 and the unexposed residents were indistinguishable by this method, both groups showing elevated ¹³⁷Cs body burdens of 10 to 20 nCi/kg body weight, or slightly less than 1 μCi total body burden and about 1/1000 this much for ⁶⁰Co body burdens. These levels are about the same as or slightly lower than those reported previously for the 1961 survey. Urine specimens were also analyzed radiochemically for ⁹⁰Sr and ¹³⁷Cs. The ⁹⁰Sr levels remain nearly the same as those found in 1961. In a comparison of the ¹³⁷Cs excretion rate with the body burdens, a mean fractional excretion rate of 0.00553/day was found.

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APPENDIX 1

The following bibliography represents a compilation of reports on the Marshallese people exposed to radioactive fallout in March, 1954. This list includes all known medical reports as of March, 1966. In many of these reports the Marshallese population is reported on only as one of several populations under study. In addition, certain radioecological studies (largely by the Laboratory of Radiation Biology, University of Washington, Seattle) carried out at Rongelap are presented as a supplementary bibliography. A third section of the bibliography comprises reports on the Japanese fishermen who were also exposed to radioactive fallout while on their boat, the Lucky Dragon, in 1954.

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APPENDIX 2 - UCRL 12273

Estimate of Radiation Dose to Thyroids of
the Rongelap Children Following the Bravo Event

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Livermore, California

December 16, 1964

General Information

The cloud arrival time is given⁽¹⁾ as H + 4 to 6 hours. The duration of the cloud passage is less well known but probably lies in the range of 8 - 16 hours. In all calculations we will assume that cloud passage was in the interval H + 6 to H + 18.

The residents of Rongelap were evacuated at H + 51 hours. Reliable dose rate measurements were not obtained at that time but the gamma dose rate 3 feet above the ground, was measured as 375 mr/hr seven days after the detonation. Assuming $t^{-1.2}$ decay the H + 24 hour dose rate was then ~3.8 rad/hr.

The sources of exposure to the thyroid which must be considered are:

- (1) Whole body gamma dose.
- (2) Internal deposition of iodine isotopes.

Whole Body Gamma Dose

The whole body dose was estimated⁽¹⁾ to be 175 rad. The exact method of making this estimate is not given, so an independent estimate is made below. In particular, it appears that this estimate does not include the dose from the cloud but only from fallout.

If we assume a linear buildup of fallout from H + 6 to H + 18, $t^{-1.2}$ decay during this interval and use the reading of 375 mr/hr at seven days, the estimated dose from fallout during cloud passage is 47 rad. The dose from the fallout from H + 18 to evacuation at H + 51 is 114 rad. Experience from Sedan indicates that the dose from the cloud, itself, is approximately equal to the dose from fallout during cloud passage. The total estimated dose is then $47 + 47 + 114 = 208$ rad.

Within the error of the measurements and the accuracy of the assumptions, this estimate, which does not contain any correction for the small effect of time indoors, does not differ significantly from the value of 175 rad⁽¹⁾. We will, therefore, take the average whole body gamma dose as 175 ± 25 rad.

Internal Deposition of Iodine Isotopes

Unfortunately, no direct measurement was possible on the radioactive iodine content of individuals from Rongelap. Urine samples were taken from which the average thyroid burden of ^{131}I has been estimated. The Los Alamos Scientific Laboratory (2) collected pooled 24-hour samples 15 days post detonation and estimated the 1-day thyroid content as $11.2 \mu\text{c}$ of ^{131}I . USNRDL collected samples from each member of the exposed group 43 and 46 days post detonation and, by an indirect method, estimated the average thyroid content as $6.4 \mu\text{c}$ ^{131}I at 1 day. (3,4) The LASL estimate of $11.2 \mu\text{c}$ was obtained by direct counting of ^{131}I in the urine and should be more reliable than the NRDL estimate. The value of $11.2 \mu\text{c}$ will be used as a basis for all following considerations. This estimate was based on the assumption of 0.1% of the maximum thyroid burden being excreted in the urine on the 15th day. Variation in the biological half-life and other factors indicate that a range of 0.05 - 0.2% should be placed on this number (5) (see following section by Ng). We, therefore, take $5.6 - 22.4 \mu\text{c}$ as the range of adult ^{131}I thyroid burden.

The pooled samples represent all age groups. The number of individuals in these age groups and the volume of urine from each age group is approximately as follows: (3,4)

<u>Age Group</u>	<u>Number of Individuals</u>	<u>Volume of Urine(ml)</u>	<u>% of Total Volume</u>
< 5	7	1155	4.8
5 - 16	11	4829	20.1
> 16	31	18011	75.0

The urine samples are typical of adults and the calculated thyroid burdens are presumably also those of adults.

Associated with this ^{131}I are the shorter lived isotopes ^{132}I , ^{133}I and ^{135}I . If the iodine entered by way of inhalation, the time of intake was H + 6 to H + 18. On the other hand, if water (and food) were the principal source, the time of ingestion would be extended from H + 6 to H + 51.

Three items contribute to the differences in dose from the various iodine isotopes. These are: (1) radioactive decay before inhalation or oral ingestion, (2) differences in the fission yields of the chains, (6) and (3) the average energy deposited in the thyroid per disintegration. These factors are presented in Table I for ^{133}I and ^{135}I . In the case of inhalation, uniform distribution in the cloud was assumed. For oral ingestion it was assumed that, on the average, 1/3 of the intake occurred at H + 10 and 2/3 at H + 30.

Table I

Ratio of Doses for the Two Modes of Intake

	Inhalation		Oral Ingestion	
	$^{133}\text{I}/^{131}\text{I}$	$^{135}\text{I}/^{131}\text{I}$	$^{133}\text{I}/^{131}\text{I}$	$^{135}\text{I}/^{131}\text{I}$
Decay	0.68	0.31	0.487	0.148
Fission yield ⁽⁶⁾	1.38	1.23	1.38	1.23
Energy	2.00	1.50	2.00	1.50
Net Factor	1.85	0.57	1.35	0.27

The dose to the thyroid in rads from all three isotopes is, thus, 3.4 times the dose due to ^{131}I alone for inhalation and 2.6 times the ^{131}I dose for oral ingestion. Delay in reaching the thyroid after inhalation or ingestion would lower these factors somewhat. However, the ^{132}I daughter of the 78 hr ^{132}Te has been neglected and would approximately compensate for decay of ^{133}I and ^{135}I before reaching the thyroid.

We can now proceed to estimate the dose to the thyroids of 3 - 4 year old girls assuming (1) inhalation as the mode of intake and (2) oral ingestion.

1. Inhalation: The ratio of volume of air respired by a 3 - 4 year old girl to that of an adult can be estimated in two ways: (a) from the maximum rate of oxygen intake⁽⁷⁾ and (b) from the vital capacity⁽⁸⁾ and maximum respiration rate. Both methods give a ratio of ~ 0.3 . The thyroid burden of these children would then be ~ 3.4 μc with a range of 1.7 - 6.8 μc .

Assuming the Rongelap children are similar to those of New York children, the mass of the thyroid of the children is 2.5 ± 0.6 grams.⁽⁹⁾

The most probable dose from ^{131}I is then 150 rad and the dose from all isotopes is 510 rad. If we consider the range of thyroid burden (1.7 - 6.8 μc) and the variation in thyroid weight (1.9 - 3.1 gms), the dose is in the range of 200 - 1350 rad.

2. Oral Ingestion: At the time of the event the Rongelap people were on a water ration of one pint per day. They were warned not to drink water after the event but most of them admitted they drank water anyway.⁽¹⁰⁾ The method of collecting water by runoff from roofs into cisterns makes it very likely that this was the main source of oral ingestion. There are reports that it "rained a little" on the afternoon of March 1 (D-Day). The village doctor reported that the "water turned yellow." As far as food is concerned, the most likely source is dried fish. Fish were dried on open racks. However, in the interviews none of them listed dried fish as having been eaten during

the time before evacuation. (10) Under these circumstances it is reasonable to assume that children drank the same amount of water and, therefore, had the same intake as adults; i.e., their thyroid burdens were also 11.2 μC of ^{131}I (range 5.6 - 22.4 μC).

The most probable dose from ^{131}I is then 490 rad and the total dose 1270 rad. Considering a range in the thyroid burden (5.6 - 22.4 μC) and a thyroid weight range of 1.9 - 3.1 gms, the range of total dose is 520 - 3300 rad.

Incidentally, LASL assumed this mode of intake and calculated a dose of 150 rad. (2) The thyroid weight used was not given but was probably 20 grams. We would calculate 160 rad, in very good agreement with the LASL estimate.

Summary

Thyroid Dose (Rads) to Rongelap Girls Ages 3 - 4

	Inhalation			Oral Ingestion		
	<u>Min</u>	<u>Max</u>	<u>Most Probable</u>	<u>Min</u>	<u>Max</u>	<u>Most Probable</u>
Whole Body	150	200	175	150	200	175
Radioiodine	200	1350	510	520	3300	1270
Total	350	1550	685	670	3500	1445

The actual intake was undoubtedly a combination of the two modes of intake. The most probable dose is, therefore, in the range 700 - 1400 rad.

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Calculation of Urinary Radioiodine Excretion

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Radioiodine appearing in urine, except for that during a relatively short period following exposure, originates from the thyroid. In the calculation for urinary radioiodine it was assumed that iodine is released from the thyroid only as thyroxine, and that the release of thyroxine and its subsequent degradation in the extrathyroidal hormonal space can be adequately described assuming first order kinetics. Ranges for normal biological half-life of iodine in the thyroid and normal turnover rate of extrathyroidal thyroxine were selected from the best available data in the literature. The uptake of radioiodine was assumed to be exponential with a half-period of increase of 4.5 hours, and 60% of the iodine released when extrathyroidal thyroxine is degraded was assumed to be excreted in urine.

Rates of urinary radioiodine excretion were calculated from the resulting expression shown below.

$$\frac{dI_U}{dt} = 0.60LKI_{Tf} \frac{e^{-(K+\lambda)t} - e^{-(L+\lambda)t}}{L-K} - \frac{e^{-(J+K+\lambda)t} - e^{-(L+\lambda)t}}{L-J-K}$$

I_U = radioiodine content of urine

L = rate of turnover of extrathyroidal thyroxine

K = rate constant for the release of iodine from the thyroid

I_{Tf} = peak radioiodine content of the thyroid

λ = physical decay constant for ^{131}I

J = rate constant for the uptake of radioiodine in the thyroid

t = time

A summary of the calculations made to determine the normal range of urinary radioiodine excretion at 15 days appears below.

Urinary Radioiodine Excretion at 15 Days

J day ⁻¹	K day ⁻¹ x 10 ³	L day ⁻¹ x 10 ²	$\frac{dI_U}{dt}$ % of I_{Tf} /day
3.7	4.85	7.2	0.050
3.7	4.85	13.8	0.066
3.7	17.15	7.2	0.16
3.7	17.15	13.8	0.21

On the basis of these calculations the normal range of urinary radioiodine excretion at 15 days was estimated to be 0.05 to 0.2% of the peak thyroid content.

The problem outlined above is a practical application of an analytical study on the uptake and excretion of iodine in man which will be described more fully in a UCRL report entitled, "The Dynamics of Iodine in Man."

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APPENDIX 3

Individual Serum Iodine Determinations of Marshall Island Populations Under Study (in $\mu\text{g}\%$)

	March 1958			March 1959			March				March 1965				Sept. 1965	March 1966				Sept. 1966	
	PBI	TI	BEI	PBI	TI	BEI	1961	1962	1963	1964	PBI	TI	Ip*	T ₃ +T ₄	PBI	PBI	TI	Ip	T ₃ +T ₄	PBI	
	<u>Rongelap Exposed</u>																				
1	10.6	10.6						12.0	9.4												
2							8.0		10.2**				7.9		5.2						2.6
3							8.8	7.2	1.9**				6.2 7.1 2.0 4.2		3.2						1.0
4	8.6	9.8											1.4								
5								4.6	2.5**				1.6		0.8						
6													1.9		2.5	3.1				1.8	2.1
													1.8								
6							10.7	9.7	7.9				8.5		Pre TSH 7.2	5.0					3.6
									8.7**						Post TSH 7.9						
7																	5.0	1.7			3.0
8									9.0**				5.7		5.4						4.0
10	11.8	11.8		7.0	5.0	5.0		9.5	12.0				8.3								
11	10.6	10.6						11.0					8.4								
12	6.6	7.8							8.8												
13													6.9					7.8	4.0		3.9
														2.8							
14								10.8	8.3				7.1								

* Iodoprotein.

** Determinations made in 1966 from frozen sera.

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	March 1958			March 1959			March				March 1965				Sept. 1965	March 1966				Sept. 1966
	PBI	TI	BEI	PBI	TI	BEI	1961	1962	1963	1964	PBI	TI	Ip*	T ₃ +T ₄	PBI	PBI	TI	Ip	T ₃ +T ₄	PBI
	<u>Rongelap Exposed (continued)</u>																			
47	8.4	10.6						7.9**												6.2
48	8.8	8.8																		
50	9.2	15.0									10.1									
51	8.0	8.8									9.7									
53	8.2	8.4									8.5									
54									7.8**		8.3				5.0				4.3	
55											5.7		3.8			6.9	2.5	3.3		
																	1.5			
58	8.6	10.4									9.2									
59	9.0	10.2						9.8**	11.0**		8.6			11.1		8.6	2.7	4.5		
												8.2	2.8	5.3		6.4		5.0		
60									10.8**											
61	11.4	11.4							11.5**		6.4					8.3			4.5	
																7.5			4.9	
64								10.7**	12.0**		10.0						5.0	2.9	2.0	
												8.6	4.5	3.4						
											7.5									
65							7.1	6.7	8.8**		7.0				Pre TSH 6.4	3.1			1.9	
															Post TSH 7.6	5.1			3.6	
66											8.0									
67											6.6									
68											7.1		4.6			11.8	6.0	5.8		
																	2.7			
69	12.2	13.2						7.8**	8.7**	10.2	7.1					5.7			1.7	2.8
70									8.7				1.9							
71									8.7											
72	8.2	8.6														5.3				

March 1958			March 1959			March				March 1965				Sept. 1965	March 1966				Sept. 1966
PBI	TI	BEI	PBI	TI	BEI	1961	1962	1963	1964	PBI	TI	Ip*	T3 + T4	PBI	PBI	TI	Ip	T3 + T4	PBI

Rongelap Exposed (continued)

73													8.4							
74	10.4	11.6	6.2																	
75													4.7							4.5
76	7.4	8.4											4.4	4.4						5.6
81	7.0	7.6																		
82	8.6	9.2																		
83						8.1	7.4						6.6							
84												9.3			6.2					3.6
86						12.0	9.2	8.2							7.1					6.0

Rongelap Unexposed

814													5.7							4.3
815	14.6	15.2	5.8	5.3	3.3								6.3							
816	9.2	9.4																		
817	9.6	10.2																		
818	11.6	12.2	6.5	9.2	7.5								6.5							
820	10.2	10.8											8.8							
821	8.0	8.8																		
822	9.8	10.4											5.2							
829							8.6	7.1						5.1	1.1	3.5				
830	9.2	9.8		6.0	4.4															
833													7.3							
835													7.2							
														6.4	2.3	4.2				

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March 1958		March 1959			March				March 1965				Sept. 1965	March 1966				Sept. 1966	
PBI	TI	BEI	PBI	TI	BEI	1961	1962	1963	1964	PBI	TI	Ip*	T ₃ + T ₄	PBI	PBI	TI	Ip	T ₃ + T ₄	PBI

Rongelap Unexposed (continued)

843										7.0										
											6.7	3.2	3.2							
849										6.5										
											6.9	2.4	4.1							
853	11.2	11.8								7.0										
855	9.6	10.0								7.0										
861	12.0	15.6																		
862	7.6	7.6																		
863	10.0	10.6		6.7	4.9															
864										10.7										
865							9.1	8.2		9.4										
867	14.0	14.0																		
869	10.2	11.2		5.9	4.7															
871	12.0	12.2																		
872	7.2	9.2																		
873	10.0	10.8																		
874	7.8	8.0		5.2	4.0															
882													7.7							
887	10.4	11.2		6.2	5.0															
891	10.4	11.0																		
892	7.6	9.2																		
893	16.4	17.4																		
913										6.9										
											6.8	1.8	5.1							
928										7.6										
											7.3	2.2	4.8							

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March 1958			March 1959			March				March 1965				Sept. 1965	March 1966				Sept. 1966		
<u>PBI</u>	<u>TI</u>	<u>BEI</u>	<u>PBI</u>	<u>TI</u>	<u>BEI</u>	<u>PBI</u>	<u>PBI</u>	<u>PBI</u>	<u>BPI</u>	<u>PBI</u>	<u>TI</u>	<u>Ip*</u>	<u>T₃</u> +	<u>T₄</u>	<u>PBI</u>	<u>PBI</u>	<u>TI</u>	<u>Ip</u>	<u>T₃</u> +	<u>T₄</u>	<u>PBI</u>

Rongelap Unexposed (continued)

938									5.6	3.9													
942																							
943																							
944									2.0														
950									6.7														
955																							
959																							
961																							
980																							
982																							
1005																							
1043																							

Utirik Exposed

2101																								
2105																								
2114																								
2115																								
2119																								
2125																								
2128																								
2136																								
2137																								
2139																								

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2142

2146

2150

2152

2155

2166

2169

2178

2193

2196

2197

2215

2216

2251

March 1958			March 1959			March				March 1965				Sept. 1965	March 1966				Sept. 1966	
PBI	TI	BEI	PBI	TI	BEI	1961	1962	1963	1964	PBI	PBI	TI	Ip*	T ₃ + T ₄	PBI	PBI	TI	Ip	T ₃ + T ₄	PBI

Utirik Exposed (continued)

8.9

12.7

11.5 8.1 2.8

10.5

26.7

8.4

13.1

12.7 7.8 3.7

10.7

8.5

8.1

9.6

8.4

9.8

8.6

9.6

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APPENDIX 4

Urinary Iodine Excretion

<u>Subject No.</u>	<u>Age</u>	<u>Sex</u>	<u>Wt. (kg)</u>	<u>24-hr. Output (ml)</u>	<u>Total I ($\mu\text{g/ml}$)</u>	<u>$\mu\text{g/Day}$</u>	<u>Subject No.</u>	<u>Age</u>	<u>Sex</u>	<u>Wt. (kg)</u>	<u>24-hr. Output (ml)</u>	<u>Total I ($\mu\text{g/ml}$)</u>	<u>$\mu\text{g/Day}$</u>
8.	14	F	41	775	0.048	37.2	73	30	M	71	660	0.270	178.2
11	62	M	52	970	0.058	56.3	84	11	M	29	730	0.096	70.1
12	30	F	62	1000	0.130	130.0	822	19	M	57	850	0.106	90.1
15	19	F	52	735	0.090	66.1	833	33	M	64	1050	0.188	197.4
20	19	M	54	410	0.118	48.4	835	32	F	51	560	0.062	34.7
27	38	M	65	1355	0.102	136.2	840	36	M	68	835	0.128	106.8
40	41	M	55	520	0.220	114.4	843	37	F	50	1350	0.030	40.5
41	56	M	54	900	0.056	50.4	853	61	M	69	1770	0.082	145.1
45	44	F	55	610	0.100	61.0	855	61	M	66	515	0.150	82.4
50	46	M	84	1830	0.104	190.3	895	36	F	67	820	0.216	177.1
51	37	M	44	665	0.402	279.3	920	34	M	62	910	0.084	76.4
53	20	F	49	300	0.252	75.0	928	53	F	57	1080	0.152	164.2
58	71	F	55	715	0.148	105.8	932	31	F	50	355	0.128	45.4
59	46	F	39	650	0.030	19.5	942	51	F	60	545	0.300	163.5
											Mean	0.138	105.0

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APPENDIX 5

Cholesterol Levels (mg%)

<u>Subject No.</u>	<u>1957</u>	<u>1959</u>	<u>Subject No.</u>	<u>1957</u>	<u>1959</u>	<u>Subject No.</u>	<u>1957</u>	<u>1959</u>
			<u>Rongelap Exposed</u>					
1	173	204	30	183	---	57	249	---
2	154	148	31	95	---	58	113	---
3	166	---	32	127	---	59	99	---
4	270	184	33	106	---	60	249	---
5	181	---	34	183	---	61	277	---
6	190	---	35	109	---	62	305	---
7	190	---	36	123	149	63	159	150
9	276	---	37	88	---	64	249	---
10	274	---	38	215	---	65	144	---
11	203	155	39	106	---	66	144	---
12	177	---	40	129	---	67	167	---
13	195	140	41	109	---	69	191	---
14	228	207	42	101	---	69	136	---
15	---	135	43	167	---	70	105	132
16	232	162	44	159	---	71	191	---
17	136	---	45	191	---	72	167	137
18	234	149	46	232	---	73	125	---
19	141	---	47	167	---	74	207	---
20	162	---	48	96	121	75	128	---
21	129	---	49	175	---	76	152	134
22	144	---	50	113	---	77	191	---
23	88	141	51	95	---	78	183	165
24	152	---	52	167	182	79	159	127
26	136	---	53	102	132	80	259	---
27	136	---	54	121	---	81	167	152
28	183	---	55	144	143	82	224	---
29	268	---	56	253	---	83	152	---

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<u>Subject No.</u>	<u>1957</u>	<u>1959</u>
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825	---	163
831	259	---
832	152	---
833	159	---
834	175	---
835	136	---
836	183	155
838	215	---
839	191	---
840	232	---
841	224	213
842	305	---
843	191	181
844	191	149
845	183	---
846	199	---
847	167	---
848	224	---
849	277	---
850	215	164
851	249	237
852	183	162
853	207	---
854	183	192
855	249	208

<u>Subject No.</u>	<u>1957</u>	<u>1959</u>
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Rongelap Unexposed

856	249	172
857	232	---
858	259	180
859	249	202
860	379	187
861	325	173
862	305	---
864	191	170
865	159	---
867	167	---
868	191	207
869	224	---
871	158	157
872	192	---
873	174	187
874	135	---
875	227	181
876	142	154
878	200	---
879	192	---
880	132	---
881	200	---
882	158	---
883	149	---
884	265	---

<u>Subject No.</u>	<u>1957</u>	<u>1959</u>
--------------------	-------------	-------------

885	122	---
886	167	---
887	127	---
888	200	---
889	183	150
891	142	---
893	158	---
894	183	152
895	158	146
896	218	165
913	---	136
916	---	135
925	---	129
928	---	239

APPENDIX 6

Hospital Summaries of Thyroid Surgical Cases

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
17	14	F	U.S. Naval Hospital, Guam	August, 1964

This 14 year old female, a native of Rongelap Atoll, was referred here for management of a palpable nodule in the left lobe of the thyroid, recently discovered, associated with no symptoms suggestive of dysfunction of the thyroid gland. Rongelap was inadvertently irradiated by fallout from an atomic bomb blast in 1954, when this child was 4 years of age. After the fallout she had developed an anemia, a leukopenia, and second degree burns, all of which lasted for several months.

Admission work-up was within the limits of normal except for the presence of a soft, 1.2 cm nodule in the left lobe of the thyroid, near the isthmus.

Laboratory studies including chest x-ray were within the limits of normal.

She was admitted on 8/17/64, and a total thyroidectomy performed on 8/19/64, with dissection of the anterior compartment of the neck. Postoperatively the patient did well so far as healing of the wound was concerned, but within 24 hours she showed a suggestion of neuromuscular hyperirritability. On that same day a serum calcium was reported as 8.5 with a phosphorus of 4.3. She was begun on oral vitamin B₂ and calcium lactate tablets. A few days later aluminum hydroxide gel was added as another oral medication, and a high calcium, low phosphorus diet was begun. Thereafter the patient began rapidly to show evidence of regeneration of parathyroid function of her own, so that a gradual weaning of medications was begun. By 9/16/64 she was off all medication except the diet and amphojel. These were discontinued on 9/23/64, and when her serum calcium and phosphorus remained normal she was discharged on 10/1/64. Throughout her hospital stay after the completion of her thyroidectomy she was maintained on thyroid extract gr. 1 - 3 times daily. This is believed necessary for replacement therapy, and it is recommended that this be continued indefinitely.

Microscopic examination of the removed thyroid was strongly suggestive of the suspected malignancy. However, review of the slide by the AFIP yielded a final diagnosis of nodular thyroid goiter with no evidence of malignancy.

Follow-Up: Since discharge from the hospital the patient has remained asymptomatic on thyroxine therapy. In March, 1966, after being off thyroid hormone for three weeks (temporarily for test purposes), her TSH level was elevated to 342 mμg/ml and ¹³²I uptake nil, indicative of athyroid status. She had maintained a euthyroid status on 3 mg levothyroxine daily and has shown no recurrence of nodules.

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
21	12	F	U. S. Naval Hospital, Guam	August, 1964

This 12 year old female, native of Rongelap Atoll, was accidentally subjected to radioactive fallout from a nuclear explosion in 1954. She subsequently suffered hematopoietic depression and "beta burns of the skin that healed in several months." Thereafter, she was subjectively and objectively in good health until March, 1964, when examination revealed a grossly irregular (nodular) thyroid gland. Protein bound iodine was reported as normal, and the child was referred to this hospital on August 17, 1964, for surgical management of the pathologic gland.

Admission studies revealed a normal physical examination except for four to five hard nodules in the thyroid, with no palpable lymph nodes. Laboratory studies were negative except for an unexplained leukocytosis of 15,700 (with a normal smear) and a sedative rate of 38. PBI was not repeated.

The increased likelihood of malignant degeneration in thyroid glands exposed to radiation (whether x-irradiation or atomic blasts as in Japan), together with the infrequency of nodular goiter during adolescence among these Island people, made us fairly certain that we were dealing with a malignant tumor of the thyroid.

At operation on August 18, 1964, the gross picture was one of malignancy, with the thyroid having a cobble-stone appearance and "feel," the nodules being for the most part extremely indurated and unlike the findings in a nodular goiter. It was decided to remove the entire thyroid gland, with dissection of the anterior compartment of the neck, en bloc. The extreme irregularity of the thyroid gland, with nodules being of different color and consistency, made identification of parathyroid tissue extremely difficult. One parathyroid gland was identified after its pedicle was divided (it was attached to the undersurface of the right lobe of the thyroid gland), and this was implanted in the belly of the right sternomastoid gland. No other parathyroids were seen, and none were subsequently found in the specimen by the pathologist.

Postoperatively, the patient had attacks of frank tetany on the second and fourth postoperative days, each time responding to intravenous calcium glucoheptonate. Since that time she has been maintained on a high calcium-low phosphorous diet, calcium lactate tablets, Deltalin (vitamin D₂), and amphogel, in varying amounts. At first an attempt was made to wean her of all medications (as her serum calcium and phosphorus suggested return of parathyroid function). However, it was found that she could not tolerate complete withdrawal, so attempts were redirected toward establishing a program that would regulate her calcium/phosphorus balance with a minimum of medication.

As of now this program includes the following: Deltalin 100,000 units daily, calcium lactate two tablets (grains XX) t.i.d., amphogel 30 cc t.i.d., a high calcium/low phosphorus diet, and thyroid extract grains I t.i.d.

The child seems to be doing quite well on this program, has no suggestion of neuromuscular hyperirritability, and participates freely in available activities. Her serum calcium is 9.4, with a serum phosphorus of 5.7, and an alkaline phosphatase of 6 B.U.

PATIENT No. 21 (continued)

We plan on discharging her on this regimen, with the recommendation that she be readmitted in January, 1965, for another attempt to wean her from medications, or at least reduce the amounts of medications necessary.

Regarding the microscopic studies of the removed thyroid gland, here is a summary. Our pathologist considered the slides positive for malignancy, but desired confirmation from the AFIP. The report from AFIP, however, though acknowledging the presence of some "bizarre fields on high power examination," concluded that the diagnosis was "nodular, adenomatoid goiter, thyroid gland." The report was signed by G. H. Klinck.

Follow-Up: Following discharge the patient was careless about her treatment regimen and had an episode of tetany a few months later. She then began seriously taking her medication and remained asymptomatic until March, 1965, when she developed swelling of the right knee suggestive of rheumatic fever. She was hospitalized for several days and responded well to salicylates. She has since remained asymptomatic. In March, 1966, the Trousseau was negative, but the Chvostek was positive. At that time she was taken off thyroid hormone for several weeks (temporarily for test purposes), and her TSH was quite high (440 m μ g/ml) and an ¹³²I uptake study showed no uptake, both results proof of completeness of thyroidectomy. She has maintained euthyroid and euparathyroid states on levothyroxine 3 mg daily and vitamin D 50,000 units daily. She has shown no recurrence of thyroid nodules.

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
69	14	F	U.S. Naval Hospital, Guam	October, 1964

This 14 year old Micronesian female received radioactive fallout on the Island of Rongelap Atoll in 1954. After recovery from the beta burns and the blood element depression, all of which required several months, the child was well until examination in 1964 revealed several nodules in the thyroid gland. The patient was referred here as had two other girls with similar problems in recent months. These other two girls had undergone total thyroidectomy for what appeared at operation to be diffuse thyroid carcinomatous degeneration, but final pathologic studies by the AFIP yielded a diagnosis of benign multinodular goiter in each case.

The child's preoperative work-up was within the limits of normal except for the nodules in each lobe of the thyroid and acutely inflamed tonsils. The child was therefore placed on warm saline gargles, procaine, penicillin and forced fluids from 10-12-64 until she was ready for thyroid surgery on 10-20-64.

Thyroidectomy was performed on 10-20-64, with removal of the entire right lobe, the isthmus, and the medial one-half of the left lobe. The portion of the left lobe and superior pole that were left were relatively uninvolved with the nodular process that permeated the rest of the thyroid. It is of interest that a nodule overlying the trachea in the region of the so-called sentinal nodule was removed as a separate specimen prior to the thyroidectomy and sent to the laboratory for examination. The nodule was thyroid tissue; yet there was absolutely no connection between this nodule and the rest of the thyroid. The patient's postoperative course was completely benign, with removal of sutures on the second postoperative

PATIENT NO. 69 (continued)

day. She was discharged back to her island home on 10-27-64 with the recommendation that she take thyroid extract, grains 2 daily for an indefinite period.

Follow-Up: Following release from the hospital the patient has remained asymptomatic and apparently euthyroid on 3.0 mg of levothyroxine daily. No recurrence of thyroid nodules has been detected. In March, 1966, three weeks after stopping thyroid hormone (temporarily for test purposes) her ^{132}I uptake showed little, if any, thyroid function.

<u>PATIENT NO.</u>	<u>AGE</u>	<u>SEX</u>	<u>HOSPITAL</u>	<u>DATE</u>
2	12	M	Hospital of Medical Research Center, BNL	June, 1965

This 12 year old Marshallese boy who was exposed to radioactive fallout in 1954 was admitted to this hospital to evaluate a nodule of the thyroid gland which was discovered this past March.

History of Present Illness: During the annual physical examination (March, 1965) of the Rongelap people who were exposed to fallout radiation in 1954, this boy was found to have a small nodule of the thyroid gland about 2 cm in diameter. He was about 1 year of age at the time of exposure to radioactive fallout. He received an estimated dose of 175-200 rads whole body gamma radiation from the fallout and an undetermined dose of radiation (largely beta) to the skin along with some internal absorption of radioactive materials. It was estimated that the thyroid gland of a child this age had received roughly 700-1400 rads largely from the radioiodines absorbed. He experienced some nausea and vomiting during the first day or so which was believed related to his radiation exposure. His peripheral blood elements dropped to about one-half of the level of unexposed Marshallese people, but recovered to about normal by one year. Beginning about two weeks after exposure, he developed lesions of the skin from fallout deposition. These lesions were confined largely to the anterior neck folds, axillary, scalp and perianal regions. He also had extensive epilation. These lesions healed within several weeks and the hair began regrowing with complete regrowth by six months. He showed no other acute effects from his exposure. However, during the 10-year period since exposure he was found to show some degree of lag in growth, based on anthropometric studies as well as bone age studies. He has lagged a year to a year and one-half behind the unexposed children of the same age. This inhibition in his growth is believed to have been due to his fallout exposure, though the mechanisms involved have not been clear. There has been no evidence of thyroid malfunction. Two PBI readings taken in the past few years were in the normal range for the Marshallese people. Six years ago his cholesterol was 150 mg% and he remained in good health.

Physical Examination: No enlargement of the thyroid was noted. A 2 cm diameter nodule, oval in shape, finely moveable, nontender but firm, was noted in the right lobe. The nodule moved on swallowing. No other nodules and no lymphadenopathy was palpated. The remainder of the physical examination was essentially negative. There were a few scars on the anterior neck region and in the perianal region, residual of previous beta burns.

PATIENT NO. 2 (continued)

Laboratory and X-Ray Data: Thyroid Workup: PBI 6.2 µg%, iodine fractionation, total I 7.1 µg%, thyronine fraction (T4+T3) 4.2 µg%. Cholesterol 167 mg%, with 143 mg% esters. Thyroid autoantibodies titer under 1:16. BMR -23%. Thyroid scan (^{99m}Tc) showed possible nodularity of the right lobe, and to a lesser extent of the lower lobe. Chest x-ray showed no diseases or abnormalities. ¹³²I uptake study showed an uptake of 39% at 6 hours. PBI 5.4. Following TSH stimulation for three days, the uptake showed a slight decrease at 5-1/2 hours over the value before TSH stimulation.

Electrocardiogram normal; sed rate slightly elevated (21 mm). Blood count within normal limits except slightly low RBC (4,100,000) and hemoglobin 12.7 gm. Alkaline phosphatase was slightly low (4.5 units). Total proteins slightly elevated (8.5 gm with globulins up to 4.5 gm). These are typical values for Marshallese people. Normal values were found for prothrombin time, bilirubin, cephalin flocculation, transaminase, calcium, P, thymol turbidity, FBS, BUN, CO₂, Cl, Na, K. Urinalysis and stools for ova and parasites were both negative.

Hospital Course: The patient's hospital course here was uneventful, and he was transferred to New England Baptist Hospital, Boston, for surgical exploration of his neck on July 5. Dr. Bentley P. Colcock, under general anesthesia, removed a 1.2 cm diameter nodule from the right lobe of the thyroid gland, including some surrounding thyroid tissue which contained multiple small nodules. The nodules varied in color from pale gray to deep red. From histological examination of the tissues, a pathological diagnosis of adenomatous goiter was made. His recovery from the operation was rapid and subsequent course was uneventful. He was returned to this hospital on 7-13-65 and was discharged on 7-18-65, fit to travel back to the Marshall Islands.

Diagnosis: Adenomatous goiter.

Discharge Medication: No immediate drug therapy. It is anticipated that he will be put on thyroid 180 mg daily for life in September along with the other exposed Rongelap people.

Follow-Up: Patient has remained in good health and apparently euthyroid on 3 mg levothyroxine daily. In March, 1966, he was taken off thyroid therapy for three weeks for test purposes. At that time he showed good thyroidal function by ¹³²I uptake though his TSH level was slightly elevated (26 mµg/ml). No recurrence of nodules has been noted.

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
20	18	M	Hospital of Medical Research Center, BNL	June, 1965

This 18 year old Marshallese male was admitted to this Hospital for evaluation of a thyroid nodule which was discovered during this past March.

History of Present Illness: During the annual physical examination in March, 1965, this 18 year old boy was found to have a small 1 cm nodule of the right lobe of the thyroid gland. He was one of the group of 64 people who had been accidentally exposed to fallout in 1954. It was estimated that he had received a whole body

PATIENT NO. 20 (continued)

gamma dose of 175 rads, an undetermined dose to the skin surfaces, and some internal absorption of fallout material. It was estimated that the thyroid gland received a dose of somewhere between 700-1,400 rads, largely from radioiodine absorbed in the fallout. He was nauseated the day following the fallout and also complained of some itching and burning of the skin. About two weeks after exposure, he had epilation of the head, along with radiation burns of the scalp, neck, and toes. These lesions were not severe and healed with only slight residual depigmentation in the following weeks. The hair regrew by 6 months. He showed mild leukopenia and platelet depression during the first several months following exposure but with no complications. His blood elements had returned to the normal range by the end of the first year. Since that time he has been generally in good health and has apparently been euthyroid (1963 PBI 5.5 $\mu\text{g}\%$). Compared with unexposed boys of the same age, however, he has shown a slight degree of retardation in growth based on anthropometric and bone age studies.

Physical Examination: The patient was well nourished and developed but appeared to be somewhat smaller than normal for his age. The thyroid was not enlarged, but a 1.5 cm diameter nodule, firm in character, was noted in the right lower pole of the gland. The nodule was not tender and moved on swallowing. No other nodules were palpated and no regional adenopathy was noted. The remainder of the physical examination was essentially negative.

Laboratory and X-Ray Data: Thyroid Work-Up: PBI 6.1 $\mu\text{g}\%$, iodine fractionation: total I 6.5 $\mu\text{g}\%$, iodoprotein 1.3 $\mu\text{g}\%$, thyronine (T4 + T3) 4.2 $\mu\text{g}\%$. Thyroid auto-antibodies under 1:16. Cholesterol 170 mg% with esters 144 mg%. BMR -12. Thyroid scan ($^{99\text{m}}\text{Tc}$) showed "cold" nodule at lower lobe. ^{132}I uptake studies showed 40% uptake in 6 hours with 32.5% urinary excretion at that time. Following TSH stimulation for three days, the uptake was only 33.7% at 5-1/2 hours, with urinary excretion 18.5%. PBI 6.8 $\mu\text{g}\%$. The blood count was within normal limits; alkaline phosphatase was slightly low (4.4 units), total protein slightly elevated, 8.2 gm with globulins 3.9 gm. Within normal ranges were: prothrombin time, sedimentation rate, bilirubin, cephalin flocculation, transaminase, Ca, P, thymol turbidity, FBS, BUN, CO_2 , Cl, Na, K. Normal also were EKG, slit lamp examination, urinalysis, and stools for ova and parasites. Chest plate showed no active disease in the chest.

Hospital Course: The patient remained asymptomatic while here. On July 5 he was taken to New England Baptist Hospital in Boston, Massachusetts for surgery. Dr. Bentley P. Colcock removed about 3.1 cm of thyroid tissue which contained multiple nodules varying in size up to 1 cm in diameter. They varied from pale grey and firm to pulpy and semicystic and deep red. The pathologic diagnosis was adenomatous goiter. His recovery was rapid and uneventful. He was returned to Brookhaven on July 13 where he remained asymptomatic and was discharged on July 18, 1965, fit to travel back to the Marshall Islands.

Diagnosis: Adenomatous goiter.

Discharge Medication: No immediate drug therapy was recommended. However, this patient will receive desiccated thyroid, 180 mg daily beginning in September, along with the remainder of the exposed Rongelap people.

PATIENT NO. 20 (continued)

Follow-Up: Since discharge the patient has been asymptomatic and apparently euthyroid even though his thyroid therapy (3 mg levothyroxine daily) has been sporadic. No recurrence of thyroid nodules has been noted.

<u>PATIENT NO.</u>	<u>AGE</u>	<u>SEX</u>	<u>HOSPITAL</u>	<u>DATE</u>
64	41	F	Hospital of Medical Research Center, BNL	June, 1965

This 41 year old Marshallese woman was admitted to the hospital for evaluation of a thyroid nodule that was discovered during the 1965 annual medical survey of the Rongelap people.

History of Present Illness: A 1 cm nodule was discovered in the right lobe of the thyroid gland of this woman in March, 1965. It was firm, non-tender, and no cervical lymph nodes were palpable. She was one of a group of 64 Rongelap people who had been exposed to fallout 11 years ago. She had received an estimated dose of 175 rads of whole body gamma exposure and in addition radiation exposure to her skin and some internal absorption of radioactive materials. Absorption of radioiodines from the fallout gave an estimated dose to the thyroid gland of 150-160 rads, in addition to the 175 rads from gamma radiation. She had early nausea and vomiting believed to be associated with her radiation exposure and her blood elements were depressed to about one-half normal levels. She developed "beta burns" of the skin beginning about two weeks after exposure, mainly on the back of her neck. These lesions healed within several weeks and her blood elements returned to near normal levels by about one year after exposure. Since that time she has remained in relatively good health with no serious illnesses or injuries. She has had nine children, four of them born since the fallout exposure. She has apparently remained euthyroid. A PBI taken in March was 10.0 µg% (high normal for the Marshallese) and serum cholesterol in 1958 was 249 mg%.

Physical Examination: The patient was a well nourished, well developed female of about her stated age of 41. Pterygia were noted in the right eye. In the right lower pole of the thyroid there was a deeply fixed 1 cm nodule that moved with the thyroid on swallowing. The overlying skin was not attached to the nodule. The nodule was very hard but not tender and no other nodules were palpable. Regional adenopathy was not noted. The blood pressure was normal. There were no other significant findings on physical examination.

Laboratory and X-Ray Data: Thyroid Work-up revealed the following: Iodine fractionation: total iodine 8.6 µg%, iodoprotein fraction 4.5 µg%, thyronine fraction (T4 + T3 iodine) 3.4 µg%, PBI 7.5 µg%, serum cholesterol 239 mg% (esters 183 mg%). Basal metabolic rate -27%. Thyroxin autoantibodies titer under 1:16. Thyroid scan showed large "cold" nodule replacing the lower pole of the right lobe of the thyroid gland with nodularity also involving the left lobe, lower pole area. Chest x-ray showed no active disease. Thyroid uptake study using ¹³²I showed a 6-hour uptake of 22.3% with urinary excretion of 33%. After three days of treatment with TSH (I.M.) thyroid uptake had increased to 33% at 5-1/2 hours. PBI 8.0 µg%. Except for slightly low RBC (3,300,000) and hemoglobin (11.6 gm%), the blood count was normal. Urinalysis was negative and the following blood chemistry studies were negative: bilirubin, cephalin flocculation, transaminase, Ca, P, thymol turbidity, FBS, BUN, CO₂, Na, K. Alkaline phosphatase was slightly low (1.4 units); the globulin fraction of proteins was slightly elevated (4.26 gm%).

PATIENT NO. 64 (continued)

The EKG was within normal limits. Stools were negative for ova and parasites. Roentgenograms of the chest and bones for metastasis were negative.

Hospital Course: Her hospital course was uneventful.

Surgery at New England Baptist Hospital, Boston, Massachusetts: The patient was transferred to the Baptist Hospital in Boston on July 5, 1965, and the following day under general anesthesia the neck was explored by Dr. Bentley P. Colcock. The thyroid nodule was found to be malignant, and he performed a subtotal thyroidectomy leaving a small portion of the left lobe intact. Microscopic study of the tissues gave the following pathologic diagnosis: "Mixed papillary and follicular carcinoma of the right lobe with blood vessel invasion and metastasis to one lymph node. Negative parathyroid (right). Left lobe of thyroid shows no significant changes." Aside from mild postoperative nausea and vomiting, she recovered very rapidly from the operation. In view of the positive diagnosis of malignancy, on July 15 she was given 30 mc ¹³¹I in order to ablate the remainder of the thyroid gland and any possible residual metastatic foci. She was returned to this hospital on July 16, 1965. She remained in good health and was discharged on July 18, fit for travel back to the Marshall Islands.

Diagnosis: Mixed papillary and follicular carcinoma of the right lobe of the thyroid gland with blood vessel invasion and metastasis to one lymph node.

Discharge Medication: On August 1 she is to begin taking desiccated thyroid, 120 mg daily. She is to remain under the direct supervision of one of the Marshallese practitioners on her return.

Follow-Up: Patient has apparently maintained "euthyroid status" on 3 mg of levothyroxine daily. In March, 1966, she was to have a complete hospital check-up at Tripler Army Hospital in Hawaii but was found to be 5 months pregnant, and the examination was delayed until after birth of her baby. In September, 1966, at Tripler General Hospital, Hawaii, thyroid uptake studies following TSH stimulation (10 units daily for two days), thyroid scans, and skeletal surveys for metastasis showed absence of the thyroid and no detectable metastasis.

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
33	13	F	Hospital of Medical Research Center, BNL	June, 1966

This 13 year old Marshallese girl was admitted to evaluate a thyroid nodule which was discovered during the past year.

History of Present Illness: The patient was 1 year old at time of exposure to fallout and received about 175 rads of gamma radiation, radiation of the skin from fallout deposited thereon and some internal absorption of radioisotopes. It was estimated that her thyroid gland received 700-1,400 rads from the radioiodines absorbed in addition to the 175 rads of gamma radiation. Acute effects of her exposure consisted of beta burns of the skin and extensive epilation of the scalp. She also developed transient leukopenia and thrombocytopenia. By one year she had recovered from these acute effects and remained generally in good health. Her growth and development has been about normal with menarche occurring at age 13. In September, 1965, she was thought to have an irregularity of the thyroid gland.

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
42	15	F	Hospital of Medical Research Center, BNL	June, 1966

This 15 year old Marshallese girl was admitted to this hospital for evaluation of a thyroid nodule which had developed during the past year.

History of Present Illness: This girl had been exposed at 3 years of age to radioactive fallout, receiving an estimated dose of 175 rads of whole-body gamma radiation, irradiation of the exposed skin surfaces from direct deposition of fallout material, and some degree of internal absorption of radioactive materials from ingestion of contaminated food and water. It was estimated that the radioiodine absorbed in the fallout delivered a dose to the thyroid gland in the range of 700-1,400 rads and an additional dose of 175 rads was received by the gland from gamma radiation. She had transient gastrointestinal symptoms during the first two days and subsequently developed a leukopenia and thrombocytopenia. Beginning about two weeks after exposure she developed rather marked "beta burns" of the skin and epilation. All of these acute effects had disappeared at about 1 year post exposure. With the exception of the present illness, subsequent examinations have revealed no serious illnesses or injuries in this girl. Her statural growth over the past 12 years has been slightly below the mean curve for the unexposed Marshallese girls in the comparison population. Menarche occurred in 1965. In September, 1965, a small nodule was noted in the right lower pole of the thyroid. In spite of treatment with thyroid hormone the nodule increased in size to about 0.5 mm by March, 1966. She had always been considered to be euthyroid. Her PBI level in March, 1966, was 5.0 μ g%.

Physical Examination: A small nodule (2-3 mm) was palpated in its left lower lobe. The remainder of the physical examination was essentially negative except for impetigo-like lesions of the skin.

Laboratory and X-Ray Data: Thyroid Work-Up: PBI was 6.5 μ g% with 4.5 μ g% of thyroxin; T₃ 12.9%; antithyroglobulin titre under 1:16; thyroid scan with ^{99m}Tc showed normal uptake with several "hot" nodules noted in the right upper lobe, right middle lobe and left upper lobe; ¹³²I uptake was normal and showed normal increase following TSH stimulation. BMR was +25 and +28; cholesterol level was low (118 mg% with 105.8 mg% esters). In contrast to some of the other Marshallese cases, these findings seemed to indicate a slightly increased functioning gland. Chest plate and EKG were normal. The hemogram and the remainder of the laboratory work-up was essentially negative.

Hospital Course: Following the initial examination at this hospital, the patient was removed on June 5 to the New England Deaconess Hospital, Boston. On June 8, 1966, a bilateral subtotal thyroidectomy was performed by Dr. Bentley P. Colcock of the Lahey Clinic, removing thyroid tissue which contained multiple nodules, the largest being about 0.5 cm. Dr. W. A. Meissner of the New England Deaconess Hospital reported that the surgical specimens contained multiple nodules of varying sizes and his pathological diagnosis was: adenomatous goiter. The patient's recovery from surgery was uneventful, and she was returned to Brookhaven on June 11. She was started on desiccated thyroid treatment, 180 mg daily to be continued indefinitely. Her surgical wound healed nicely and she was asymptomatic.

Diagnosis: Adenomatous goiter.

PATIENT NO. 42 (continued)

Discharge Medication: Thyroid hormone medication to be continued indefinitely.

This patient was seen in September, 1966, in the Marshall Islands, and she was found to be euthyroid on the hormone treatment, with no complications.

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
59	46	F	Hospital of Medical Research Center, BNL	June, 1966

This 46 year old Marshallese woman had been exposed to radioactive fallout in 1954 and was admitted for evaluation of a nodule of the thyroid gland.

History of Present Illness: In September, 1965, during a thyroid survey of the Rongelap people, a 0.5 cm nodule was noted in the left isthmic region of the thyroid. This patient had received a smaller dose of radiation at the time of the fallout than the other Rongelap patients admitted here at this time. The calculated dose to the thyroid gland was about 115 rads (69 rads from whole body gamma radiation, and about 40 rads from radioiodines). She had few signs of acute radiation effects except for mild "beta burns", and slight thrombocytopenia following exposure. Since this time she had remained generally in good health. Her disease history includes chickenpox, mumps, yaws, and gonorrhoea. She has had recurring attacks of bronchitis though x-rays of the chest have been considered negative in the past. She had always appeared to be euthyroid with PBIs in 1958 and 1965 in the normal range for the Marshallese. Iodine fractionation studies in March, 1966, were normal.

Physical Examination: On admission the patient was febrile, and it was thought that she might have bronchopneumonia, bronchitis, influenza, or possibly tuberculosis. Thyroid examination showed the gland not to be enlarged, but a freely moveable 1 cm nodule in the lower part of the gland was palpated. There was no lymphadenopathy. The remainder of the physical examination was essentially negative.

Laboratory and X-Ray Data: Pulmonary Studies: Chest plates on May 26 and 27 showed possible pneumonitis in the upper and middle right lobes of the lungs which were believed to be of partly acute, partly chronic nature (Tbc?). A chest plate on 5/31 showed that the middle lobes remained essentially unchanged. PPD showed slight reaction to second strength test. Hemogram showed neutrophilic leukocytosis on admission which returned to normal about 5 days later. Thyroid Studies: BMR -27 and -24. PBI 6.4 µg%, T3 13.9%, thyroxin iodine 5.0 µg%. Antithyroid antibodies under 1:16. Cholesterol 102.8 (94.3 esters). Thyroid scan using ^{99m}Tc revealed no nodules and normal uptake. ¹³²I showed 19.8% uptake in 6 hours with 45% urinary excretion. Following TSH (10 units daily for 3 days), the uptake increased to 54.9% in 6 hours with urinary excretion rate of 20.3%. EKG was normal. Except for weakly positive VDRL and REITER protein complement fixation test, other laboratory tests were generally negative.

PATIENT NO. 59 (continued)

Hospital Course: The acute pneumonitis noted on admission responded well to acromycin and penicillin therapy, and the patient became afebrile and asymptomatic within a few days. The right middle lobe showed clearing, but the upper lobe showed persistent changes, and in view of the slightly positive second-strength reaction to the PPD, Dr. L. R. Sonders, Boston, felt that tuberculous infection was consistent with the findings though he considered it likely that the process was inactive and not contraindicative to surgery. The results of the thyroid study did not suggest any thyroid metabolic imbalance. Though the BMR was somewhat low and the cholesterol low, the iodine fractionation studies and uptake studies as well as effect of stimulus of TSH were normal. She was admitted to the New England Deaconess Hospital on June 6, and surgical excision of the thyroid isthmus containing a 0.4 cm nodule was accomplished on June 8. The patient withstood the surgery well and the wound healed per primam. The histological diagnosis was adenomatous goiter. She was returned to this hospital on June 11. On Dr. Sonders' recommendation she was placed on isoniazid therapy, 100 mg t.i.d. In view of her positive serology she was placed on penicillin, 600,000 units daily for a total dose of 6.5 million units. In addition, she was started on desiccated thyroid, 180 mg daily. She was discharged on June 16 to return to the Marshall Islands. A letter was sent to the medical authorities in the Marshalls advising them to continue the isoniazid for 2 years and thyroid therapy indefinitely. They would be informed later of the results of the tuberculous cultures, and, if possible, it was recommended that she be placed in the hospital for treatment with PAS.

Diagnosis: (1) Adenomatous goiter. (2) Pulmonary tuberculosis--unspecified activity (002.9). (3) Positive serology (028.3).

Discharge Medication: To continue indefinitely on thyroid hormone medication.

This patient was seen in September, 1966, in the Marshall Islands, and she was found to be euthyroid on the hormone treatment, with no complications.

PATIENT NO.	AGE	SEX	HOSPITAL	DATE
61	20	F	Hospital of Medical Research Center, BNL	June, 1966

This 20 year old Marshallese female was admitted to this hospital for evaluation of a thyroid nodule which was discovered during the past year.

History of Present Illness: As a 7 year old girl the patient was exposed to radioactive fallout in 1954. She was exposed to an estimated dose of 175 rads of whole-body gamma radiation, significant amounts of radiation to the exposed skin surfaces from deposition of fallout material thereon, and some internal absorption of fallout. It was estimated that the radioiodines absorbed delivered a dose to the thyroid in a range of 300-1,000 rads in addition to the 175 rads of gamma radiation. She had transient early gastrointestinal symptomatology which was followed some two weeks later by mild beta burns of the skin and moderate degree of epilation of the head. She also had leukopenia and thrombocytopenia within the first six weeks. She had largely recovered from these acute effects by one year post exposure. Her subsequent medical history shows no findings which could be related to radiation exposure. There were no noteworthy illnesses. Her growth

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PATIENT NO. 61 (continued)

and development has appeared to be entirely normal, if not somewhat precocious. A 1 cm nodule of the left lower pole of the thyroid was noted in September, 1965, which did not decrease on thyroid hormone therapy. She appeared to be euthyroid with PBIs in the normal Marshallese range. Her cholesterol was slightly elevated in 1957 (277 mg%).

Physical Examination: The patient was obese. The remainder of the physical examination was essentially negative except for slight acne and dental caries.

Laboratory and X-Ray Data: Thyroid Studies: PBI was 7.5 µg% with thyroxin iodine 4.9 µg%; T3 was 12.9%. Antithyroglobulin titre was 1:32. Cholesterol was 162 mg% (esters 151 mg%). Thyroid scan using ^{99m}Tc showed normal uptake but the gland was asymmetrical, with the right lobe showing uptake only in the upper portion. A "hot" nodule was noted in the lower left pole of the thyroid. ¹³²I uptake was normal and there was normal increase in uptake following TSH stimulation. The chest plate and EKG were normal. The remainder of the laboratory studies were essentially negative except for an increased sedimentation rate which is an unexplained finding in most of the Marshallese.

Hospital Course: Following the studies at this hospital, the patient was transferred to the New England Deaconess Hospital in Boston on June 5, 1966. On June 8, Dr. Bentley P. Colcock of Lahey Clinic performed a bilateral subtotal thyroidectomy removing a 1.5 cm nodule in the left lower thyroid lobe and a small cyst in the right lower lobe. Dr. W. A. Meissner of the New England Deaconess Hospital examined the surgical specimens, and his pathological diagnosis was "adenomatous goiter." Recovery from surgery was uneventful, and the patient was readmitted to this hospital on June 11. She was placed on desiccated thyroid, 180 mg daily, indefinitely. Her wound healed nicely, and she was asymptomatic.

Diagnosis: Adenomatous goiter.

Discharge Medication: Thyroid hormone therapy to be continued indefinitely.

This patient was seen in September, 1966, in the Marshall Islands, and she was found to be euthyroid on the hormone therapy, with no complications.

<u>PATIENT NO.</u>	<u>AGE</u>	<u>SEX</u>	<u>HOSPITAL</u>	<u>DATE</u>
65	13	F	Hospital of Medical Research Center, BNL	June, 1966

This 13 year old Marshallese girl was admitted to this hospital for evaluation of a 0.5 cm nodule of the right lobe of the thyroid gland discovered during the past year.

History of Present Illness: This child was exposed to fallout radiation in 1954 at 1 year of age and received an estimated dose of 175 rads of whole-body gamma radiation, irradiation of the skin from deposition of fallout material thereon, and absorption of significant amounts of radionuclides from ingestion of contaminated food and water. It was estimated her thyroid gland received a dose in the range of 700-1,400 rads from radioiodines in the fallout and in addition 175 rads from gamma radiation. She had early gastrointestinal symptoms

PATIENT NO. 65 (continued)

followed two weeks later by the development of marked beta burns of the skin and epilation of the scalp and leukopenia and thrombocytopenia. She had recovered from these effects by one year. Her subsequent medical history revealed no serious illnesses, but the child was thin and appeared to be somewhat retarded in growth and development. Menarche had not yet occurred. She had recurrent impetigo infections of the skin. On discovery of her thyroid nodule in September, 1965, she was placed on thyroid hormone therapy. The nodule did not reduce in size on this therapy and therefore she was brought here for treatment. Until recently she had been thought to be euthyroid with normal PBI and cholesterol levels. However, by March of this year her thyroxine iodine level was down to 1.9 $\mu\text{g}\%$, and she showed increased TSH levels suggestive of a hypofunctioning thyroid gland.

Physical Examination: This slender girl appeared younger than her stated age. A 1 cm thyroid nodule was noted in the lower pole of the right thyroid. No lymphadenopathy was noted. The remainder of the physical examination was essentially negative, except for the presence of a small pilonidal sinus.

Laboratory and X-Ray Data: Thyroid Work-Up: Iodine fractionation studies on her plasma revealed thyroxine iodine of 3.5 $\mu\text{g}\%$ (however, she had only been off of thyroid hormone therapy for about two weeks). Her TSH levels were quite elevated (125 $\text{m}\mu\text{g}/\text{ml}$). Antithyroglobulin titre was under 1:16. Thyroid scan using $^{99\text{m}}\text{Tc}$ showed a small but apparently normal thyroid. Though $^{99\text{m}}\text{Tc}$ uptake was normal, the ^{132}I uptake at 6 hours was somewhat low and little increase was noted after TSH stimulation. These findings are in conformity with a hypofunctioning gland. A chest plate was negative except for suggestive extrinsic pressure on the right side of the trachea at the level of T-1. Alkaline phosphatase level was elevated (13.0 units).

Hospital Course: The patient's hospital course here was uneventful. On June 5 she was transferred to the New England Deaconess Hospital, and on June 6, Dr. Bentley P. Colcock of Lahey Clinic performed thyroid surgery in that hospital. A right subtotal thyroidectomy was performed with removal of the lower right pole of the thyroid as well as a small cyst from the left lobe. The microscopic diagnosis of removed tissues by Dr. W. A. Meissner of the New England Deaconess Hospital was "adenomatous goiter." She was returned to Brookhaven on June 11, and her recovery from surgery was uneventful. She was placed on desiccated thyroid, 180 mg daily, to be continued indefinitely. The wound healed nicely, and she was asymptomatic.

Diagnosis: Adenomatous goiter.

Discharge Medication: To continue thyroid hormone therapy indefinitely.

This patient was seen in September, 1966, in the Marshall Islands, and she was found to be euthyroid on the hormone treatment, with no complications.

APPENDIX 7

A. Malignancies Recorded in Marshall Islands, May 1952-October 1962*

<u>Malignancy</u>	<u>No. of Cases</u>
Ca, cervix	11
Adenocarcinoma, large bowel	3
Carcinomatosis, abdominal cavity--primary site unknown	5
Osteosarcoma	1
Ovarian ca	5
Bronchogenic ca (x-ray and clinical)	3
Bronchogenic ca (autopsy and tissue examination)	1
Skin (squamous cell ca of lip)	1
Adenocarcinoma of thyroid	2
Adenocarcinoma of breast	3
Hypernephroma	1
Ca of liver (exploratory surgery)	1
Ca of liver (biopsy and/or autopsy)	3
Leukemia	2

*Statistics obtained from District Director of Public Health, Marshall Islands. Probably represents more than 58% of cases.

B. NUMBER OF PATIENTS WITH MALIGNANT NEOPLASMS, CLASSIFIED BY TYPE
DISCHARGED FROM DISTRICT HOSPITALS
TRUST TERRITORY, 1962-1965

SITE OF MALIGNANT NEOPLASM	DISTRICT HOSPITAL						
	TOTAL	MARSHALL ISLANDS	MARSHALL ISLANDS	PALAU	PONAPE	TRUK	YAP
ALL SITES, TOTAL	162	36	40	23	33	37	13
BUCCAL CAVITY AND NASOPHARYNX (140-148), TOTAL	13	6	1	1	3	2	-
LIP (140)	1	-	1	-	-	-	-
TONGUE (141)	-	-	-	-	-	-	-
SALIVARY GLAND (142)	2	-	-	-	1	1	-
FLOOR OF MOUTH (143)	1	-	-	-	1	-	-
OTHER PARTS OF MOUTH, AND MOUTH UNSPECIFIED (144)	3	1	-	1	-	1	-
ORAL NASOPHARYNX (145)	-	-	-	-	-	-	-
NASOPHARYNX (146)	5	5	-	-	-	-	-
HYPOPHARYNX (147)	-	-	-	-	-	-	-
PHARYNX, UNSPECIFIED (148)	1	-	-	-	1	-	-
DIGESTIVE ORGANS AND PERITONEUM (150-159), TOTAL	36	7	7	9	6	4	3
ESOPHAGUS (150)	1	-	-	-	1	-	-
STOMACH (151)	11	5	2	1	3	-	-
SMALL INTESTINE, INCLUDING DUODENUM (152)	-	-	-	-	-	-	-
LARGE INTESTINE, EXCEPT RECTUM (153)	5	-	1	3	-	1	-
RECTUM (154)	1	-	1	-	-	-	-
BILIARY PASSAGES AND LIVER (PRIMARY) (155)	14	1	3	5	2	1	2
LIVER (SECONDARY AND UNSPECIFIED) (156)	2	-	-	-	-	1	1
PANCREAS (157)	2	1	-	-	-	1	-
PERITONEUM (158)	-	-	-	-	-	-	-
RESPIRATORY SYSTEM (160-165), TOTAL	14	3	3	-	2	5	1
NOSE, NASAL CAVITIES, MIDDLE EAR AND ACCESSORY SINUSES (160)	1	-	-	-	-	1	-
LARYNX (161)	1	1	-	-	-	-	-
BRONCHUS AND TRACHEA AND LUNG SPECIFIED AS PRIMARY (162)	7	2	2	-	2	-	1
LUNG, UNSPECIFIED AS TO WHETHER PRIMARY OR SECONDARY (163)	3	-	-	-	-	3	-
MEDIASTINUM (164)	1	-	1	-	-	-	-
THORACIC ORGANS (SECONDARY) (165)	1	-	-	-	-	1	-
BREAST AND GENITOURINARY SYSTEM (170-181), TOTAL	62	8	18	7	13	13	3
BREAST (170)	11	1	6	2	1	1	-
CERVIX UTERI (171)	24	1	4	1	9	8	1
CORPUS UTERI (172)	6	3	2	-	1	-	-
OTHER PARTS OF UTERUS, INCLUDING CYSTIC EPITHELIOMA (173)	-	-	-	-	-	-	-
UTERUS, UNSPECIFIED (174)	3	-	1	-	-	2	-
OVARY, FALLOPIAN TUBE AND BROAD LIGAMENT (175)	4	-	3	-	-	1	-
OTHER AND UNSPECIFIED FEMALE GENITAL ORGANS (176)	1	-	-	-	1	-	-
PROSTATE (177)	5	1	1	2	-	1	-
TESTIS (178)	1	-	-	-	-	-	1
OTHER AND UNSPECIFIED MALE GENITAL ORGANS (179)	2	-	-	2	-	-	-
KIDNEY (180)	3	1	1	-	1	-	-
BLADDER AND OTHER URINARY ORGANS (181)	2	1	-	-	-	-	1
OTHER AND UNSPECIFIED SITES (190-199), TOTAL	34	5	7	2	6	9	5
MALIGNANT MELANOMA OF SKIN (190)	3	-	-	-	2	1	-
OTHER MALIGNANT NEOPLASM OF SKIN (191)	9	3	1	-	1	3	1
EYE (192)	-	-	-	-	-	-	-
BRAIN AND OTHER PARTS OF NERVOUS SYSTEM (193)	2	-	-	-	-	-	2
THYROID GLAND (194)	5	1	2	2	-	-	-
OTHER ENDOCRINE GLANDS (195)	-	-	-	-	-	-	-
BONE (INCLUDING JAW BONE) (196)	3	-	1	-	1	1	-
CONNECTIVE TISSUE (197)	4	-	-	-	-	3	1
SECONDARY AND UNSPECIFIED MALIGNANT NEOPLASM OF LYMPH NODE (198)	2	1	-	-	-	-	1
OTHER AND UNSPECIFIED SITES (199)	6	-	3	-	2	1	-
LYMPHATIC AND HEMATOPOIETIC TISSUES (200-205), TOTAL	23	7	4	4	3	4	1
LYMPHOSARCOMA AND RETICULOSARCOMA (200)	5	-	2	1	1	1	-
HODGKIN'S DISEASE (201)	1	-	-	1	-	-	-
OTHER FORMS OF LYMPHOMA (RETICULOSIS)	-	-	-	-	-	-	-
MULTIPLE MYELOMA (PLASMOCYTOMA) (203)	5	4	-	-	-	1	-
LEUKEMIA AND ALEUKEMIA (204)	12	3	2	2	2	2	1
MYCOSIS FUNGOIDES (205)	-	-	-	-	-	-	-

SOURCE: DISEASE SUMMARY CARDS FROM THE HOSPITALS.

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APPENDIX 8

Rongelap Group and Control Mean Blood Counts at Various Times After Exposure

Postexposure Day	WBC ($\times 10^{-3}$)		Neutrophils ($\times 10^{-3}$)		Lymphocytes ($\times 10^{-3}$)		Platelets ($\times 10^{-4}$)				Hematocrit %			RBC ($\times 10^{-6}$)		
	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female All Ages	Total Group	Male <15	Male >15	Female All Ages	Male <15	Male >15	Female All Ages
	3	9.0	8.2	6.4	4.7	1.8	2.2	----	----	----	----	----	----	----	----	----
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	----	----	----	----	----	----
12	5.9	6.3	3.5	3.9	2.1	1.7	----	----	----	----	----	----	----	----	----	----
15	5.9	6.5	3.2	4.1	2.4	1.9	27.1	21.3	21.7	22.5	----	----	----	----	----	----
18	6.7	7.2	3.4	4.7	2.4	2.1	21.8	19.1	21.8	21.0	----	----	----	----	----	----
22	7.0	7.4	4.3	5.0	2.6	2.1	16.8	14.6	15.2	15.3	37.5	43.9	39.0	----	----	----
26	5.7	6.1	3.0	3.9	2.3	1.8	13.2	12.9	10.9	11.9	36.3	41.6	37.5	----	----	----
30	7.6	7.8	4.0	5.3	3.2	2.1	14.1	12.3	11.8	12.3	37.9	42.2	37.1	----	----	----
33	6.5	6.2	3.1	3.8	3.2	2.0	17.9	16.6	15.1	16.0	37.4	42.2	36.8	----	----	----
39	5.7	5.5	3.0	3.3	2.6	2.0	29.5	22.0	22.4	22.8	37.8	42.4	37.4	----	----	----
43	5.2	5.2	2.0	2.6	2.9	2.3	26.8	20.9	23.2	23.2	37.3	41.8	37.6	----	----	----
47	5.9	5.8	2.6	3.3	3.1	2.4	24.6	20.6	23.9	23.1	39.0	43.4	38.3	----	----	----
51	6.7	5.6	2.6	3.5	3.4	2.1	22.1	17.5	21.2	20.3	----	----	----	----	----	----
56	7.0	6.0	3.5	3.5	3.7	2.4	----	----	----	----	----	----	----	----	----	----
63	7.7	6.0	3.9	3.6	3.7	2.3	23.1	18.2	20.2	20.1	----	----	----	----	----	----
70	7.6	6.5	3.8	4.0	3.3	2.2	----	----	----	----	----	----	----	----	----	----
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	38.0	41.7	38.2	----	----	----
1-yr survey	10.1	8.1	4.7	4.8	4.6	2.8	26.6	19.5	27.6	24.9	37.5	41.1	36.9	----	----	----
2-yr survey	11.8	8.6	5.9	4.8	4.7	3.1	30.0	21.4	25.5	24.7	38.7	41.2	38.1	----	----	----
3-yr survey	8.6	6.9	4.1	3.7	3.7	2.7	32.0	22.1	28.1	----	35.6	38.7	35.4	----	----	----
4-yr survey	8.9	7.5	3.3	3.4	4.6	3.6	32.5	27.1	30.8	----	35.6	41.0	35.8	----	----	----
5-yr survey	13.5	9.5	6.9	4.8	6.0	4.0	32.3	24.4	27.6	----	----	----	----	4.45	4.71	4.21
6-yr survey	----	6.5	---	3.5	---	2.7	----	----	----	----	----	----	----	----	----	----
7-yr survey	----	7.4	---	3.9	---	2.9	----	24.6 ^a	27.3	----	37.6	41.7	37.0	4.54	4.45	4.11
8-yr survey	----	6.9	---	3.6	---	2.6	----	32.8 ^b	32.1	----	38.5	43.0	39.3	4.68	4.67	4.44
9-yr survey	----	7.4	---	3.7	---	3.0	----	23.1 ^c	28.4	----	39.1	43.7	38.4	4.29	4.38	4.12
10-yr survey	----	8.2	---	3.8	---	3.5	----	32.8	37.2	----	40.4	43.5	39.3	----	----	----
11-yr survey	----	7.4	---	3.7	---	3.0	----	26.3	28.5	----	39.9	44.0	37.7	4.65	4.60	3.94
12-yr survey	----	6.8	---	3.6	---	2.5	----	----	----	----	38.8	42.7	38.3	----	----	----
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	39.6	46.0	39.9	----	----	----
Rita cont. 6 mo	10.7	7.6	5.4	5.2	4.7	3.7	35.0	27.3	30.9	30.4	----	----	----	----	----	----
Rita cont. 1 yr	----	----	----	----	----	----	37.5	24.5	29.4	27.6	----	----	----	----	----	----
Rita cont. 2 yr	14.0	8.9	7.0	4.4	5.6	3.6	35.5	24.2	31.2	29.5	38.9	42.1	39.8	----	----	----
Rong. cont. 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	----	35.6	41.0	35.9	----	----	----
Rong. cont. 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	----	35.5	42.8	35.1	----	----	----
Rong. cont. 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	----	----	----	----	4.60	4.80	4.40
Rong. cont. 7 yr	----	7.8	---	4.2	---	3.1	----	28.5 ^a	31.4	----	37.2	44.4	37.0	4.52	4.68	4.12
Rong. cont. 8 yr	----	7.7	---	4.2	---	2.9	----	34.8 ^b	34.5	----	38.3	44.1	39.0	4.60	4.90	4.47
Rong. cont. 9 yr	----	7.7	---	3.9	---	3.1	----	29.1 ^c	32.5	----	39.4	43.8	38.3	4.33	4.50	4.13
Rong. cont. 10 yr	----	9.1	---	4.8	---	3.5	----	35.4	37.9	----	37.4	44.1	38.3	----	----	----
Rong. cont. 11 yr	----	7.3	---	3.9	---	2.8	----	28.1	28.3	----	39.6	44.4	37.6	4.65	4.71	4.14

^aIncludes all males >7.^bIncludes all males >8.^cIncludes all males >9.

APPENDIX 9

Ailingnae Group and Control Mean Blood Counts at Various Times After Exposure

Postexposure day	WBC ($\times 10^{-3}$)		Neutrophils ($\times 10^{-3}$)		Lymphocytes ($\times 10^{-3}$)		Platelets ($\times 10^{-4}$)				Hematocrit %			RBC ($\times 10^{-6}$)		
	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female all ages	Total group	Male <15	Male >15	Female all ages	Male <15	Male >15	Female all ages
	3	6.0	7.0	3.0	5.0	2.8	2.2	----	----	----	----	----	----	----	----	----
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	----	----	----	----	----	----
12	6.3	7.6	1.8	4.7	3.1	2.2	----	----	----	----	----	----	----	----	----	----
15	7.1	7.0	2.3	4.5	4.2	2.2	29.0	20.2	24.6	23.9	----	----	----	----	----	----
18	6.8	7.8	2.9	5.0	3.5	2.4	27.5	21.7	24.9	24.3	----	----	----	----	----	----
22	8.9	8.7	5.3	5.4	2.7	2.9	23.5	17.0	22.9	21.3	37.5	43.7	39.2	----	----	----
26	8.4	7.0	4.8	4.4	3.2	2.2	20.0	13.8	17.4	16.7	36.5	43.2	36.8	----	----	----
30	9.6	8.6	5.3	6.2	3.7	2.0	19.5	12.8	18.2	16.8	36.0	44.6	36.7	----	----	----
33	7.7	7.8	3.3	5.2	3.5	2.2	24.0	15.8	22.7	17.6	35.5	43.8	37.3	----	----	----
39	7.5	6.2	2.9	4.2	4.7	1.9	26.5	20.8	27.0	25.2	35.0	45.6	37.4	----	----	----
43	6.9	6.5	2.7	3.6	3.9	2.7	28.0	19.6	25.3	24.0	36.0	45.2	36.8	----	----	----
47	7.3	6.7	3.5	3.8	3.4	2.7	27.0	20.0	26.1	24.5	----	46.5	40.2	----	----	----
51	8.4	6.3	3.8	3.6	4.0	2.2	32.0	18.2	25.0	23.9	----	----	----	----	----	----
54	4.6	6.3	2.8	3.5	3.2	2.5	37.0	19.8	23.8	24.2	----	----	----	----	----	----
6-mo survey	7.7	6.5	4.8	3.9	2.7	2.2	25.2	19.2	23.9	22.7	37.5	40.1	37.3	----	----	----
1-yr survey	11.1	7.8	4.2	4.7	6.5	5.6	38.7	21.4	28.3	27.5	33.0	44.6	36.2	----	----	----
2-yr survey	11.0	9.1	4.9	5.1	4.8	3.2	51.2	17.4	26.4	26.7	35.7	44.4	37.5	----	----	----
3-yr survey	12.1	7.0	5.5	3.9	5.6	2.6	40.8	22.4	31.2	----	37.5	40.6	35.6	----	----	----
4-yr survey	11.5	7.5	2.8	3.7	7.0	3.3	33.2	24.7	33.6	----	36.1	43.1	35.7	----	----	----
5-yr survey	----	9.7	----	5.1	----	3.7	40.9	26.3	26.8	----	----	----	----	4.46	5.15	4.31
6-yr survey	----	7.3	----	3.6	----	3.0	----	----	----	----	----	----	----	----	----	----
7-yr survey	----	7.7	----	4.1	----	3.1	----	25.6 ^a	28.1	----	36.0	44.2	37.0	4.56	5.11	4.19
8-yr survey	----	6.5	----	3.4	----	2.6	----	33.4 ^b	32.7	----	37.0	42.5	37.8	4.51	5.12	4.35
9-yr survey	----	7.1	----	4.0	----	2.4	----	23.5 ^c	23.6	----	36.0	44.0	38.3	3.77	4.69	4.10
10-yr survey	----	7.5	----	3.6	----	3.1	----	32.4	41.5	----	37.0	43.0	38.3	----	----	----
11-yr survey	----	7.1	----	3.8	----	2.7	----	33.5	34.7	----	37.5	46.0	37.6	4.33	5.09	4.11
12-yr survey	----	6.2	----	3.3	----	2.3	----	----	----	----	38.5	44.2	37.8	----	----	----
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	39.6	46.0	39.9	----	----	----
Rita cont. 6 mo	10.7	7.6	5.4	5.2	4.7	3.7	35.0	27.3	30.9	30.4	----	----	----	----	----	----
Rita cont. 1 yr	----	----	----	----	----	----	37.5	24.5	29.4	27.6	----	----	----	----	----	----
Rita cont. 2 yr	14.0	8.9	7.0	4.4	5.6	3.6	35.5	24.2	31.2	29.5	38.9	42.1	39.8	----	----	----
Rong. cont. 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	----	35.6	41.0	35.9	----	----	----
Rong. cont. 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	----	35.5	42.8	35.1	----	----	----
Rong. cont. 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	----	----	----	----	4.60	4.80	4.40
Rong. cont. 7 yr	----	7.8	----	4.2	----	3.1	----	28.5 ^a	31.4	----	37.2	44.4	37.0	4.52	4.68	4.12
Rong. cont. 8 yr	----	7.7	----	4.2	----	2.9	----	34.8 ^b	34.5	----	38.3	44.1	39.0	4.60	4.90	4.47
Rong. cont. 9 yr	----	7.7	----	3.9	----	3.1	----	29.1 ^c	32.5	----	39.4	43.8	38.3	4.33	4.50	4.13
Rong. cont. 10 yr	----	9.1	----	4.8	----	3.5	----	35.4	37.9	----	37.4	44.1	38.3	----	----	----
Rong. cont. 11 yr	----	7.3	----	3.9	----	2.8	----	28.1	28.3	----	39.6	44.4	37.6	4.65	4.71	4.14

^a Includes all males >7.^b Includes all males >8.^c Includes all males >9.

PATIENT NO. 33 (continued)

Six months later, in spite of being on thyroid hormone therapy, the thyroid gland had enlarged and a hard 5 mm nodule was palpable in the lower left lobe with irregularity of the remainder of the gland. She had been thought to be euthyroid. In 1965 her PBI was 7.0 $\mu\text{g}\%$. However, in March, 1966, a thyroxin iodine level of only 3.1 $\mu\text{g}\%$ was obtained, and it was thought that her Achilles reflexes were somewhat sluggish. Her TSH level was elevated.

Physical Examination: The patient appeared well developed and slightly older than her stated age of 13. A firm 1 cm nodule was palpated in the left lateral thyroid area which was freely movable. There was no regional adenopathy palpable. The remainder of the physical examination was essentially negative.

Laboratory and X-Ray Data: Thyroid Studies: PBI was 6.2 $\mu\text{g}\%$ with thyroxin iodine 4.5 $\mu\text{g}\%$; T_3 was 12.8%. BMR was -32 and -19; antithyroglobulin titre under 1:16; thyroid scan with $^{99\text{m}}\text{Tc}$ showed slight asymmetry of the gland with the right lobe being slightly larger than the left. There was a suggestion of a "cold" nodule in the isthmus. ^{132}I uptake studies showed normal uptake and normal increase following TSH stimulation. Cholesterol was normal (138 mg% with esters 119 mg%). The sedimentation rate was slightly elevated as is usual with the Marshallese. The homogram, chest plate, EKG, as well as other laboratory findings were all within normal limits.

Hospital Course: The patient was taken to the New England Deaconess Hospital in Boston, Massachusetts, on June 5, 1966. On June 6, Dr. Bentley P. Colcock of Lahey Clinic carried out a bilateral subtotal thyroidectomy, removing multinodular thyroid tissue from both lobes. One nodule was white and firmer than the rest in the left lobe and proved to be Hurthle cell adenoma without evidence of invasion of the capsule. Dr. W. A. Meissner of the New England Deaconess Hospital examined these tissues and his pathological diagnosis was "adenomatous goiter, both lobes; small follicular adenoma, Hurthle cell type right lobe; one negative parathyroid, left; fragment of thymus." Recovery from surgery was uneventful, and the patient was transferred back to this hospital on June 11. She was started on thyroid hormone therapy (desiccated thyroid), 3 grains daily, indefinitely. By June 16 her wound had healed nicely, she was asymptomatic, and she was discharged in order to return to the Marshall Islands.

Diagnosis: (1) Adenomatous goiter. (2) Small follicular adenoma, Hurthle cell type, right lobe.

Discharge Medication: To continue on thyroid hormone therapy indefinitely.

This patient was seen in September, 1966, in the Marshall Islands, and she was found to be euthyroid on the hormone treatment, with no complications.

5008398

APPENDIX 10

Utirik Group Mean Blood Counts at Various Times After Exposure

Postexposure Day	WBC (X10 ⁻³)		Neutrophils (X10 ⁻³)		Lymphocytes (X10 ⁻³)		Platelets (X10 ⁻⁴)			Hematocrit %			RBC (X10 ⁻⁶)			
	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female All Ages	Male <15	Male >15	Female All Ages	Male <15	Male >15	Female All Ages	
4	9.4	8.2	4.7	4.2	4.9	3.2										
14	10.0	8.6	4.1	3.2	5.1	2.9										
19							38.9	28.1	35.6	39.9						
29	10.1	9.7	4.9	5.8	4.8	3.2	34.5	25.6	31.7	39.9	45.1	39.4				
3-yr survey	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	35.6	41.0	35.9				
9-yr survey		7.6		3.9		3.0		35.6*	38.9	37.9	42.4	37.7	4.42	4.39	4.12	
12-yr survey		8.1		4.5		3.0				39.8	45.1	39.9				

*Includes all males >9.

5008399

APPENDIX 11

Individual Hematological Findings, 1965

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
<u>Rongelap Exposed Males 11-15</u>												
2	328	7.36	3.39	3.31	0.07	0.59	0	40	437	13.6	8.1	
3	285	13.00	5.72	3.38	0.65	3.12	1.30	37	429	12.8	9.0	33
5	333	7.62	4.80	2.59	0.15	0.08	0	39	426	12.8	7.9	33
19	329	7.84	6.59	0.63	0.08	0.55	0	46	584	14.4	7.4	7
23	215	10.10	4.04	4.04	0.10	1.82	1.00	40		12.8	8.4	
32	293	9.72	3.79	4.76	0.39	0.78	0	40	448	12.8	7.5	9
54	348	6.87	2.95	3.09	0.14	0.69	0	39	453	12.4	7.2	11
83*	363	7.62	3.20	3.58	0.38	0.38	0.70	38	449	13.2	7.6	27
85*	205	8.30	3.32	3.57	0.17	1.00	2.50	40	497	13.2	7.8	25
Mean	300	8.71	4.20	3.21	0.24	1.00	0.61	39.9	465	13.1	7.9	20.7
	±54**	1.8	1.1	1.1				2.5	50	0.6	0.5	6.5
<u>Ailingnae Exposed Males 11-15</u>												
6	329	7.82	1.88	4.38	0.47	0.94	1.50	39	445	12.8	7.4	19
84*	568	6.36	2.80	2.93	0.13	0.51	0	36	421	11.8	7.8	
Mean	449	7.09	2.34	3.66	0.30	0.73	0.75	37.5	433	12.3	7.6	19.0
<u>Rongelap Exposed Females 11-15</u>												
17	272	7.68	3.23	3.99	0.15	0.15	1.50	33	388	11.5	8.5	28
21	290	8.19	4.83	2.78	0.16	0.41	0	36	421	10.9	8.3	
33	393	12.10	7.02	4.48	0.12	0.36	1.20	42	398	14.0	7.9	40
42	331	8.38	4.78	3.18	0.17	0.25	0	38	402	13.6	7.3	24
65	353	6.29	2.64	2.89	0	0.69	0.60	37	402	12.1	7.2	27
69	272	6.47	1.49	4.27	0.13	0.45	1.30	39	402	12.4	8.3	
86*	318	8.09	4.45	3.07	0.24	0.24	0.80	38	441	12.8	7.4	20
Mean	318	8.17	4.06	3.52	0.14	0.36	0.77	37.6	408	12.5	7.8	27.8
	±41	1.8	1.7	0.7				2.6	16	1.0	0.5	
<u>Ailingnae Exposed Females 11-15</u>												
8	383	6.52	4.50	1.50	0.13	0.33	0.60	38	411	13.6	7.0	19

*Exposed in utero.

**Standard deviation.

5008400

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
<u>Rongelap Exposed Males >15-40</u>												
10	320	7.35	3.97	2.42	0.37	0.51	0.70	44	488	14.8	7.4	25
20	276	6.81	3.75	2.86	0.14	0.07	0	49	535	16.0	7.3	16
27	183	6.73	1.88	4.23	0.34	0.27	0	40	429	14.0	7.2	19
35	258	7.86	4.79	2.51	0.24	0.24	0.80	52	510	16.0	7.6	
36	222	10.28	6.07	3.39	0.41	0.41	0	46		13.6	8.5	
37	230	6.24	3.06	2.56	0.06	0.44	1.20	44	456	14.4	7.2	
40	353	7.36	3.31	3.31	0.29	0.44	0	42	437	12.6	6.6	3
47	227	7.90	4.50	2.61	0.16	0.63	0	45		14.8	7.9	
73	210	7.64	5.04	2.55	0	0.15	0	49	528	11.8	7.5	5
76	183	6.77	3.32	2.84	0.20	0.34	0.70	46		13.6	7.7	
77	253	7.35	3.67	3.01	0.51	0.15	0	49	523	16.4	8.5	25
Mean	247	7.48	3.94	2.93	0.25	0.33	0.31	46.0	488	14.5	7.6	15.5
	±51	1.0	1.1	0.5				3.3	39	1.7	0.5	8.7

Rongelap Exposed Females >15-40

12	237	7.71	3.31	3.54	0.31	0.46	0.80	42	453	13.2	7.8	40
14	293	7.24	3.40	3.33	0.07	0.43	0	36	376	12.1	7.5	
15	412	6.26	2.44	2.88	0.25	0.69	0	33	342	10.0	7.6	21
18	239	5.00	3.15	1.35	0.10	0.40	0	35	386	11.2	6.8	22
24	213	7.10	3.34	2.98	0.71	0.07	0	34	358	10.6	7.2	39
39	230	7.78	4.75	2.65	0.08	0.31	0	39	402	12.4	7.5	43
49	275	7.10	4.05	3.48	0.16	0.41	0	36	399	11.7	7.7	35
61	462	9.44	4.63	4.34	0.19	0.28	0	38	404	12.1	7.8	41
66	212	4.54	1.45	2.81	0.05	0.23	0	37	392	12.1	7.5	29
67	238	5.55	4.16	0.78	0.33	0.17	1.10	43		14.0	8.1	
71	143	9.62	4.71	4.14	0.38	0.38	0	42	454	14.8	8.0	46
72	272	7.54	4.75	2.11	0.38	0.22	0.80	35	379	12.4	7.5	
74	260	10.70	5.99	3.53	0.21	0.96	0	43	472	14.8	8.8	
75	129	6.55	3.47	1.83	0	1.11	0	42		13.2	8.3	
Mean	258	7.37	3.83	2.84	0.23	0.44	0.19	38.2	401	12.5	7.7	35.1
	±91	1.7	1.1	1.0				3.5	38	1.4	0.5	8.5

5008401

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
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Ailingnae Exposed Females >15-40

48	319	7.12	4.91	1.92	0.21	0.07	0	39	433	13.6	7.7	37
51	430	6.57	3.48	2.76	0.07	0.20	0.70	40	401	12.6	7.7	41
53	425	7.16	4.01	2.94	0.14	0.07	0	39	434	12.4	8.3	49
70	273	8.90	6.59	1.69	0.36	0.27	0	27	462	7.3	9.0	19
81	287	8.37	5.36	2.51	0.17	0.33	0	35	358	11.5		
Mean	347	7.62	4.87	2.36	0.19	0.19	0.14	36.0	418	11.7	8.2	36.5

Rongelap Exposed Males >40

4	223	7.18	2.80	3.95	0.14	0.29	0	44	503	15.2	7.6	25
7	418	5.70	2.45	2.79	0.23	0.17	0.60	41	433	13.6	7.7	26
11	307	4.38	2.54	1.58	0.13	0.13	0	37	368	12.1	6.7	
55	227	5.34	1.82	3.10	0.32	0.05	0.50	30	323	9.7	7.2	25
68	193	7.91	3.56	3.24	0.08	1.03	0	45	448	15.2	7.4	33
79	120	6.07	2.49	3.10	0.36	0.12	0	49	493	16.0	7.7	18
80	212	6.19	3.03	2.85	0.19	0.06	0.60	42	440	14.0	7.2	34
82	260	5.48	2.30	2.47	0.11	0.60	0	42	445	14.0	7.7	32
Mean	245	6.03	2.62	2.88	0.19	0.31	0.21	41.3	432	13.7	7.4	37.6
	±82	1.0	0.5	0.7				5.3	56	1.9	0.3	5.3

Ailingnae Exposed Males >40

16	259	5.38	2.64	2.10	0.22	0.33	1.10	46	5.88	14.0	7.4	22
29	215	9.04	4.88	3.53	0.27	0.36	0	49	505	16.4	8.3	31
41	278	5.72	2.34	3.15	0.11	0.11	0	42	457	14.4	7.0	
50	360	9.45	6.43	1.89	0.66	0.37	0.90	46	485	15.2	7.9	29
Mean	278	7.40	4.07	2.66	0.32	0.29	0.50	46.0	509	15.0	7.7	27.3

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
<u>Rongelap Exposed Females >40</u>												
13	335	5.52	2.93	1.82	0.22	0.50	0.60	32	329	10.0	6.9	37
34	296	6.79	3.26	3.19	0.13	0.20	0	37	315	11.2	6.9	43
58	233	5.57	2.28	2.95	0.11	0.22	0	39	389	13.6	7.7	29
60	231	8.28	3.15	4.06	0.08	0.99	0	33	354	10.6	7.5	33
63	283	5.54	2.99	1.72	0.22	0.55	0.60	42	433	14.0	7.3	
64	292	5.35	2.62	2.35	0.21	0.11	0.50	37	388	12.4	7.9	21
78	473	8.03	5.06	2.65	0.32	0	0	40	388	13.2	7.7	26
Mean	306	6.44	3.18	2.67	0.18	0.37	0.24	37.1	371	12.1	7.4	31.5
	±76	1.1	0.8	0.8				3.5	39	1.4	0.4	7.2
<u>Ailingnae Exposed Females >40</u>												
1	383	6.35	2.92	2.67	0.32	0.32	1.30	41	438	13.6	8.0	46
28	228	5.89	2.06	3.42	0	0.41	0	41	393	13.2	8.4	47
45	255	4.84	2.08	2.08	0.05	0.62	0	37	380	12.1	7.7	37
59	484	8.51	3.49	4.26	0.17	0.60	0	39	402	13.2	8.5	41
Mean	338	6.40	2.63	3.10	0.14	0.49	0.32	39.5	403	13.0	8.2	42.8
<u>Male Children of Exposed Parent(s) Age <11</u>												
88	353	7.11	2.70	3.84	0.21	0.36	0	35	399	11.8	7.2	19
89	355	7.63	1.90	4.50	0.23	0.99	0	38	372	13.2	6.8	3
90	422	9.72	6.03	2.72	0.49	0.49	0	35	400	11.8	6.6	
91	368	9.01	4.60	3.87	0.27	0.27	0	38	421	13.2	7.8	28
93	400	15.80	8.85	5.06	0.47	1.42	0	37	409	11.8	7.6	28
96	285	8.52	3.75	3.83	0.26	0.51	1.70	37	421	11.2		
97	313	7.44	4.09	2.98	0.15	0.15	0.70	39	470	12.4		
98	285	8.59	3.95	3.78	0.34	0.43	0.90	37	445	11.5		15
104	430	10.60	4.45	5.62	0.21	0.32	0	37	470	12.4	7.6	23
109	498	11.20	5.49	4.59	0.56	0.56	0	33	402	11.2		
110	395	10.00	5.50	3.20	0.60	0.60	1.00	37	505	12.1		7

5008403

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
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Male Children of Exposed Parent(s) Age < 11 (cont'd)

111	630	10.10	5.25	4.25	0.20	0.40	0	33	420	10.6	7.2	23
113	450	8.95	5.01	2.95	0.81	0.81	0	39	493	12.4		
115	443	14.10	7.05	6.10	0.42	0.99	0	35	370	11.5	7.3	
116	570	12.70	7.62	3.56	0.76	0.76	0	42	497	13.2		
118	515	10.16	2.84	6.81	0	0.51	0	39	447	12.8		
126	510	6.35	3.56	2.22	0.25	0.32	0	36	456	10.9		
130	465	8.60	1.55	5.93	0.26	0.69	1.70	35	456	8.8		
131	348	7.66	3.45	3.52	0.30	0.30	0.80	42	479	13.2		
132	490	10.60	1.38	8.90	0.21	0.11	0	34	431	11.5		
Mean	426	9.74	4.45	4.41	0.32	0.55	0.34	36.9	438	11.9	7.3	18.3
	+90	2.3	1.9	1.7				2.5	+40	1.0	0.4	8.7

Female Children of Exposed Parent(s) Age < 11

87	300	5.50	0.99	3.41	0.22	0.83	0.60	38	448	12.8		
92	273	11.20	7.50	2.24	0.22	1.23	0	39	469	12.8	7.5	33
94	310	11.70	8.89	2.11	0.23	0.47	0	43	515	14.4		36
100	208	6.84	3.90	2.53	0.07	0.34	0	36	402	12.4		26
101	350	14.90	2.68	10.13	0.15	1.79	1.50	39	445	12.8		
103	535	12.40	7.07	4.84	0	0.50	0	35	482	12.1	6.5	23
105	225	11.00	5.39	3.41	0.55	1.65	0	37	457	11.2	7.2	25
106	435	8.08	3.07	4.12	0.16	0.73	0	40	460	13.2		34
108	425	10.35	5.07	3.31	0.21	1.66	0	40		12.1		
112	431	7.15	1.72	5.14	0	0.29	0	34	426	10.9	7.5	24
117	573	8.16	2.77	4.73	0.16	0.33	1.60	39	402	11.5		
119	199	9.43	4.90	3.68	0.19	0.66	0	35	434	12.1		
120	290	9.35	3.84	5.32	0.30	0.39	0	39	467	13.2		
121	360	10.67	5.01	3.84	0.21	1.28	3.20	38		10.9		
122	363	8.25	2.81	4.95	0.17	0.33	0	44	400	11.8		23
123	333	10.47	6.91	2.51	0.31	0.63	1.00	36		10.0		
124	430	14.60	6.28	7.74	0.15	0.29	1.50	38	454	12.4		
125	543	8.41	3.53	3.70	0.50	0.67	0	35	437	12.1		
127	512	9.30	1.21	7.63	0.09	0.37	0	38	414	12.8		
128	358	11.40	4.79	4.90	0.57	1.14	0	42	500	13.6		
134	313	9.90	4.55	4.26	0.30	0.79	0	33	399	10.0		
135	975	16.30	9.62	5.22	0.33	0.82	3.30	34	490	9.0		
137	407	14.20	2.84	10.79	0.28	0.14	1.40	31	402	10.6		
138	460	9.09	2.91	5.27	0.18	0.64	0.90	33	448	10.6		

119

5008404

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
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Female Children of Exposed Parent(s) Age <11 (cont'd)

139	362	14.80	6.66	7.55	0.30	0.30	0	31	374	10.3		
140	525	10.90	2.07	7.96	0.44	0.33	1.10	49	526	15.2		
143	546	8.26	3.55	4.30	0.17	0.25	0	33	381	10.0		
145	427	10.31	2.17	5.67	0.21	2.17	1.00	36		10.6		
Mean	410	10.48	4.38	5.04	0.24	0.75	0.61	37.3	443	11.8	7.2	28.0
	+150	2.6	2.2	2.2				4.0	41	1.4		5.0

Unexposed Males 11 - 15

813	335	7.22	3.61	2.89	0.14	0.58	0	35	411	12.1	6.9	13
814	385	9.95	5.97	3.08	0.30	0.60	0	41	454	13.6	8.3	34
815	124	5.40	2.32	2.65	0.22	0.22	0	43	498	14.4	8.2	30
817	208	7.96	3.90	2.79	0.32	0.88	0.80	39	454	12.8	7.2	
818	336	7.24	4.49	2.17	0.14	0.43	0	43	498	14.0	7.5	17
863	269	5.76	3.23	1.70	0.23	0.45	0.60	47	515	15.6	7.7	30
912	333	7.06	3.74	2.33	0.14	0.78	0.70	33	409	11.5	7.5	19
913	413	5.78	3.76	1.21	0.23	0.46	1.20	39	518	13.2	7.6	17
921	385	6.95	3.96	1.39	0.35	1.11	1.40	39	467	12.4	7.6	17
931	425	10.60	4.35	4.66	0.11	1.38	1.10	38	445	12.4	7.6	25
981	343	7.07	3.11	3.68	0.21	0.07	0	38	444	13.2	8.0	
1033	243	5.40	2.05	3.19	0.11	0	0.50	42	505	14.0	7.9	33
1036	240	6.83	3.28	3.01	0.27	0.27	0	39	469	13.6	7.5	20
1052	523	6.99	3.43	3.22	0.21	0.07	0.70	38	429	12.8	7.2	16
Mean	326	7.16	3.66	2.71	0.21	0.52	0.50	39.6	465	13.3	7.6	22.6
	+ 98	1.5	0.9	0.9				3.3	36	1.0	0.4	7.0

Unexposed Females 11 - 15

805	433	6.64	1.39	3.72	0.40	1.00	0.13	39	477	12.8	8.0	20
811	412	7.74	3.10	3.79	0.23	0.62	0	34	393	12.4	7.4	0
812	330	7.11	3.48	2.92	0.14	0.57	0	39	473	11.8	8.0	27

5008405

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
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Unexposed Females 11 - 15 (cont'd)

816	305	5.31	3.29	1.43	0.21	0.32	0.50	38	420	12.1	7.4	24
879	257	9.00	3.60	4.14	0.09	1.17	0	39	441	12.8	8.5	25
909	228	10.60	5.19	3.50	0.42	1.48	0	40	426	12.1		27
911	373	6.96	4.32	1.95	0.28	0.42	0	35	416	11.5	8.0	39
925	250	7.85	4.63	2.36	0.31	0.55	0	40		12.4	9.3	
926	268	9.52	4.76	4.28	0	0.48	0	40	521	13.6	8.0	26
937	210	6.70	3.75	2.35	0.27	0.34	0	40		13.2	8.0	
946	258	8.90	5.70	2.67	0.27	0.27	0	40	442	14.0	7.7	44
955	262	7.07	4.74	1.48	0.14	0.70	0	39	405	12.4	8.1	40
960	266	14.00	11.59	1.82	0.28	0.42	0	34	388	11.2	7.3	28
962	273	8.13	4.07	2.68	0.16	1.06	1.60	37	449	11.8	8.4	
978	288	10.80	4.75	4.64	0.22	1.08	1.10	40	467	14.4	8.0	
980	308	6.29	3.46	2.39	0	0.44	0	43	477	13.6	7.6	28
996	232	8.80	5.72	2.11	0.26	0.70	0	37	420	11.5	7.8	38
1035	438	9.88	3.85	5.43	0.10	0.49	0	40	442	12.8		27
Mean	300	8.39	4.52	2.98	0.21	0.67	0.19	38.6	441	12.6	8.0	28.1
	+ 68	2.1	2.0	1.1				2.4	34	0.9	0.5	10.4

Unexposed Males >15-40

819	210	5.78	2.54	2.60	0.06	0.58	0	39	442	12.4	7.5	
820	240	6.60	2.71	3.10	0.13	0.66	0	48	528	15.2	7.9	13
822	225	5.86	2.40	3.03	0.18	0.18	0.60	44	483	14.0	7.0	9
823	270	5.25	2.94	1.68	0.32	0.26	0.50	45	453	14.4	7.0	18
827	258	6.46	3.62	2.45	0.06	0.32	0	47	486	14.4	7.8	8
830	168	5.87	3.52	1.53	0.41	0.41	0	44	449	14.8	7.0	18
831	220	7.45	2.91	3.50	0.45	0.60	0	52		15.2	7.7	8
833	328	4.69	1.74	2.44	0.23	0.23	0.50	48	555	16.0	7.5	
836	239	9.80	5.19	3.72	0.10	0.78	0	48	539	16.0	8.2	14
840	318	8.01	4.17	3.28	0.24	0.24	0.80	48	586	16.0		9
845	228	6.12	2.69	3.06	0.12	0.18	0.60	46	481	14.4	7.2	14
864	344	7.17	2.29	4.44	0.07	0.29	0.70	46	469	15.6	7.3	15
881	214	8.65	2.85	4.84	0.43	0.52	0	44	459	14.8	8.0	32
882	309	5.74	3.04	2.35	0.06	0.29	0	44	506	14.8	7.3	10

5008406

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
<u>Unexposed Males >15-40 (cont'd)</u>												
885	213	20.75	14.73	3.74	1.25	1.04	0	54		16.9	8.3	
892	240	11.00	4.84	5.17	0.44	0.44	1.10	44	497	14.8	8.6	
920	210	9.32	6.06	2.24	0.47	0.56	0	50	505	16.9	7.9	
940	283	7.82	2.97	3.75	0.39	0.70	0.80	43	509	14.8	8.2	
943	220	10.45	5.43	4.18	0.52	0.31	0	48		15.2	7.9	
966	268	8.57	6.08	2.14	0.17	0.17	0	45	462	13.6	7.2	18
967	208	6.67	4.00	2.00	0.13	0.53	0	47	479	14.4	7.3	9
971	259	5.63	3.49	1.91	0.17	0	0.60	47		14.4	7.9	
976	232	8.28	4.30	3.64	0.33	0	0	45	527	15.2	7.3	
1501	222	6.80	3.94	2.38	0.20	0.27	0	45	510	15.2	8.0	19
Mean	247	7.86	4.10	3.05	0.29	0.40	0.26	46.3	496	14.9	7.6	14.3
	± 42	3.1	2.5	1.0				4.9	37	1.0	0.4	6.1

Unexposed Females >15-40

821	371	7.27	2.84	3.78	0.29	0.36	0	36	399	12.1	7.2	
826	175	7.27	5.38	1.82	0.07	0	0	30		8.5		
829	219	5.66	4.08	1.36	0	0.23	0	28	295	8.8	7.1	25
832	321	6.56	3.80	1.77	0.26	0.72	0	39	435	12.1	7.9	34
835	265	6.80	2.85	3.60	0.14	0.20	0	36	414	12.4	6.7	13
841	223	6.56	4.79	1.38	0.20	0.20	0	30	348	9.4	7.1	34
843	235	4.73	2.19	2.10	0.19	0.29	0	32	324	10.9	6.5	10
865	268	6.22	3.98	1.74	0.12	0.19	0.60	28	306	10.3	6.4	23
891	258	6.44	2.51	3.16	0.64	0.06	0.60	42	437	14.8	7.8	
895	303	9.76	4.68	4.10	0.39	0.59	0	43	467	14.8	7.2	
896	342	6.76	3.52	2.70	0.14	0.41	0	35	388	13.2	7.0	36
914	305	7.28	3.71	2.62	0.22	0.73	0	35	391	11.5	6.8	
932	290	5.06	2.73	1.77	0.10	0.46	0	36	392	11.5	7.5	25
934	326	7.86	3.38	4.24	0.08	0.16	0	38	442	13.2	8.1	27
938	271	7.49	5.39	1.80	0.15	0.15	0	36	409	12.1	7.6	
945	278	11.40	9.12	1.60	0.23	0.46	0	47	506	15.6	8.2	
951	208	7.15	2.86	4.15	0.07	0	0.70	45		13.2	7.8	

5008407

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
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Unexposed Females > 15 - 40 (cont'd)

959	225	6.56	3.67	1.84	0.39	0.66	0	38	400	12.8	7.3	34
993	201	5.23	2.30	2.46	0.16	0.21	1.00	42		12.4	7.9	
998	317	8.33	4.58	3.08	0.17	0.50	0	35	412	12.8	8.3	
1001	226	5.06	3.14	1.62	0.10	0.20	0	39	460	12.4	7.3	19
1050	333	8.74	4.80	3.58	0.09	0.26	0	36	389	12.1	8.0	37
1502	216	6.41	2.43	3.27	0.19	0.38	1.30	36	393	13.2	8.2	36
Mean	269	6.98	3.86	2.59	0.19	0.32	0.18	36.6	400	12.2	7.4	27.2
	± 51	1.5	1.5	0.9				5.0	51	1.7	0.6	8.7

Unexposed Males > 40

842	235	9.74	4.31	4.01	0.10	1.27	1.00	52	503	17.9	7.9	16
849	273	11.40	5.36	4.45	0.11	1.48	0	48	515	16.0	8.1	3
850	117	5.96	2.80	2.68	0.24	0.24	0	43	453	14.0	7.8	22
853	273	5.77	2.42	3.00	0.23	0.06	0	41	429	13.2	8.3	33
855	265	10.40	7.59	2.70	0.10	0	0	44	421	14.0	8.0	33
856	235	6.07	2.31	2.73	0.06	0.97	0	38	407	12.8	7.5	23
860	373	5.00	2.25	1.85	0.15	0.75	0	35	376	11.2	8.0	30
875	218	6.31	4.29	1.70	0.06	0.25	0	52	528	16.4	7.7	9
878	194	4.90	1.76	2.40	0.15	0.54	0.50	39	465	12.1	9.0	25
880	265	9.06	5.07	3.90	0	0.09	0	46	518	15.6	7.8	
884	283	8.95	5.64	2.69	0.27	0.36	0	40	442	12.1	8.8	35
897	266	8.24	4.70	3.05	0.16	0.41	0	42	441	14.0	9.0	
899	240	5.10	2.75	1.99	0.15	0.20	0	39	407	12.4	7.4	
915	240	4.16	1.87	1.62	0.08	0.50	0.80	41	425	15.6	7.4	15
917	215	8.89	6.67	1.87	0.27	0.09	0	49	570	16.4	8.3	10
935	223	5.39	2.47	2.43	0.32	0.16	0	41	423	14.0	8.1	34
947	249	7.76	5.43	1.62	0.23	0.47	0	37	382	12.1	7.1	20
948	245	5.17	2.17	2.59	0.26	0.16	0	40	433	14.0	7.0	17
961	151	8.55	3.76	2.91	0.26	1.54	0.90	44		14.4	8.2	
963	205	5.10	1.84	2.75	0.15	0.36	0	49		15.2	7.2	
964	243	4.38	2.54	1.66	0.04	0.13	0	37	351	12.4	7.2	31
969	505	9.75	6.44	2.73	0.29	0.29	0	40	492	11.8	8.3	19

5008408

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
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Unexposed Males > 40 (cont'd)

1007	290	6.41	3.85	2.05	0.26	0.19	0.60	41	448	12.8	8.0	26
1041	147	4.94	2.47	2.17	0.10	0.15	0.50	42	450	14.6	8.0	32
Mean	248	6.98	3.78	2.56	0.17	0.44	0.18	42.5	449	14.0	7.9	22.8
	± 74	2.1	1.7	0.7				1.4	53	1.0	0.5	9.2

Unexposed Females >40

844	323	4.71	2.69	1.42	0.19	0.42	0	37	421	11.5	7.3	34
846	425	6.68	2.81	3.27	0.47	0.13	0	40	431	13.2	8.6	41
851	218	5.81	2.32	3.25	0.12	0.06	0.60	35	376	12.4	7.0	28
852	330	8.97	3.23	5.38	0.18	0.09	0.90	38	423	11.8	8.0	39
858	293	4.81	2.31	1.97	0.14	0.38	0	37	402	11.8	7.5	41
859	410	7.93	4.83	2.70	0	0.40	0	39	390	12.4	7.5	30
898	197	8.67	4.68	3.38	0.17	0.43	0	40	414	12.8	8.3	
908	238	6.90	1.52	5.11	0.07	0.21	0	44	401	14.4		
916	311	7.86	2.67	4.56	0.16	0.47	0	38	405	14.0	7.8	39
922	260	6.81	3.95	1.97	0.27	0.62	0	36		10.6	8.5	
928	325	5.84	3.50	2.10	0.18	0.06	0	35	366	11.2	8.3	43
929	242	5.35	2.46	2.68	0.21	0	0	36		12.4	8.5	
936	262	8.20	3.85	4.18	0	0.16	0	37	391	13.2	8.4	39
941	245	6.43	3.86	2.12	0.13	0.32	0	38	407	12.1	7.6	40
942	215	4.95	2.38	2.23	0.15	0.20	0	40	414	13.2	8.0	40
956	323	4.40	1.98	1.98	0.09	0.35	0	35	386	11.5	7.2	37
957	328	6.57	2.76	3.29	0	0.46	0.60	36	377	12.1	7.4	39
970	353	8.00	2.32	5.52	0	0.08	0.80	38	433	12.4	8.0	38
982	200	8.50	4.42	3.66	0.09	0.26	0.90	40	452	13.2	7.5	
991	223	7.94	6.11	1.35	0.08	0.40	0	42	460	16.0	7.5	
1042	235	6.93	4.02	1.80	0.62	0.49	0	36	392	12.1	7.4	34
Mean	284	6.77	3.27	3.04	0.16	0.29	0.18	38.0	407	12.6	7.8	37.5
	± 64	1.4	1.1	1.3				2.3	24	1.2	0.5	4.0

5008409

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
<u>Unexposed Males <11</u>												
801	305	7.86	1.89	4.72	0.16	1.10	0	39	459	12.8	7.4	10
802	403	8.23	5.18	2.80	0	0.25	0	37	435	12.1	6.5	6
803	273	13.60	7.21	4.90	0.14	1.36	0	37	409	12.4	7.9	20
806	270	9.19	3.95	4.32	0.18	0.64	0.90	37	433	12.1	8.0	26
807	267	9.48	5.50	3.03	0.76	0.19	0	35	414	11.5	6.8	11
809	268	9.40	5.17	2.82	0.47	0.85	0.90	33	389	11.2	8.2	26
870	418	9.46	4.45	3.03	0.47	1.42	0.90	39	465	12.4	7.4	16
904	434	9.20	3.59	4.69	0.37	0.55	0	37	412	12.1	7.6	
905	350	7.38	3.25	3.17	0.22	0.59	1.50	37	468	12.1	7.3	20
952	428	9.55	3.82	5.06	0.29	0.19	1.90	35	421	11.8		
972	363	6.74	2.63	3.24	0.13	0.74	0	39	470	12.4	7.9	32
1004	548	13.70	6.30	5.62	0.41	1.23	1.40	35	401	11.8	7.6	23
1006	218	12.30	5.41	5.53	0.37	0.98	0	40	452	12.4	8.1	
1009	312	6.57	2.30	3.15	0.39	0.72	0	38		12.1		
1010	481	9.79	6.17	3.23	0.20	0.20	0	36	423	12.1	8.1	30
1013	238	13.55	7.45	4.47	0.27	1.36	0	35		10.9		
1014	288	6.83	2.05	4.17	0.14	0.41	0.70	35	423	11.2	7.5	
1015	194	10.40	4.58	4.47	0.21	1.04	1.00	35		11.2	7.2	
1018	485	11.20	5.49	4.59	0.22	0.78	2.20	36	472	10.6		13
1024	245	6.74	3.84	2.63	0.07	0.20	0	35	459	11.8	7.4	11
1027	393	6.24	1.68	4.37	0.12	0.06	0	38	420	11.5		
1028	525	11.60	5.34	5.45	0.23	0.58	0	37	426	11.2		
1029	330	10.30	4.43	5.56	0.31	0	0	39	486	11.8		
1030	265	16.30	6.85	6.85	0.49	1.96	1.60	39	465	13.2		
1032	507	6.91	3.46	3.11	0.21	0.14	0	40	473	14.0	7.7	25
1037	449	6.65	2.19	4.19	0.13	0.07	0.70	36	401	12.1		
1038	459	8.25	4.54	2.06	0.17	1.49	0	35	396	10.3		
1039	545	15.00	10.05	4.80	0.15	0	0	38	460	12.8		
1040	348	7.86	5.90	1.41	0.24	0.24	0.80	36	481	11.2		
1046	283	7.87	2.36	4.49	0.08	0.94	0	35	441	10.6		
1047	334	11.20	5.04	5.82	0	0.34	0	36	463	11.5		
1049	328	15.30	7.34	7.80	0	0.15	0	38	513	12.4	7.9	23
1053	315	10.50	8.08	1.89	0	0.53	0	38	467	12.4	7.3	25
1054	420	8.75	3.06	4.99	0.18	0.53	0	40	517	12.1		
1056	530	7.61	2.59	4.49	0.23	0.15	1.50	35	444	10.0		
1058	520	13.10	5.90	6.29	0.52	0.39	0	33	518	10.0		
1059	333	10.40	5.93	4.06	0.42	0	0	35	441	12.1		
1061	347	10.80	2.59	7.13	0.32	0.43	3.20	30	391	11.8		
1062	535	8.99	1.16	7.01	0.36	0.45	0	34	434	10.9		

5008410

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Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)	Sed. Rate (mm)
<u>Unexposed Males < 11 (cont'd)</u>												
1063	145	12.10	2.18	9.56	0.24	0.12	0	32	399	10.0		
1503	245	7.66	1.99	5.13	0.38	0.15	0	37	469	12.8	7.8	30
1504	406	13.00	8.32	3.77	0.26	0.65	0	40	450	14.0		
Mean	365	9.94	4.55	4.38	0.25	0.58	0.46	36.5	445	11.8	7.6	20.4
	±106	2.6	2.1	1.6				2.4	34	0.9	0.4	7.7
<u>Unexposed Females < 11</u>												
808	378	11.10	5.88	3.33	0.44	1.33	1.10	40	452	12.4	8.7	30
810	330	9.49	4.75	3.89	0.19	0.47	1.90	36	344	12.5	7.8	16
866	218	8.19	4.42	3.19	0.33	0.25	0	40	459	13.6	7.5	32
900	430	6.88	4.33	1.65	0.28	0.55	0.70	35	395	11.5	7.6	40
901	358	7.12	3.28	2.99	0.50	0.36	0	36	437	11.8	7.6	26
902	395	6.68	2.94	3.21	0.33	0.20	0	35	414	11.8	7.6	21
903	443	9.25	3.79	4.53	0.37	0.56	0	35	416	11.5	8.2	38
906	248	6.74	2.09	4.04	0.20	0.40	0	38	459	14.0	7.9	26
923	435	11.40	5.59	4.79	0.34	0.68	0	39	456	13.6	7.8	37
930	295	13.90	5.00	6.53	0.56	1.67	1.40	38	452	13.2		
954	395	13.40	8.31	4.29	0.27	0.54	0	35	400	11.8	7.6	
979	310	9.79	4.21	4.69	0.39	0.39	1.00	39	441	12.8		25
995	223	12.11	5.69	4.71	0.48	1.20	0	35		11.5	7.7	
1008	208	9.10	3.17	4.98	0.27	0.63	0	38		12.8	7.7	
1011	425	11.80	5.42	4.84	0.47	0.94	1.20	36	468	11.8		31
1012	413	8.08	3.31	3.72	0.40	0.65	0	34	402	12.4	7.3	30
1020	402	9.64	2.89	6.46	0.19	0.10	0	31	390	10.0		13
1021	337	13.00	5.98	5.59	0.26	1.04	1.30	39	445	12.4		
1022	341	6.33	2.28	3.17	0.32	0.57	0	35	410	11.8	7.1	19
1025	423	13.60	6.94	5.58	0.68	0.41	0	38	483	12.1		
1026	413	7.07	2.26	3.89	0.28	0.64	0	35	409	10.9	7.5	36
1031	557	10.40	5.41	3.95	0.21	0.73	1.00	35	407	11.2		
1034	323	11.70	8.07	2.57	0.35	0.70	0	39	490	12.4	7.7	39
1044	388	6.58	2.63	3.36	0.33	0.26	0	34	410	9.7		
1055	415	8.86	4.25	3.46	0.53	0.53	0.90	40	483	12.1		
1057	446	15.20	4.56	10.34	0	0	1.50	33	409	9.4		
1060	363	9.10	2.37	6.19	0.18	0.27	0.90	33	407	11.2		
Mean	367	9.87	4.43	4.44	0.34	0.56	0.48	36.3	430	11.9	7.7	28.7
	± 80	2.5	1.7	1.6				2.4	35	1.1	0.4	8.1

APPENDIX 12
Individual Hematological Findings, 1966

Subject No.	Plat. (x10 ⁻³)	WBC (x10 ⁻³)	Neut. (x10 ⁻³)	Lymph. (x10 ⁻³)	Mono. (x10 ⁻³)	Eosin. (x10 ⁻³)	Baso. (x10 ⁻²)	Hct. (%)	RBC (x10 ⁻⁴)	Hgb. (g)	Serum Protein (g)
<u>Rongelap Exposed Males 12-15</u>											
2		12.84	7.44	3.85	0.51	1.03	0.0	39			
3		5.79	2.49	2.08	0.12	0.93	1.70	37			
5		6.61	2.97	2.97	0.13	0.53	0.0	39			
19		8.95	4.83	2.86	0.27	0.98	0.90			13.0	
32		6.57	2.63	2.83	0.07	0.92	1.30	41			
54		3.70	1.00	1.73	0.07	0.85	0.40	40			
83*		8.19	4.75	2.78	0.25	0.33	0.80	38			
85*		9.57	4.98	3.82	0.48	0.29	0.0	38			
Mean		7.77 +2.16**	3.88 1.9	2.86 0.8	0.24	0.73	0.64	38.8 2.2		13.0	
<u>Ailingnae Exposed Males 12-15</u>											
6		7.58	3.87	2.58	0.38	0.76	0.0	40			
84*		4.87	1.31	2.97	0.10	0.44	0.50	37			
		6.22	2.59	2.78	0.24	0.60	0.25	38.5			
<u>Rongelap Exposed Females 12-15</u>											
17		5.78	2.20	2.49	0.17	0.92	0.0	33			
21		5.74	2.58	2.41	0.17	0.46	1.10	37			
33		5.37	2.95	1.66	0.11	0.64	0.0	42			
42		6.69	3.81	1.94	0.0	0.80	1.30	38			
65		7.40	5.03	1.70	0.37	0.22	0.70	39			
86*		9.81	3.63	3.24	0.0	2.84	1.00	40			
		6.79 +1.5	3.36 0.9	2.24 0.5	0.14	0.98	0.68	38.1 3.5			
<u>Ailingnae Exposed Females 12-15</u>											
8		12.45	9.71	2.24	0.37	0.0	1.30	36			

*Exposed in utero.

**Standard deviation.

5008411

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Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)
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Rongelap Exposed Males > 15-40

9		8.04	3.38	3.94	0.24	0.32	1.60	44			
10		4.92	2.61	2.02	0.15	0.15	0.0	37			
20		5.90	2.89	2.24	0.12	0.53	1.20			16.0	
27		6.64	3.78	2.19	0.07	0.40	1.30	43			
36		7.53	3.39	3.24	0.30	0.53	0.80			15.0	
37		7.48	3.81	2.84	0.52	0.30	0.0	50			
47		4.63	2.13	1.71	0.09	0.65	0.40			16.0	
73		7.04	3.80	2.39	0.28	0.42	1.40	45			
76		6.38	2.62	3.00	0.19	0.51	0.60			14.6	
77		4.92	3.10	1.48	0.20	0.10	0.50	50			
Mean		6.35 ±1.1	3.15 0.5	2.51 0.7	0.22	0.39	0.78	44.8 3.6		15.4	

Ailingnae Exposed Males > 15-40

44		4.99	2.44	1.85	0.10	0.50	1.00	40			
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Rongelap Exposed Females > 15-40

12		5.72	3.83	1.54	0.11	0.23	0.0	34			
14		6.84	3.49	2.12	0.14	1.03	0.70	36			
15		6.36	2.99	2.73	0.19	0.38	0.60	39			
18		9.80	6.76	2.01	0.29	0.59	1.00	40			
22		7.80	4.99	1.95	0.0	0.70	1.60	31			
24		6.32						37			
39		3.80	1.29	1.94	0.15	0.30	1.10	40			
49		6.24	3.37	2.24	0.19	0.37	0.60	36			
61		6.36	3.75	2.16	0.19	0.19	0.60	41			
67		9.90	2.87	5.64	0.10	1.19	1.00			13.0	
69		6.32	2.40	3.16	0.19	0.51	0.60	41			
71		6.36	3.56	1.97	0.19	0.57	0.0	43			
74		9.72	6.03	2.43	0.19	0.97	1.00	46			
75		10.75	7.63	2.04	0.11	0.86	1.10			12.5	
Mean		7.30 ±1.9	4.07 1.7	2.46 1.0	0.16	0.61	0.76	38.7 3.9		12.8	

5008412

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5008413

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Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)
<u>Ailingnae Exposed Females > 15-40</u>											
48		7.78	3.89	3.03	0.16	0.62	0.80	40			
51		5.36	2.84	2.09	0.21	0.16	0.50	39			
53		7.68	4.15	3.00	0.08	0.46	0.0	39			
70		3.96	2.45	1.19	0.16	0.16	0.0	26			
81		5.58	3.07	1.84	0.28	0.33	0.60	40			
Mean		6.07	3.28	2.23	0.18	0.35	0.38	36.8			
<u>Rongelap Exposed Males > 40</u>											
4		5.88	1.82	3.41	0.18	0.41	0.60	43			
7		5.68	3.12	1.99	0.28	0.17	1.10	39			
11		5.88	3.41	1.76	0.29	0.41	0.0	36			
40		6.48	2.85	2.98	0.26	0.32	0.60	44			
55		4.79	2.59	1.96	0.14	0.10	0.0	33			
68		4.76	2.33	1.95	0.05	0.42	0.0	45			
79		7.55	4.30	2.87	0.15	0.23	0.0	48			
80		9.00	5.22	3.15	0.27	0.36	0.0	44			
82		6.68	3.81	2.07	0.27	0.47	0.70	40			
Mean		6.30 +1.2	3.27 1.0	2.46 0.6	0.21	0.32	0.33	41.3 3.8			
<u>Ailingnae Exposed Males > 40</u>											
16		3.64	2.07	1.42	0.11	0.04	0.0	47			
41		4.16	2.12	1.74	0.17	0.17	0.0	44			
50		9.02	3.07	5.20	0.27	0.36	0.90	46			
Mean		5.60	2.42	2.79	0.18	0.19	0.30	45.6			

5008414

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Subject No.	Plat. (x10 ⁻³)	WBC (x10 ⁻³)	Neut. (x10 ⁻³)	Lymph. (x10 ⁻³)	Mono. (x10 ⁻³)	Eosin. (x10 ⁻³)	Baso. (x10 ⁻²)	Hct. (%)	RBC (x10 ⁻⁴)	Hgb. (g)	Serum Protein (g)
<u>Rongelap Exposed Females > 40</u>											
13		7.28	4.88	1.67	0.17	0.51	0.0	37			
34		7.04	3.80	2.53	0.14	0.56	0.0	37			
58		5.96	3.10	2.32	0.24	0.24	0.60	41			
60		6.64	3.20	2.72	0.20	0.40	0.0	36			
63		4.32	1.99	2.16	0.08	0.08	0.0	43			
64		5.76	3.92	1.32	0.29	0.17	0.60	31			
66		6.20	3.22	2.48	0.12	0.37	0.0	38			
78		6.44	3.74	2.32	0.0	0.32	0.60	40			
		6.20	3.48	2.19	0.16	0.33	0.23	37.8			
		+0.9	0.8	0.4				3.3			
<u>Ailingnae Exposed Females > 40</u>											
1		5.46	3.33	1.64	0.16	0.22	0.60	41			
45		5.08	2.69	1.73	0.25	0.41	0.0	39			
59		6.04	2.47	2.17	0.24	1.09	0.60	41			
		5.52	2.83	1.85	0.22	0.57	0.40	40.3			
<u>Utirik Exposed Males 12-15</u>											
2115		12.24	6.12	5.39	0.37	0.24	1.20	42			
2102		10.47	4.29	5.44	0.31	0.21	2.10	39			
2124		5.90	2.18	2.77	0.41	0.53	0.0	38			
2136		5.80	2.78	2.61	0.17	0.23	0.0	41			
2143		5.11	2.20	2.61	0.15	0.10	0.50	41			
2151		7.53	2.86	4.21	0.30	0.0	0.80	39			
2153		9.88	4.84	4.45	0.30	0.30	0.0	36			
2155		6.27	2.19	3.76	0.13	0.19	0.0	37			
2174		10.89	6.43	3.38	0.22	0.54	3.30	39			
2179		6.26	1.94	3.44	0.31	0.56	0.0	39			
2232		7.64	3.82	3.29	0.07	0.31	0.0	41			
2236		9.33	4.94	3.73	0.19	0.37	0.90			12.1	
2242		7.40	4.44	2.29	0.30	0.37	0.0	39			
2233		5.76						46			
Mean		7.89	3.77	3.64	0.25	0.30	0.68	39.8		12.1	
		+2.2	1.5	1.0				2.4			

5008415

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)
<u>Utirik Exposed Females 12-15</u>											
2111		7.92	3.72	3.56	0.16	0.40	0.80	46			
2113		10.63	5.63	3.83	0.32	0.85	0.0	40			
2130		8.50	4.25	3.15	0.42	0.60	0.80	36			
2132		7.23	3.69	2.75	0.29	0.43	0.70			11.1	
2210		9.78	5.77	3.13	0.0	0.88	0.0	40			
2218		12.40	6.70	5.08	0.12	0.50	0.0	46			
2227		8.55	5.81	2.22	0.09	0.43	0.0	40			
2277		14.43	10.10	3.75	0.29	0.29	0.0	39			
Mean		9.93 +2.3	5.70 1.9	3.43 0.8	0.21	0.55	0.29	41.0 3.0		11.1	

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<u>Utirik Exposed Males > 15-40</u>											
2123		7.86	5.97	1.10	0.31	0.39	0.80	50			
2135		9.04	5.88	2.53	0.18	0.45	0.0	50			
2137		6.54	2.81	3.20	0.06	0.39	0.60	44			
2142		7.90	3.56	3.63	0.08	0.63	0.0	44			
2144		6.28	3.64	2.20	0.38	0.06	0.0	50			
2150		8.42	5.30	2.95	0.17	0.0	0.0	48			
2152		5.67	3.52	1.87	0.17	0.11	0.0	45			
2157		8.97	5.11	3.23	0.45	0.18	0.0	44			
2165		5.36	1.77	3.27	0.05	0.21	0.50	43			
2167		7.92	4.67	2.38	0.55	0.33	0.0	47			
2168		7.16	3.65	2.86	0.07	0.57	0.0	45			
2176		7.05	3.74	2.75	0.28	0.21	0.70	46			
2178		7.03	2.81	3.59	0.42	0.14	0.70	44			
2188		7.16	2.00	4.51	0.29	0.36	0.0	47			
2207		9.58	6.42	2.20	0.38	0.48	1.00			11.8	
2250		7.48	2.62	3.44	0.30	1.12	0.0	44			
2257		8.15	5.95	1.79	0.16	0.24	0.0	48			
Mean		7.50 +1.1	4.08 1.4	2.79 0.8	0.25	0.35	0.25	46.2 2.2			

5008416

Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)
<u>Utirik Exposed Females > 15-40</u>											
2104		7.19	5.03	1.86	0.07	0.22	0.0	39			
2119		5.74	2.35	2.87	0.17	0.34	0.0	45			
2128		6.03	3.32	2.65	0.06	0.0	0.0	33			
2129		8.60	4.90	3.18	0.17	0.26	0.90	40			
2138		8.10	5.18	2.43	0.08	0.32	0.80			11.1	
2139		9.52	3.81	3.62	0.10	1.90	1.00	43			
2147		7.10	2.20	4.47	0.14	0.28	0.0			11.8	
2159		11.48	9.87	1.38	0.23	0.0	0.0	43			
2160		10.92	6.44	3.28	0.55	0.55	0.0	48			
2164		6.67	4.14	2.07	0.0	0.40	0.70	37			
2172		8.47	3.98	3.56	0.25	0.59	0.80	43			
2189		7.50	4.58	2.48	0.15	0.22	0.80			12.5	
2195		7.09	4.25	2.27	0.21	0.21	1.40	41			
2197		9.41	5.55	3.58	0.0	0.19	0.90	36			
2217		9.10	4.46	4.19	0.09	0.27	0.90			11.4	
2220		8.62	5.43	2.59	0.09	0.52	0.0	41			
2225		8.73	5.59	2.53	0.17	0.35	0.90			10.8	
2228		12.98	7.53	5.06	0.13	0.26	0.0	40			
2230		8.58	4.72	3.09	0.17	0.51	0.90			12.5	
2246		9.97	4.59	4.09	0.0	1.30	0.0	30			
2247		11.30	4.97	5.42	0.34	0.34	2.30	36			
2251		9.28	5.01	3.71	0.09	0.37	0.90	42			
2254		8.24	4.20	3.38	0.16	0.49	0.0	39			
2256		5.68	2.90	2.27	0.17	0.28	0.60	44			
Mean		8.59	4.79	3.17	0.15	0.42	0.58	40.0		11.7	
		+1.8	1.6	1.0				4.3		0.7	

5008417

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Subject No.	Plat. ($\times 10^{-3}$)	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-2}$)	Hct. (%)	RBC ($\times 10^{-4}$)	Hgb. (g)	Serum Protein (g)
<u>Utirik Exposed Males >40</u>											
2101		7.08	4.11	2.62	0.21	0.07	0.70	43			
2103		5.65	3.45	1.98	0.11	0.0	1.10	40			
2105		7.68	4.30	2.84	0.23	0.31	0.0	45			
2110		6.40	4.16	1.98	0.13	0.13	0.0	45			
2114		7.44	4.98	2.01	0.07	0.37	0.0	44			
2125		7.91	5.38	1.82	0.32	0.31	2.30	48			
2145		6.29	2.33	3.84	0.0	0.06	0.60	47			
2148		6.27	3.32	2.57	0.19	0.19	0.0	44			
2166		5.41	2.11	3.02	0.11	0.16	0.0	41			
2169		4.29	1.72	2.19	0.13	0.20	0.40	43			
2175		4.19	2.10	1.93	0.08	0.04	0.40	42			
2181		7.19	5.25	1.44	0.07	0.43	0.0	40			
2206		7.35	4.63	2.65	0.07	0.0	0.0	47			
2211		5.16	2.32	2.22	0.21	0.36	0.50	50			
2214		11.56	7.86	2.31	0.12	1.27	0.0	40			
2240		8.17	5.31	2.29	0.16	0.33	0.80	43			
Mean		6.75	3.96	2.36	0.14	0.26	0.43	43.9			
		+1.7	1.6	0.6				2.8			

APPENDIX 13

Individual Basophil Determinations, 1965

<u>Subject No.</u>	<u>% Baso./ 4000 Cell Count</u>	<u>Subject No.</u>	<u>% Baso./ 4000 Cell Count</u>	<u>Subject No.</u>	<u>% Baso./ 4000 Cell Count</u>
1	0.75	61	0.53	113	0.53
2	0.55	63	0.48	115	0.25
3	1.13	64	0.48	116	0.35
4	0.70	65	0.58	117	0.53
5	0.45	66	0.65	118	0.38
6	0.65	67	0.38	119	0.38
7	0.50	68	0.30	120	0.30
8	0.55	69	0.73	121	0.45
10	0.68	70	0.53	122	0.30
11	0.38	71	0.25	123	0.50
12	0.50	72	0.35	124	0.45
13	0.53	73	0.40	125	0.25
14	0.40	74	0.48	126	0.33
15	0.40	75	0.30	127	0.38
16	0.60	76	0.45	128	0.40
17	0.53	77	0.50	130	0.48
18	0.50	78	0.43	131	0.48
19	0.45	79	0.43	132	0.43
20	0.45	80	0.30	134	0.45
21	0.38	81	0.48	135	0.38
23	0.45	82	0.30	137	0.38
24	0.50	83	0.38	138	0.48
27	0.45	84	0.40	139	0.40
28	0.53	85	0.75	140	0.58
29	0.50	86	0.63	143	0.45
32	0.50	87	0.43	145	0.43
33	0.43	88	0.38	801	0.53
34	0.55	89	0.48	802	0.50
35	0.45	90	0.50	803	0.60
36	0.30	91	0.53	805	0.48
37	0.75	92	0.38	806	0.53
39	0.43	93	0.30	807	0.38
40	0.28	94	0.38	808	0.40
41	0.58	96	0.58	809	0.55
42	0.38	97	0.50	810	0.48
45	0.38	98	0.63	811	0.53
47	0.50	100	0.50	812	0.25
48	0.63	101	0.43	813	0.35
49	0.45	103	0.38	814	0.58
50	0.50	104	0.53	815	0.50
51	0.48	105	0.48	816	0.38
53	0.53	106	0.40	817	0.48
54	0.58	108	0.38	818	0.38
55	0.53	109	0.48	819	0.50
58	0.55	110	0.48	820	0.50
59	0.50	111	0.55	821	0.53
60	0.38	112	0.40	822	0.45

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<u>Subject No.</u>	<u>% Baso./ 4000 Cell Count</u>	<u>Subject No.</u>	<u>% Baso./ 4000 Cell Count</u>	<u>Subject No.</u>	<u>% Baso./ 4000 Cell Count</u>
832	0.35	913	0.45	1001	0.43
826	0.28	914	0.45	1004	0.53
827	0.43	915	0.58	1006	0.58
820	0.25	916	0.50	1007	0.33
831	0.38	917	0.40	1008	0.33
832	0.30	920	0.40	1009	0.23
833	0.48	921	0.25	1010	0.43
835	0.30	922	0.40	1011	0.25
836	0.45	923	0.48	1012	0.40
840	0.50	925	0.25	1013	0.38
841	0.45	926	0.30	1014	0.48
842	0.43	928	0.45	1015	0.40
843	0.58	929	0.25	1018	0.45
844	0.28	930	0.28	1020	0.50
845	0.48	931	0.40	1021	0.43
846	0.43	932	0.48	1022	0.45
849	0.38	934	0.55	1024	0.50
850	0.60	936	0.48	1025	0.30
851	0.50	937	0.40	1026	0.48
852	0.53	938	0.45	1027	0.30
853	0.30	940	0.50	1028	0.43
855	0.23	941	0.28	1029	0.28
856	0.38	942	0.50	1030	0.23
858	0.48	943	0.55	1031	0.53
859	0.28	945	0.48	1032	0.43
860	0.30	946	0.38	1033	0.30
863	0.48	947	0.43	1034	0.43
864	0.28	948	0.43	1035	0.45
865	0.43	951	0.38	1036	0.48
866	0.43	952	0.45	1037	0.43
870	0.53	954	0.40	1038	0.35
875	0.58	955	0.35	1039	0.40
878	0.50	956	0.43	1040	0.48
879	0.38	957	0.50	1041	0.58
880	0.50	959	0.48	1042	0.38
881	0.48	960	0.50	1044	0.43
882	0.53	961	0.60	1046	0.28
884	0.28	962	0.50	1047	0.45
885	0.20	963	0.38	1049	0.25
891	0.30	964	0.43	1050	0.30
892	0.40	966	0.28	1052	0.53
895	0.23	967	0.38	1053	0.25
896	0.30	969	0.35	1054	0.50
897	0.35	970	0.48	1055	0.50
898	0.25	971	0.20	1056	0.48
899	0.53	972	0.33	1057	0.58
900	0.55	976	0.38	1058	0.40
901	0.48	978	0.25	1060	0.45
902	0.35	979	0.40	1061	0.65
903	0.48	980	0.28	1062	0.50
904	0.43	981	0.35	1063	0.50
905	0.48	982	0.48	1501	0.35
906	0.55	991	0.25	1502	0.48
907	0.40	993	0.30	1503	0.25
909	0.43	995	0.35	1504	0.40
911	0.40	996	0.40	1509	0.40
912	0.25	998	0.48		

5008420

APPENDIX 14

Chromosome Studies on Marshallese People Exposed to Fallout Radiation

Hermann Lisco, M.D.* and Robert A. Conard, M.D.

Chromosome aberrations in blood lymphocytes have been demonstrated in a number of population groups exposed to ionizing radiation. These have included patients during and after radio-therapy for ankylosing spondylitis (1,2) and malignant tumors (3,4,5), persons exposed in diagnostic procedures (6), and others exposed in the course of their work (7). Similar findings have been reported in individuals involved in radiation accidents (8,9) and in survivors of the atomic bombings in Hiroshima and Nagasaki in 1945 (10,11,12). One of the more interesting and possibly significant points in all of these studies was the observation that chromosome aberrations can persist in circulating lymphocytes in variable degree for many years following exposure. It seemed of interest to determine whether residual damage of this type could be found in Marshallese people who had been exposed to fallout radiation (13).

The accidental exposure of these people to radioactive fallout occurred on Rongelap Island in the Pacific Ocean in 1954. This was caused by an unpredicted shift in winds following detonation of a high yield nuclear device at Bikini 100 miles away. There were 64 people living on the island who received an estimated dose of 175 rads whole-body gamma-ray exposure. Eighteen other Rongelap people were away fishing on another island nearby and received an estimated dose of 70 rads. The exposure resulted in temporary blood cell depression, skin burns from beta radiation, and internal absorption of radionuclides, the most important of which were iodine and strontium. It was estimated that the thyroid glands of the adults received 150 rads and those of the children as much as 1,000 rads from absorbed radioiodine in addition to the whole-body gamma-ray exposure. The recent development of thyroid pathology is believed to be a consequence of this exposure (14).

Chromosome preparations were obtained on 51 individuals during the annual medical review of the Marshallese people in 1964. Of these, 30 had been exposed to 175 rads and 13 to 70 rads of gamma rays. Eight Marshallese who had not been exposed served as a comparison group. There were 20 males and 20 females in the exposed group and the ages ranged from an embryo in utero to 67 years at the time of exposure. Twenty-one were below and 22 above the age of 20, with males and females in about equal numbers. Of the comparison group, three were males and five females, ranging in age from 10 to 71 years. Unfortunately, it was not possible to examine a larger number of unexposed individuals matched to the exposed with respect to age and sex.

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Methods

Cultures of peripheral blood lymphocytes were made using a modification of the method of Moorhead *et al* (15). It had been determined from previous attempts of leukocyte cultures in these people that the chances of successful cultures were greatly enhanced if most of the plasma above the buffy coat in the settled blood sample was discarded and only a few ml of plasma used for culture along with the buffy coat. Cultures were harvested at 48 and 72 hours. The slides were stained with aceto-orcein and examined with phase microscopy. Intact and well-spread cells in metaphase were selected by scanning the slides with low-power magnification. Each cell thus selected was then examined with oil immersion. Examination was begun by comparing aberration rates seen in 48-hour and 72-hour cultures from 5 subjects chosen at random. No significant differences were found. Thereafter, with the exception of one case, all other examinations were made on 50 cells of each individual from the 72-hour cultures only. Thus, a total of 2150 cells was examined in the 43 exposed and 400 cells in the 8 comparison individuals.

Aberrations were scored as follows: 1) aneuploidy, including polyploid cells, 2) chromosome aberrations, and 3) chromatid aberrations. Karyotypes were made in cases where the counts were equivocal or where chromosomes of questionable morphology were seen. Only those aberrations were included in the final tabulation that were agreed upon by four observers. Additional karyotypes were made from eight euploid cells showing no gross abnormalities from each of four subjects who showed more aberrations than most others. This was done in an effort to detect in a small sample of this population minor structural abnormalities such as small deletions or inversions that could easily escape notice on microscopic examination. Finally, bone marrow preparations were made from two exposed persons and one control. Only one preparation from an exposed subject contained enough suitable cells in metaphase to warrant evaluation.

Results

As seen in Table I, aneuploid cells ranged from 5.5 per cent in the unexposed people to 10 per cent in the high exposure group, the low exposure group falling between the two with 8 per cent. In the majority of cells aneuploidy was due to loss of chromosomes. Although the percentage of aneuploid cells in the high exposure group was almost twice that seen in the comparison group, an incidence of 10 per cent may be considered at the upper limits of normal. Polyploid cells were seen with about equal frequency in all three groups.

The number of chromatid gaps and breaks and of isochromatid gaps were 2.75 per cent in the unexposed, 3.86 per cent in the high exposure and 5.31 per cent in the low exposure group. These differences were not significant.

Chromosome-type aberrations were found in 23 of the 43 exposed people and 5 of the 8 unexposed individuals (Table II). Proportionally, the largest number of persons with chromosome aberrations was found in the low exposure group with 84 per cent. The unexposed group was next with 62 per cent and this was followed by the high exposure group in which only 40 per cent of the people showed aberrations. In each of the two exposure groups one half of the aberrations consisted of two-hit events such as dicentric chromosomes, exchanges and a ring, the remainder being acentric fragments. The incidence of acentric fragments was unusually high in the unexposed group, but in contrast to the exposed people no two-hit aberrations were found among them. The difference between the exposed and unexposed groups with regard to the incidence of two-hit aberrations was found to be significant at the 1 per cent level ($p < 0.001$).

Aberrations ranged from one to four per person. Sixteen of the 23 exposed people (70%) with chromosome aberrations had more than one such aberration; two of the 5 unexposed (40%) had multiple aberrations. In most instances no more than one aberration was noted per cell.

There was no correlation between the occurrence of thyroid pathology and chromosome aberrations. Ten of the exposed people included in this group developed thyroid pathology since these examinations were made. Only four of them had shown chromosome aberrations earlier; three were children and one an adult at the time of exposure.

No aberrations were found in the karyotypes that had been made from the 32 grossly intact cells from the four subjects showing relatively high aberration rates. Likewise, no abnormalities were seen in 50 cells from the bone marrow of one of the exposed subjects.

Discussion

The results of this study demonstrate that a small but significant number of chromosome aberrations has persisted in blood lymphocytes of some of the Marshallese people for ten years following exposure to whole-body gamma radiation from fallout. This conclusion rests on the finding of the large number of two-hit aberrations (dicentrics, exchanges and a ring chromosome) in the exposed people which constituted one half of all chromosome aberrations seen. This was in striking contrast to the findings in the unexposed people in whom no two-hit aberrations were found even though the overall number of chromosome aberrations in this group was about the same and even a fraction higher than that in the exposed people taken as one group. Since two-hit aberrations were seen only in the people who had whole-body gamma ray exposure and not in the unexposed group, we interpret this finding as a residual effect of radiation.

We are unable to explain the greater incidence of chromosome aberrations in the group exposed to 70 rads as compared to the group that received 175 rads. We are also unable to account for the unusually high incidence of acentric fragments in the unexposed and their relative deficit in the exposed people. One might consider causative factors such as the low levels of internal radiation exposure from absorption of radio-nuclides such as ^{137}Cs , ^{65}Zn , ^{90}Sr , or exposure to such things as virus

infections or chemicals (14, 16, 17). However, such factors are probably not involved since they did not produce a similar effect in the irradiated group who were exposed to the same factors.

Correlation of chromosomal aberrations and severity of early radiation syndrome in the Marshallese was not apparent. For instance, the degree of early hematological depression did not seem to correlate with the occurrence or number of chromosomal aberrations on an individual basis. And, as was pointed out, the lower dose group had a higher incidence of chromosomal aberrations than did the higher dose group, though the radiation effects were more pronounced in the latter. These findings are in conformity with those of the ABCC studies (12) which failed to show correlation of chromosomal aberrations with severity of early radiation illness.

There was also no correlation of chromosomal findings in the Marshallese with age or sex. Neither did the presence or absence of thyroid pathology appear to be related. However, the development of thyroid abnormalities was related to the dose to the thyroid gland from internally absorbed radioiodines (based largely on the size of the gland) rather than the whole-body exposure.

Similar chromosome aberrations have been reported in the Japanese fishermen exposed to radiation from the same fallout (11). The incidence of aberrations, excluding aneuploid cells, was 2.1 per cent and thus identical with that seen in the Marshallese people. The majority were two-hit aberrations. The incidence of acentric fragments in a control population of Japanese studied by the same authors was 0.11 per cent; this is twenty times less than that seen among the eight unexposed Marshallese in the present study.

In a carefully controlled cytogenetic study of a sample of survivors of the atomic bombings in Hiroshima and Nagasaki, Bloom *et al.* have found an incidence of chromosome aberrations of 0.6 per cent in 94 persons 20 years after exposure (12). Thirty-eight per cent of the aberrations were of the multiple-hit type and the rest acentric fragments. Only a single minute fragment was seen in 8847 cells of the 94 controls, an unusually low incidence indeed. The low number of aberrations and the fact that only 35 per cent of the survivors showed aberrations is not surprising since 20 years have elapsed since exposure.

Although the Marshallese and Japanese exposed population and the conditions of their exposures are not strictly comparable, it is interesting that in the Marshallese people who were examined 10 years after exposure, the aberration rate as well as the number of aberration-positive individuals (53 per cent) was somewhat higher, as might have been expected.

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TABLE I
Analysis of Ploidy

<u>Subject No.</u>	<u><45</u>	<u>45</u>	<u>46</u>	<u>47</u>	<u>>47</u>	<u>Polyploid</u>	
		<u>Exposed-175 rads</u>					
2	--	1	49	-	-	-	
3	-	2	48	-	-	-	
4	-	4	46	-	-	-	
18	-	1	48	-	1	-	
19	-	1	46	1	-	2	
23	-	3	46	1	-	-	
24	1	2	47	-	-	-	
27	-	2	47	1	-	-	
32	2	1	45	-	-	2	
34	2	4	43	-	1	-	
35	-	3	45	1	-	1	
37	-	3	46	-	1	-	
39	-	2	48	-	-	-	
40	1	4	43	1	1	-	
42	1	6	41	1	-	1	
49	-	6	42	2	-	-	
54	1	1	45	3	-	-	
58	-	6	43	-	1	-	
60	1	3	45	1	-	-	
61	2	7	41	-	-	-	
63	-	3	45	2	-	-	
64	-	-	46	1	-	3	
68	-	1	48	-	-	1	
69	-	3	47	-	-	-	
73	1	1	47	1	-	-	
74	1	3	44	-	-	2	
78	4	6	40	-	-	-	
79	2	4	42	2	-	-	
80	4	5	41	-	-	-	
82	1	1	45	2	-	1	

		<u>Exposed-70 rads</u>					
1	-	1	49	-	-	-	
29	-	5	43	1	-	1	
41	-	1	49	-	-	-	
43	-	6	43	-	-	1	
45	1	2	46	1	-	-	
48	-	2	48	-	-	-	
50	-	2	47	1	-	-	
51	-	2	47	1	-	-	
53	2	3	42	3	-	-	
59	1	1	46	1	-	1	
70	1	2	45	1	-	1	
81	-	1	49	-	-	-	
84	-	1	44	5	-	-	

<u>Subject</u> <u>No.</u>	<u><45</u>	<u>45</u>	<u>46</u>	<u>47</u>	<u>>47</u>	<u>Polyploid</u>
			<u>Unexposed</u>			
105	-	-	48	1	-	1
830	-	2	48	-	-	-
859	-	2	48	-	-	-
931	-	1	48	1	-	-
938	-	2	48	-	-	-
982	1	2	46	-	-	1
1005	3	2	45	-	-	-
1043	-	4	46	-	-	-

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Subject No.	Plat. (X10 ⁻³)	WBC (X10 ⁻³)	Neut. (X10 ⁻³)	Lymph. (X10 ⁻³)	Mono. (X10 ⁻³)	Eosin. (X10 ⁻³)	Baso. (X10 ⁻²)	Hct. (%)	RBC (X10 ⁻⁴)	Hgb. (g)	Serum Protein (g)
<u>Utirik Exposed Females >40.</u>											
2109		19.12	10.70	7.27	0.38	0.76	0.0			12.5	
2140		6.44	3.48	2.45	0.26	0.13	0.60	43			
2146		5.46	2.02	2.95	0.0	0.49	0.0	42			
2158		5.45	2.62	2.62	0.11	0.05	0.50	36			
2161		8.70	6.35	1.83	0.44	0.09	0.0	39			
2162		11.47	8.37	2.87	0.23	0.0	0.0	36			
2186		5.74	2.35	2.30	0.29	0.69	0.60	39			
2191		7.83	5.35	2.04	0.16	0.23	0.80	35			
2193		8.49	3.99	3.91	0.17	0.34	0.80	38			
2194		8.88	4.88	3.20	0.18	0.53	0.90			11.8	
2196		7.90	3.16	3.71	0.24	0.63	1.60	41			
2198		4.93	2.07	2.61	0.05	0.15	0.50	39			
2200		6.23	4.17	1.81	0.06	0.19	0.0	45			
2202		10.43	6.57	3.23	0.21	0.42	0.0	40			
2212		7.53	3.24	3.24	0.08	0.83	1.50	41			
2215		8.78	5.88	2.46	0.18	0.26	0.0	41			
2216		8.85	5.22	3.19	0.18	0.09	1.80	39			
2221		9.54	6.85	1.05	0.57	1.14	1.90	39			
2223		4.13	2.35	1.49	0.04	0.21	0.0	37			
2224		9.68	4.65	4.07	0.29	0.58	1.00	39			
Mean		8.27 +3.1	4.71 2.2	2.92 1.3	0.21	0.39	0.63	39.4 2.5		12.2	

5008428

TABLE II
Analysis of Structural Aberrations

Subject No.	Age ⁺	Sex	Frag-ments	Dicen-trics	Rings	Exch-anges	No. of		Chromatid Breaks	Isogaps	Remarks*
							Chromosome Aberrations	Cells with Aberrations			
							<u>Exposed, 175 rad</u>				
2	12	M	2				2	2	1	1	Thyroid pathology
3	11	M							4		Thyroid pathology
4	48	M		2			2	2	1		
18	31	F							1	1	
19	15	M									Thyroid pathology
23	14	M									
24	23	F	2	2			4	3	1		
27	36	M							3	2	2 breaks scored for one quadriradial
32	13	M	1				1	1			
34	55	F							3		
35	23	M		1			1	1	1		
37	30	M									
39	25	F							3		
40	39	M									Thyroid pathology
42	13	F				1	1	1	4		Thyroid pathology
49	25	F							1	2	
54	11	M							4		Thyroid pathology
58	69	F		1		1	2	2	1		
60	66	F				1	1	1	1		
61	18	F	1			1	2	2		1	Thyroid pathology

⁺Age in years at time of examination, i.e., 10 years after exposure.

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<u>Subject No.</u>	<u>Age</u>	<u>Sex</u>	<u>Frag-ments</u>	<u>Dicen-trics</u>	<u>Rings</u>	<u>Exch-anges</u>	<u>No. of Chromosome Aberrations</u>	<u>No. of Cells with Aberrations</u>	<u>Chromatid Breaks</u>	<u>Isogaps</u>	<u>Remarks*</u>
<u>Exposed, 175 rad (continued)</u>											
63	46	F							1		
64	40	F							1	3	Thyroid pathology
68	55	M							2	1	
69	14	F							1		Thyroid pathology
73	28	M	2				2	2	2		
74	26	F	1				1	1	5	3	
78	47	F							1		
79	49	M							1	1	
80	56	M									
82	60	M	2				3	2	1		
<u>Exposed, 70 rad</u>											
1	64	F							3		
29	75	M	2			1	3	2	1		
41	54	M	1		1		2	2	1		
43	77	F	1			1	2	2	2		
45	42	F	2				2	2	2		
48	16	F		1			1	1		1	
50	44	M							2	1	
51	35	F	1			1	2	2	1	1	
53	18	F	1			2	3	1	11		
59	44	F	1	1			2	2	4	1	Thyroid pathology

5008430

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<u>Subject No.</u>	<u>Age</u>	<u>Sex</u>	<u>Frag-ments</u>	<u>Dicen-trics</u>	<u>Rings</u>	<u>Exch-anges</u>	<u>No. of Chromosome Aberrations</u>	<u>No. of Cells with Aberrations</u>	<u>Chromatid Breaks</u>	<u>Isogaps</u>	<u>Remarks*</u>
<u>Exposed, 70 rad (continued)</u>											
70	25	F				1	1	1			
81	18	F	1				1	1	2		
84	9	M				2	2	2	2		Exposed <u>in utero</u>
<u>Unexposed</u>											
105	5	F							1		Child of exposed
830	25	M							1		
859	71	F	1				1	1	1	1	
931	10	M	1				1	1			
938	25	F	1				1	1		1	
982	43	F									
1005	31	M	3				3	3	1		
1043	29	F	3				3	2	2	3	

* Except for Subject Nos. 3 and 64, all thyroid pathology was adenomatous goiter. Case 3 is a hypothyroid child. Case 64 had a mixed papillary and follicular carcinoma of the thyroid.

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APPENDIX 15

Total Blood and Red Cell Volume Data

(WT=gross weight; TBW=total body water; FAT=fat as % gross weight;
LBM=lean body mass; RCV=red cell volume; BV=blood volume)

<u>Subject No.</u>	<u>WT (kg)</u>	<u>TBW (l)</u>	<u>TBW (%)</u>	<u>FAT (%)</u>	<u>LBM (kg)</u>	<u>RCV (l)</u>	<u>BV (l)</u>	<u>RCV/LBM (ml/kg)</u>	<u>BV/LBM (ml/kg)</u>
<u>A - Marshallese</u>									
822	54.54	38.1	68.8	4.4	52.1	1.402	3.260	26.9	62.6
832	46.36	25.0	53.0	26.4	34.1	0.849	2.358	24.9	69.2
836	56.36	35.3	61.7	14.3	48.3	1.428	3.320	29.6	68.7
838	66.13	41.7	62.2	13.6	57.1	2.108	4.053	36.9	71.0
841	66.81	31.9	47.0	34.7	43.6	1.150	3.196	26.4	73.3
873	61.36	43.2	69.4	3.6	59.1	1.670	3.631	28.3	61.4
881	68.63	32.8	47.1	34.6	44.7	1.996	4.247	44.7	95.0
882	54.77	39.9	71.8	0.3	54.6	1.131	3.426	20.7	62.7
885	61.81	41.0	65.3	9.3	56.1	1.760	3.825	31.4	68.2
895	55.90	29.0	51.5	28.5	40.0	1.070	2.488	26.8	62.2
916	63.63	32.6	50.4	30.0	44.5	1.091	3.031	24.5	68.1
928	46.30	26.2	55.7	22.6	35.8	1.274	2.963	35.6	82.8
938	40.00	22.0	54.1	24.9	30.1	0.886	2.331	29.4	77.4
942	57.72	27.6	47.1	34.5	37.8	0.860	2.150	22.8	56.9
959	60.00	32.2	52.8	26.7	44.0	1.151	2.877	26.2	65.4
960	38.63	24.8	63.1	12.4	33.9	0.774	2.150	22.8	63.4
1007	71.36	41.2	56.9	21.0	56.4	1.620	4.155	28.7	73.7
1043	41.81	26.4	62.3	13.5	36.2	1.066	2.664	29.4	73.6
1501	66.81	43.3	64.0	11.2	59.3	1.843	3.840	31.1	64.8
2000	63.18	39.8	61.9	14.0	54.4	1.310	2.675	24.1	49.2
MEAN		33.5				1.303	3.102	28.3	68.2

B - Caucasians Residing in Pacific Ocean Area

G.B.	104.55	52.7	49.65	31.0	72.1	2.425	5.390	33.6	74.8
D.B.	71.36	45.9	63.34	12.0	62.8	1.747	3.970	27.8	63.2
J.C.	75.91	45.2	58.56	18.6	61.7	1.809	4.308	29.3	69.8
P.C.	68.18	36.0	52.10	27.6	49.3	1.588	3.379	32.2	68.5
A.C.	90.91	55.2	59.88	16.8	75.61	2.097	5.116	27.7	67.7
W.D.	84.09	51.9	60.73	15.6	70.9	2.412	5.610	34.0	79.1
R.J.	86.36	50.2	57.18	20.5	68.6	2.457	5.341	35.8	77.9
D.J.	70.45	44.3	61.83	14.1	60.5	1.794	4.078	29.7	67.4
C.P.	84.09	43.8	51.23	28.8	59.8	2.657	5.776	44.4	96.6
J.S.	84.09	50.8	59.47	17.4	69.5	2.428	5.518	34.9	79.4
C.T.	80.91	44.6	54.25	24.6	61.0	2.540	5.405	41.6	88.6
A.T.	61.36	37.5	60.19	16.4	51.3	1.199	4.064	23.3	79.2
I.W.	77.27	50.1	63.74	11.4	68.4	2.575	5.479	37.6	80.1
A.B.	65.91	33.9	50.69	29.6	46.4	1.986	4.225	42.8	91.1
T.B.	67.27	33.8	49.44	31.3	46.2	1.424	3.561	30.8	77.1
B.C.	60.00	30.7	50.40	30.0	42.0	1.714	4.181	40.8	99.5
C.M.	60.91	32.1	51.98	27.8	44.0	1.672	4.181	38.5	95.0
D.P.	52.27	27.8	52.42	27.2	38.1	1.149	3.023	30.2	79.3
P.R.	72.73	42.8	57.98	19.5	58.5	2.006	4.458	34.3	76.2
MEAN		42.6				1.983	4.582	34.2	79.5

APPENDIX 16

Studies on Australia Antigen in Micronesian Populations

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Australia antigen is a serum protein first detected in the serum of Australian aborigines, using an antiserum developed in a hemophilia patient who had received a large number of transfusions.⁽¹⁾ The distribution of this trait has now been studied in more than 50 human populations. (See Table 1.) It was very rare in normal United States populations (none in 1,500 sera) but fairly common (3-6%) in sera collected from Marshall Islanders, Australian aborigines, Vietnamese, Filipinos, Polynesians, and other Southeast Asian populations. It is less common in Mediterranean populations and is extremely rare in Northern European populations and in most autochthonous New World populations. In addition to the relevance of this trait to studies on cancer (see below), it is of interest in anthropological studies of human populations.

Australia antigen migrates as an alpha protein on immuno-electrophoresis. The precipitin band which forms between the hemophilia antiserum and the serum containing Australia antigen stains faintly with Sudan black, indicating that the protein contains some lipid. Australia antigen has a specific gravity between 1.063 and 1.30, that is, it is more dense than low density lipoprotein but less so than most other serum proteins. It appears in the first peak in Sephadex G200 column chromatography, indicating that it is a high molecular weight serum protein. It is found in the fifth and sixth peaks on DEAE column chromatography. The protein can be isolated by a combination of Sephadex chromatography and high speed ultracentrifugation. The antibody in the hemophilia serum which reacts with the Australia antigen travels in the I_g (7S) gamma globulin fraction.⁽²⁾

Antisera against the protein have been produced by the immunization of rabbits with serum containing Australia antigen and the subsequent absorption of the antiserum with the sera of normal persons.⁽³⁾

In order to initiate studies on the inheritance of Australia antigen, it was necessary to determine if the trait was persistent over the course of weeks, months, or years. Sera collected from the population of Rongelap Atoll over the course of up to eight years were available. Samples from a total of 300 Micronesians from Rongelap were collected from the period 1958 to 1965. Of these, serial samples were available from 250 individuals. Of the latter, 237 were consistently negative, eleven were consistently positive and two were inconsistent. In each case, the inconsistency was apparent in only one of multiple sera. (See Figure 1.)

Family studies were undertaken on a total of 36 families, including 5 families from Rongelap. The results were consistent with the hypothesis that individuals homozygous for a gene termed Au¹ (genotype Au¹/Au¹) have detectable Australia antigen in their blood (phenotype Au⁽¹⁾). Individuals

homozygous for the alternate allele (genotype Au/Au) or heterozygotes (Au¹/Au) do not have detectable Australia antigen (phenotype Au (o)).(4)

Although Au (1) has not been found in normal United States populations, it is relatively common in some forms of leukemia, i.e., 18% of acute myelogenous leukemia, 17% of chronic lymphocytic leukemia. It has not as yet been found in chronic myelogenous leukemia. A consideration of the United States data alone, therefore, would suggest that Australia antigen is a leukemia-specific antigen and may be associated with an increased risk of developing leukemia. It has also been found in high frequency (approximately 30%) in patients with Mongolism (Down's syndrome), a disease characterized by a high risk of developing leukemia.(5) During our studies in Cebu, we found that Australia antigen is significantly more common in patients with lepromatous leprosy than those with tuberculoid leprosy or non-leprosy individuals from the same area.(6)

Consideration of all the patient data suggests that a common factor in the various diseases in which the trait is found is a deficiency of the immunological system associated in some as yet unexplained manner with the presence of Australia antigen. Since the Rongelap people will be medically examined for many years, it will be of considerable interest to see if there is a differential susceptibility to disease, particularly leukemia, in respect to Australia antigen.

In several populations, including Marshall Islanders, a significant decrease in Australia antigen frequency with age has been detected. This may be due to selection against individuals with this trait, presumably compensated for by some, at present unknown, selective advantage in respect to one of the genotypes.

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Table 1

Australia Antigen in Non-Hospitalized Populations
(from Blumberg et al.⁽⁴⁾)

<u>Population</u>	<u>Location</u>	<u>No. Tested</u>	<u>No. Pos.</u>	<u>% Pos.</u>
<u>AMERICAS</u>				
Eskimos	Alaska, U.S.A.	394	1	0.3
Indians, Athabascan	Alaska, U.S.A.	204	0	0
Indians, Cashinahua	Peru	89	18	20.2
Indians, Haida	Canada	338	0	0
Indians, Mexico	Mexico	252	1	0.4
Indians, Quechua	Peru	102	0	0
Indians, Sioux	S. Dakota, U.S.A.	130	0	0
Negros	Ga., Md., U.S.A.	607	0	0
Whites	Ga., Md., U.S.A.	896	0	0
<u>AFRICA</u>				
Pare	Tanzania	120	1	0.8
Tristan da Cunha	Tristan da Cunha	42	0	0
Ghanese	Ghana	95	9	9.5
<u>ASIA</u>				
Japanese	Osaka, Japan	301	1	0.3
Chinese	U.S.A., Taiwan	100	0	0
Taiwanese	Taiwan	23	3	13.0
Israelis	Israel	340	4	1.2
Jordanese	Jordan	40	0	0
Filipinos	Cebu, P.I.	764	37	4.8
Filipinos	Manila, P.I.	197	9	4.6
Vietnamese	Vietnam	128	8	6.3
<u>EUROPE</u>				
Finns	Finland	924	1	0.1
Lapps & Finnlapps	Finland	127	0	0
Italians	Ferara	101	0	0
Greeks	Greece	857	15	1.8
Portugese	Hawaii	44	1	2.2
<u>OCEANIA</u>				
Aborigines	Australia	233	12	5.1
Maoris	New Zealand	4	1	0
Melanesians	New Guinea	166	6	3.6
Micronesians	Marshall Is.	474	34	7.2
Polynesians	Bora Bora	119	3	2.5
Polynesians	Hawaii	43	1	2.3
Total		8,254	166	-

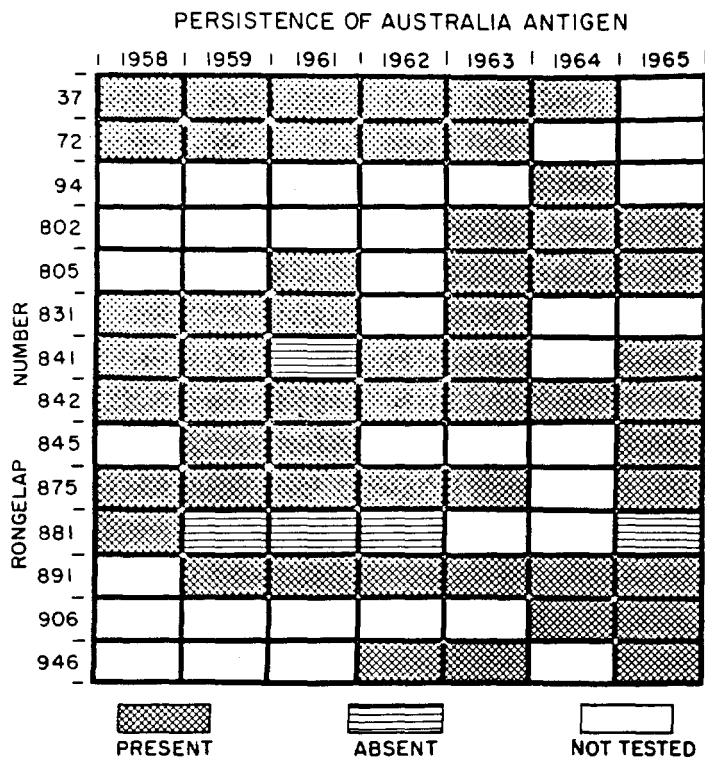


Figure 1

Studies on the persistence of Australia antigen in blood samples collected serially from apparently normal individuals on Rongelap. The subject numbers are shown on the left. The reactions of sera collected in the years indicated at the top are given in the body of the figure.

(from Blumberg et al.⁽⁴⁾)

APPENDIX 17

Gamma Spectrographic Data, Rongelap People, 1965

Subject No.	Age	Wt.(Kg)	Potassium g/Kg Body Wt.	¹³⁷ Cs nCi/Kg Body Wt.	⁶⁰ Co pCi/Kg Body Wt.
<u>Rongelap Exposed Males (Age 11-15)</u>					
83*	11	33.6	2.11	9.0	18.8
54	12	40.4	2.03	12.6	28.5
2	13	32.4	2.26	18.4	41.0
6	13	30.7	2.34	17.8	17.0
19	14	42.3	2.21	12.4	27.2
Mean			2.19	14.0	26.5
<u>Rongelap Unexposed Males (Age 11-15)</u>					
921	11	29.3	1.78	22.5	32.3
813	11	27.7	1.84	27.8	31.6
912	12	28.9	2.32	20.0	39.6
1052	12	37.1	2.19	18.9	22.7
814	13	35.1	2.33	23.3	31.9
1036	13	34.4	2.08	13.9	29.3
1033	14	57.3	1.80	13.1	27.7
818	14	41.4	2.14	23.5	32.5
913	14	39.1	2.06	15.9	31.2
815	15	47.7	2.32	17.7	19.1
863	15	50.0	2.13	22.6	48.1
Mean			2.09 +0.20**	19.9 +4.3	31.5 +7.3
<u>Rongelap Exposed Females (Age 11-15)</u>					
86*	11	24.3	2.45	15.2	6.5
65	12	26.6	2.28	11.9	42.5
33	12	45.5	1.82	21.0	29.8
8	13	40.3	1.67	12.6	36.6
42	14	36.3	1.69	16.5	42.9
17	14	49.7	1.61	20.4	13.8
Mean			1.92	16.3	28.7
<u>Rongelap Unexposed Females (Age 11-15)</u>					
811	11	29.4	2.13	19.2	37.1
805	11	32.3	1.64	14.5	38.5
911	12	41.6	1.93	19.9	23.5
955	12	47.3	1.52	7.4	13.1
996	12	34.8	2.28	20.9	29.9
960	15	49.5	1.82	17.0	27.8
816	15	50.0	1.38	12.2	32.2
Mean			1.81	15.9	28.9

* Exposed in utero.

$$** \text{ Standard deviation } = \sqrt{\frac{\sum (x^2)}{N} - \left(\frac{\sum x}{N}\right)^2}$$

Subject No.	Age	Wt.(Kg)	Potassium g/Kg Body Wt.	¹³⁷ Cs nCi/Kg Body Wt.	⁶⁰ Co pCi/Kg Body Wt.
<u>Rongelap Exposed Males (Age >15)</u>					
20	18	54.0	2.17	14.3	36.0
73	29	71.2	2.17	26.1	27.6
10	35	63.5	2.04	17.9	40.4
27	37	64.9	1.88	20.4	48.0
40	40	55.3	2.47	18.9	26.1
50	45	85.3	1.61	8.5	36.4
7	47	56.7	1.97	16.5	69.3
4	49	66.9	1.85	24.7	47.7
79	50	65.8	1.55	12.9	31.0
16	50	52.2	1.94	16.5	59.5
41	55	54.4	2.23	22.3	43.9
68	56	58.5	1.76	19.6	26.6
80	57	60.6	2.09	8.1	45.0
82	61	57.2	1.74	19.3	36.8
11	61	51.7	2.10	10.2	36.5
Mean			1.97 <u>+0.24</u>	17.1 <u>+5.3</u>	40.7 <u>+11.5</u>
<u>Rongelap Unexposed Males (Age >15)</u>					
820	16	56.9	2.55	15.7	30.3
822	18	56.5	2.42	12.3	43.7
939	19	69.4	2.08	15.6	33.5
832	21	66.2	2.20	16.2	37.6
967	22	63.5	2.22	15.1	19.8
827	25	60.1	2.14	18.1	16.0
1501	28	69.2	1.78	11.9	28.0
882	32	54.0	2.04	9.3	64.6
836	32	63.5	1.93	10.5	11.4
833	32	64.4	1.68	9.9	32.1
881	33	76.2	1.74	10.2	35.0
845	35	69.9	2.05	14.6	29.2
840	35	68.0	2.03	21.4	42.7
864	39	74.2	1.88	22.1	47.5
917	46	83.7	1.44	12.9	44.7
875	48	59.9	2.07	22.4	61.6
850	54	60.8	1.93	23.0	56.6
1007	54	74.4	1.64	11.7	30.8
948	57	75.8	1.47	10.5	38.4
947	56	49.0	2.02	18.1	57.0
855	60	65.8	1.58	9.2	36.8
853	60	68.9	1.46	11.8	34.7
1041	60	83.0	1.43	16.5	32.3
910	62	53.1	2.11	27.4	59.8
878	65	83.0	1.39	5.7	19.0
935	67	58.9	1.79	15.2	50.0
856	66	55.8	1.85	14.6	25.7
915	68	55.3	1.94	13.4	34.7
860	75	56.7	1.22	8.1	20.8
Mean			1.88 <u>+0.32</u>	14.7 <u>+4.9</u>	37.6 <u>+13.8</u>

Subject No.	Age	Wt(Kg)	Potassium g/Kg Body Wt.	¹³⁷ Cs nCi/Kg Body Wt.	⁶⁰ Co pCi/Kg Body Wt.
<u>Rongelap Exposed Females (Age >15)</u>					
48	17	52.4	1.72	15.2	33.2
15	18	52.2	2.68	7.8	161.8
53	19	48.5	1.91	20.0	17.1
61	19	75.3	1.13	4.0	21.5
49	26	68.5	1.49	6.7	31.4
18	32	48.9	1.40	8.1	50.6
14	36	62.1	1.44	12.5	30.8
51	36	43.3	1.90	11.2	46.9
66	40	65.8	1.49	8.6	37.2
64	41	61.7	1.41	12.7	24.6
59	45	38.7	1.70	18.9	51.5
63	47	49.4	1.72	18.3	43.9
78	48	68.0	1.12	11.3	32.8
34	56	62.1	1.46	10.8	38.8
1	65	73.0	0.96	5.4	25.8
60	67	59.0	1.24	7.7	25.0
13	69	33.1	1.58	19.9	31.7
58	70	54.9	1.05	10.5	15.5
Mean			1.52 ±0.39	11.6 ±5.0	40.0 ±10.6 32.8* ±10.6

<u>Rongelap Unexposed Females (Age >15)</u>					
959	16	63.5	1.49	9.2	34.4
821	17	59.6	1.51	7.2	16.0
998	18	56.2	1.62	9.3	24.4
896	24	46.3	1.77	12.8	45.4
1502	26	52.2	1.75	13.5	29.6
832	27	49.9	1.28	8.7	40.8
1050	30	59.4	1.48	15.7	61.6
932	30	50.3	1.55	10.9	24.8
914	30	45.8	1.78	14.7	25.7
934	30	63.7	1.44	15.2	34.2
835	31	51.0	1.35	12.6	37.7
1001	31	58.5	1.33	22.0	22.0
841	32	69.9	0.93	5.8	25.3
865	32	49.4	1.69	11.7	35.3
834	36	50.3	1.56	8.3	33.0
945	40	39.0	1.94	12.9	31.0
916	41	61.7	1.47	7.1	28.5
844	46	50.3	1.44	13.8	49.1
1042	47	58.5	1.36	12.1	24.4
942	50	60.1	1.43	17.6	44.0

*Mean with subject No. 15 excluded.

Subject No.	Age	Wt(Kg)	Potassium g/Kg Body Wt.	¹³⁷ Cs nCi/Kg Body Wt.	⁶⁰ Co pCi/Kg Body Wt.
<u>Rongelap Unexposed Females (Age >15)-contd.</u>					
928	52	56.9	1.28	8.6	22.9
851	56	77.6	1.15	7.0	17.7
956	56	60.3	1.39	11.1	21.4
957	57	72.6	1.06	7.2	7.2
852	61	46.3	1.41	8.1	39.5
858	70	45.8	1.59	7.3	38.9
936	74	54.4	1.04	7.0	27.7
Mean			1.45 <u>+0.22</u>	11.0 <u>+3.8</u>	31.2 <u>+11.2</u>
<u>Rongelap Unexposed Males (Age <11)</u>					
1038	3	15.1	1.84	19.5	20.1
110	5	17.4	3.29	26.5	9.8
1018	5	15.3	1.73	11.1	22.1
111	6	14.2	2.35	18.9	43.1
1024	6	19.1	1.92	17.8	50.5
1053	6	17.5	2.43	35.7	33.8
98	7	18.2	2.22	14.1	22.8
905	7	18.0	3.41	20.2	9.1
1004	7	19.0	2.60	26.2	35.4
904	7	22.0	2.18	16.5	35.2
1503	7	15.7	2.17	16.0	56.8
93	8	23.0	2.74	7.8	10.3
89	9	22.6	2.75	16.9	0.0
801	9	19.9	2.39	19.6	36.8
803	9	23.0	2.56	15.7	37.7
809	9	23.3	2.67	33.2	18.4
90	9	27.8	2.56	34.1	27.9
802	9	23.7	2.56	13.2	42.7
88	10	26.8	2.02	23.8	17.4
91	10	27.4	2.60	18.5	39.2
807	10	20.7	1.90	22.7	75.9
Mean			2.42 <u>+0.42</u>	20.4 <u>+7.2</u>	30.7 <u>+17.6</u>
<u>Rongelap Unexposed Females (Age <11)</u>					
140	<1	5.0	4.71	9.6	0.0
120	5	17.0	2.25	17.2	38.1
1026	5	15.5	2.75	18.4	25.4
112	6	15.5	2.42	15.1	43.1
1020	6	14.3	2.71	12.4	41.0
139	<1	5.5	2.72	1.0	0.0
106	6	17.5	3.01	8.2	22.7
1025	6	14.5	2.33	14.8	25.1
1022	5	16.1	2.39	13.2	35.4
906	7	17.7	2.66	14.7	28.1

Subject No.	Age	Wt.(Kg)	Potassium g/Kg Body Wt.	¹³⁷ Cs nCi/Kg Body Wt.	⁶⁰ Co pCi/Kg Body Wt.
<u>Rongelap Unexposed Females (Age <11)-contd.</u>					
1034	7	20.1	2.23	22.0	13.2
902	7	20.3	2.08	14.4	25.4
1012	7	21.4	2.15	17.4	22.2
103	7	18.0	2.03	28.6	18.1
901	8	22.3	2.35	19.3	22.5
900	8	23.3	2.36	14.7	32.0
92	9	23.3	1.29	18.9	75.4
94	9	20.5	1.39	11.8	10.4
810	10	28.5	2.09	11.5	30.4
808	10	30.2	2.26	17.6	27.5
866	10	24.5	2.65	19.0	30.4
Mean			2.42 ±0.44	15.2 ±5.4	27.0 ±15.6
<u>Rongelap People Residing At Ebeye</u>					
85*	11M	30.2	2.01	0.89	39.0
2	12M	36.2	1.38	0.86	32.2
81	19F	50.8	1.62	1.56	10.4
830	26M	74.4	1.80	0.22	8.8
70	28F	51.3	1.39	0.62	25.6
71	39F	57.4	2.17	1.85	175.0
842	41M	72.6	1.39	0.23	7.3
Mean			1.68	0.89	42.6 20.6**
<u>U. S. Medical Team</u>					
2-34	32	100.0	1.43	0.30	0
2-90	35	78.2	1.66	0.12	0
2-91	35	81.8	1.62	0.23	0
2-35	40	73.6	1.39	0.13	0
2-33	45	83.3	1.44	0.17	0
2-89	46	69.6	1.59	0.08	0
3-93	48	75.0	1.76	0.20	0
4-32	51	77.3	1.41	0.12	0
4-18	59	81.8	1.24	0.12	0
Mean			1.50 ±0.18	0.16 ±0.11	
<u>Trust Territory Medical Team</u>					
4-52	29	72.7	1.78	0.49	0
3-17	37	80.9	1.43	0.42	0
Mean			1.61	0.45	

*Exposed in utero.

**Mean excluding subject No. 71.

APPENDIX 18

Cesium-137 and Strontium-90 Retention Following an Acute
Ingestion of Rongelap Food

by

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SUMMARY

The cesium-137 and strontium-90 body burdens of people living on Rongelap Island are high compared to most other populations of the world. The reason for this is that the natives consume foods which are contaminated with long-lived fission product radioactivity resulting from an accidental fallout incursion in 1954. Their Cs-137 body burdens are comparable to those of people living in other limited areas such as Lapland and northern Alaska where unique ecological conditions are conducive to high concentrations of cesium-137 in indigenous foods. The metabolism of cesium-137 and strontium-90 has been studied in the Lapland and Alaskan groups but not in the Rongelap natives.

Since facilities for a metabolic balance study were not available on Rongelap Island, one of us (R.A.C.) brought several native food items (pandanus fruit and coconut meat and milk) back to Brookhaven and consumed them under controlled conditions. Urinary and fecal specimens were collected and whole body counting measurements were made over a period of 180 days. The intake of strontium-90 over a seven day period was twenty times higher than normal and that of cesium-137, sixty times higher than normal.

Fifty percent of the ingested cesium-137 in the Rongelap food was excreted in urine after 85 days while fourteen percent was eliminated via the feces at the same time. In contrast, most of the strontium-90 was unabsorbed. Fifty percent was excreted in feces at 10 days while only two and one-half percent was excreted in urine. The retention of cesium-137 as determined by both whole body counting and excretion measurements showed a biological half-life of 74 days. Strontium-90 retention as a function of time was best described as a series of exponentials, and approached a value of 25 percent after 140 days.

These findings fall within the range of results of many other studies conducted under a wide variety of natural, accidental, and experimental conditions. Table I and Figures 1-4 summarize the above findings.

TABLE I

Sr⁹⁰, Cs¹³⁷, AND Ca IN EXCRETA

Sampling period	days	series	Urine total vol. ml.	Stool total wt. g	pc Sr ⁹⁰		g Ca		pc Cs ¹³⁷		pc Sr ⁹⁰ /day		g Ca/day		pc Cs ¹³⁷ /day		pc Sr ⁹⁰ /g Ca	
					Urine per l	Stool per kg	Urine per l	Stool per kg	Urine per l	Stool per kg	Urine	Stool	Urine	Stool	Urine	Stool	Urine	Stool
5/26-28/63	3	Pre	2520	645	3.1	48	0.25	0.84	58	50	2.6	10.2	0.21	0.18	49	11	12	57
5/29-7/1/63	3	"	3430	573	2.6	110	0.19	1.88	43	73	3.0	21.0	0.21	0.35	49	14	14	58
7/2-4/63	3	Rong. food	4240	1081	10.8	991	0.11	1.53	431	707	15.3	357	0.15	0.55	609	255	98	648
7/5-8/63	4	"	4420	809	8.6	1406	0.06	1.97	617	1835	9.5	284	0.06	0.39	682	371	143	714
7/9-11/63	3	Post	3610	482	10.8	264	0.13	1.28	439	1102	13.0	42.4	0.15	0.20	528	177	83	206
7/12-14/63	3	"	3900	544	*	153	*	2.49	*	709	*	27.8	*	0.45	*	128	*	61
7/19/63	1	"	1150	327	4.7	*	0.14	0.57	508	306	5.4	*	0.16	0.18	584	100	34	*
7/26/63	1	"	609	211	5.9	166	0.17	1.48	710	514	4.1	35.0	0.12	0.31	490	108	35	112
8/2/63	1	"	905	186	4.7	138	0.17	1.31	513	581	4.2	25.7	0.15	0.24	464	108	28	105
8/9/63	1	"	1900	156	1.7	121	0.09	2.12	218	594	3.3	18.9	0.17	0.33	414	93	19	57
8/16/63	1	"	1350	297	3.0	104	0.15	2.22	341	*	4.0	31.9	0.20	0.66	460	*	20	47
8/23/63	1	"	1280	270	2.4	131	0.11	2.66	273	350	3.1	35.4	0.14	0.72	349	95	22	49
8/30/63	1	"	770	221	3.6	75	0.16	1.15	297	357	2.8	16.5	0.12	0.25	229	79	23	65
9/6/63	1	"	660	441	3.9	46	0.19	0.79	*	135	2.6	20.2	0.12	0.35	*	60	20	58
9/13/63	1	"	-	176	-	97	-	2.22	-	557	-	17.0	-	0.39	-	98	-	44
9/20/63	1	"	678	-	3.7	-	0.29	-	295	-	2.5	-	0.20	-	200	-	13	-
9/21/63	1	"	1040	-	2.9	-	0.19	-	282	-	3.0	-	0.20	-	293	-	15	-
10/4/63	1	"	920	160	3.0	105	0.26	2.30	236	386	2.7	16.7	0.24	0.37	217	62	11	46
10/21/63	1	"	1180	230	2.6	109	0.19	2.30	219	274	3.0	25.1	0.22	0.53	258	63	13	47
11/5/63	1	"	680	320	4.7	*	0.24	1.22	217	299	3.2	*	0.16	0.39	148	96	20	*
12/6/63	1	"	1100	60	2.5	172	0.21	3.33	128	417	2.7	10.3	0.23	0.20	141	25	12	52
1/7/64	1	"	780	94	3.2	149	0.18	2.76	196	356	2.5	14.0	0.14	0.26	152	34	18	54

*Sample lost.

-No sample received.

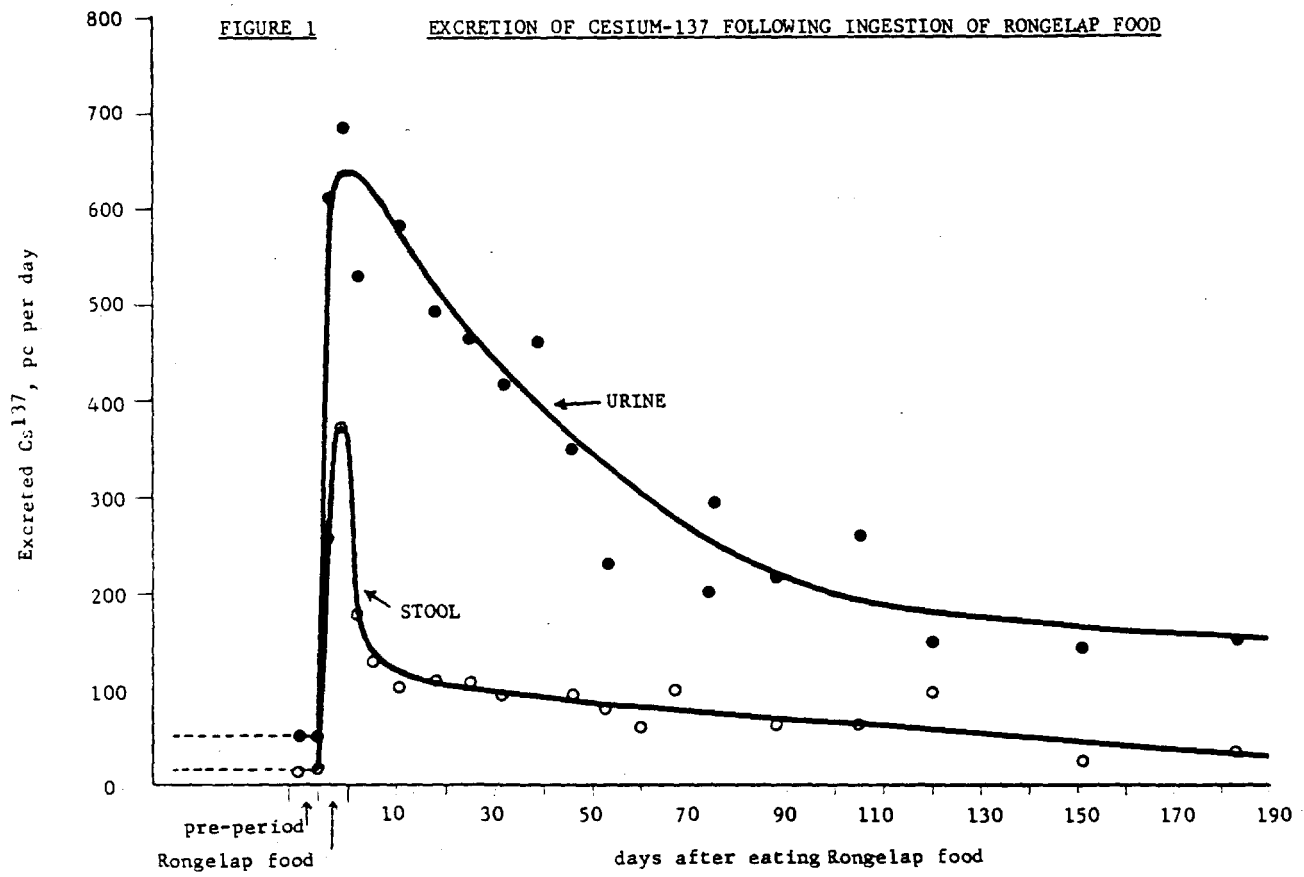


FIGURE 2 EXCRETION OF STRONTIUM-90 FOLLOWING INGESTION OF RONGELAP FOOD

