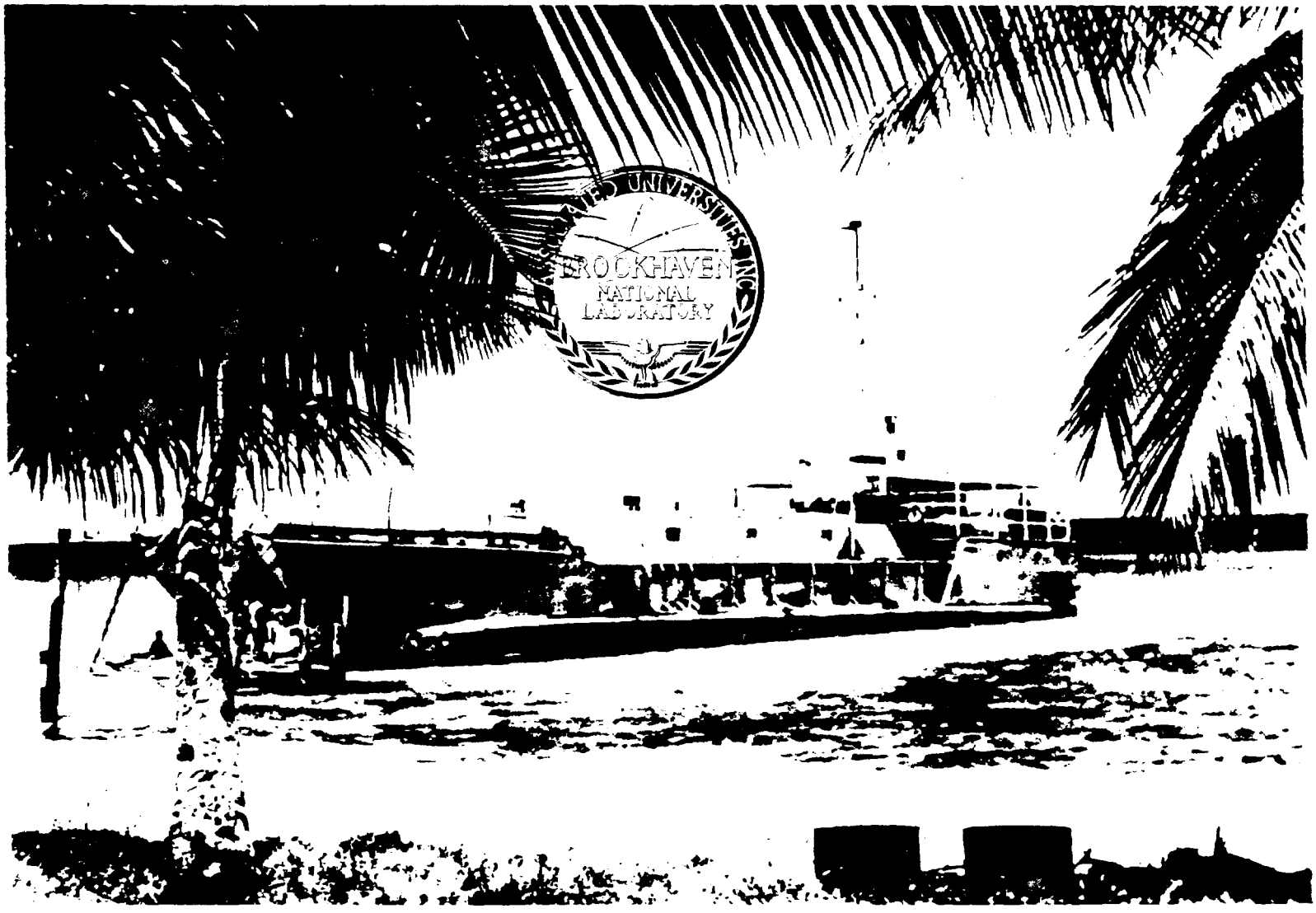
BNL 50424

D. Stannard
With best regards.
Bob Conard

A TWENTY-YEAR REVIEW OF MEDICAL FINDINGS IN A MARSHALLESE POPULATION ACCIDENTALLY EXPOSED TO RADIOACTIVE FALLOUT

ROBERT A. CONARD, M.D., ET AL.



BROOKHAVEN NATIONAL LABORATORY
ASSOCIATED UNIVERSITIES, INC.
UNDER CONTRACT NO. AT(30-1)-16 WITH THE
UNITED STATES ENERGY RESEARCH AND DEVELOPMENT ADMINISTRATION

BNL 50424
(Biology and Medicine - TID-4500)

A TWENTY-YEAR REVIEW OF MEDICAL FINDINGS IN A MARSHALLESE POPULATION ACCIDENTALLY EXPOSED TO RADIOACTIVE FALLOUT

PROFESSIONAL TEAM (1970-1974)

Robert A. Conard, M.D.[1] ('70, '71, Mar, Sep '72, Mar '73, Mar '74)
Knud D. Knudsen, M.D.[1] ('71, Mar, Sep '72, Mar, Sep '73, Mar, Sep '74)
Brown M. Dobyns, M.D.[2] ('70, Mar '72, Mar '73)
Leo M. Meyer, M.D.[3] (Mar '72, Mar '73)
Wataru W. Sutow, M.D.[4] ('71, Sep '72)
Austin Lowrey, Jr., M.D.[5] ('71, Sep '72)
P. Reed Larsen, M.D.[6] (Sep '72, Mar '74)
Joseph E. Rall, M.D., Ph.D.[7] ('71)
Jacob Robbins, M.D.[7] (Mar '72)
Kanti R. Rai, M.D.[1] ('70)
Jan Wolff, M.D.[7] (Mar '74)
John Steele, M.D.[8] (Mar '74)
Stanton H. Cohn, Ph.D.[1] (Mar '74)
Yang H. Oh, Ph.D.[1] ('71)
Nathaniel A. Greenhouse[1] (Mar '74)
Maynard Eicher[9] (Mar '73)
Francis Momotaro, Practitioner[9] (Mar '72)
Ezra Riklon, Practitioner[9] (Sep '72, Mar '73)
Jeton Anjain, Practitioner[9] (Sep '72)

OBSERVERS, MEDICAL

William Peck, M.D.[8] (Mar '72)
Haruo Ezaki, M.D.[10] (Sep '72)
Toshyuki Kumatori, M.D.[11] (Sep '72)
William Cole, M.D.[12] (Sep '72)
E. Eric Pochin, M.D., FRPC[13] (Sep '72)
C. Hayakawa, M.D.[14] (Sep '72)

OBSERVERS, CONGRESS OF MICRONESIA

Olympia Borja (Mar '73)
Charles Dominik (Mar '72, Mar '73)
Brian Farley (Sep '72, Mar '73)
Ataji Balos (Sep '72, Mar '73)
Hans Willianda (Sep '72)
John Del Rosario (Mar '74)

OBSERVERS, US AEC

William Streenan (Mar '74) James Miller (Sep '74)

MEDICAL TECHNICAL SPECIALISTS

William A. Scott[1] ('70, '71, Mar, Sep '72, Mar, Sep '73, Mar, Sep '74)
Douglas Clareus[1] ('70, Mar, Sep '72, Mar '73, Mar '74)
Michael S. Makar[1] ('70, '71, Mar, Sep '72)
Peter Heotis[1] (Sep '73, Mar, Sep '74)
Robert A. Brown[1] (Mar '73, Mar '74)
Sebio Shoniber[9] ('70, '71, Mar, Sep '72, Mar '73, Mar '74)
Nelson Zetkeia[9] ('70, '71, Mar, Sep '72, Mar, Sep '73, Mar, Sep '74)
Ernest Libby[9] ('71, Mar '72, Mar '73)
Kosang Mizutani[9] (Mar, Sep '72, Mar '74)
Joseph Saul[9] ('71, Mar, Sep '72)
Peter Bien[9] ('70, Mar '74)
Jude Jomule[9] (Sep '72)
Kimra Riklon[9] (Sep '72)
Laijo Elanjo[9] (Sep '74)
John C. Rothmann[1] (Mar '74)
Joseph A. Ash[1] (Mar '74)

TECHNICAL SPECIALISTS

Douglas Humphrey[1] ('70) Charles Tomesch[1] ('71)

[1] Brookhaven National Laboratory, Upton, New York 11973
[2] Case Western Reserve University, Cleveland, Ohio 44109
[3] Veterans Administration, Brooklyn, New York 11209
[4] M.D. Anderson Hospital, University of Texas, Houston, Texas 77025
[5] U.S. Army (Ret.), Box 503, Route 2, Lorton, Virginia 22079
[6] University of Pittsburgh, Pittsburgh, Pennsylvania 15213
[7] National Institutes of Health, Bethesda, Maryland 20014
[8] Department of Medical Services, Trust Territory of the Pacific Islands, Saipan, Mariana Islands 96950
[9] Naval Medical Research Institute, Bethesda, Maryland 20014
[10] Hiroshima University School of Medicine, Hiroshima, Japan
[11] National Institute of Radiological Sciences, Chiba-shi, Japan
[12] U.S. Public Health Service, Rockville, Maryland 20850
[13] University College Hospital Medical School, London, England
[14] Tokyo, Japan

BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973

NOTICE

This report was prepared as an account of work sponsored by the United States Government. Neither the United States nor the United States Energy Research and Development Administration, nor any of their employees, nor any of their contractors, subcontractors, or their employees, makes any warranty, express or implied, or assumes any legal liability or responsibility for the accuracy, completeness or usefulness of any information, apparatus, product or process disclosed, or represents that its use would not infringe privately owned rights.

Printed in the United States of America
Available from
National Technical Information Service
U.S. Department of Commerce
5285 Port Royal Road
Springfield, VA 22161
Price: Printed Copy, Domestic $6.25;
Foreign $8.75; Microfiche $1.45

September 1975 1400 copies

Preface

The medical examinations of the Marshall Islanders accidentally exposed to radioactive fallout in 1954 have now covered a span of 20 years. A 5-year period has elapsed since findings have been presented in full, and this report includes details of this period as well as a summary of the data for the entire 20 years. Some background information is presented first.

The Marshall Islands were chosen as an atomic bomb proving ground at the time of Operation Crossroads at Bikini in 1946 and were subsequently used for numerous tests at both Bikini and Eniwetok until the moratorium was declared in 1958. These islands were chosen because of their isolated location in the vast, nearly empty ocean area of Micronesia just north of the equator, east of the Caroline Islands, and northwest of the Gilbert Islands, about halfway between the Hawaiian Islands and Australia. The Marshall Islands comprise 34 islands and atolls spread over some 180,000 square miles of ocean. Each atoll consists of a low-lying chain of sandy, palm covered islands connected by a reef surrounding and protecting a central blue-green lagoon. Habitable land area is surprisingly small – only about 70 square miles in the entire Marshall Islands. The climate is tropical, and the trade winds blow much of the year. The atolls involved in the 1954 accident are located in the northern Marshalls: Rongelap and Ailingnae about 100 nautical miles east of Bikini, Rongerik about 135 miles east of Bikini, and Utirik 275 miles east of Bikini.

During the past century the Marshallese people have been governed by four countries. Germany purchased the Marshall Islands from Spain in 1886 and governed them until 1917, when Japan took over under a League of Nations mandate; Japan declared ownership in 1933 when she broke from the League of Nations. The United States liberated the Marshalls in 1944, during World War II, and was given authority under a United Nations mandate to administer the scattered islands of Micronesia as the Trust Territory of the Pacific Islands; this was done first by the U.S. Navy and later by the Department of the Interior.

The early explorers and traders found the islands inhabited by friendly, gentle, primitive people, living in thatched huts, fishing from outrigger canoes, and subsisting entirely from the local environment. The increasing encroachment of civilization via traders, missionaries, and governing authorities has changed their way of life. The missionaries brought religion, clothing, and changed customs. A dollar economy based on copra production and employment by local governments has tended gradually to supplant the environmental subsistence.

Sanitary conditions and health care in the Marshalls had been primitive, with treatment of the sick often involving a large element of superstition and magic, and apparently did not improve under Japanese rule. During World War II the Marshallese were forced to live on low calorie diets with inadequate medical care. When the U.S. was made trustee in 1947, the Navy, under the auspices of its Bureau of Medicine and Surgery, undertook a comprehensive medical survey of island populations in the Trust Territory including the Marshall Islands, using one of its vessels, the USS *Whidbey*.[1] The survey showed that unsanitary conditions with regard to flies, garbage disposal, and excretory habits made for multiple intestinal parasitic infestations and diseases. A high percentage of the people had positive Kahn tests, associated with yaws, and widespread treatment with penicillin proved extremely effective. Diseases of the eyes and skin, acute and chronic respiratory diseases, and vitamin deficiencies were especially common. No malaria, filariasis, yellow fever, or cholera was seen. Subsequent health care in the Trust Territory was hampered by lack of trained medical personnel and poor transportation to the outer islands. This was the status when our special medical examinations began.

Some of the events over the years in the Marshall Islands that are relevant to the medical surveys are listed in Appendix 1.

The first examinations in 1954 after the fallout were conducted by an emergency medical team, requested by the Atomic Energy Commission, directed by Dr. E.P. Cronkite and composed largely of Navy medical personnel whose service experience proved extremely helpful for this field-type operation. Resurveys were conducted at 6 months under Dr. V.P. Bond and at 12 months under Dr. Cronkite. In 1956 the surveys were placed under the direction of the author at Brookhaven National Laboratory. Since then the surveys have been conducted in conjunction with the Department of Health Services of the Trust Territory and with the participation of medical specialists from the Armed Forces and from medical centers

Exposed Marshallese at Kwajalein immediately after evacuation, March 1954.

Ejet Village, where Rongelap people lived for 3 years.

Part of decontamination procedures at Kwajalein, March 1954.

Typical Rongelap house, 1954.

Initial medical examinations at Kwajalein, March 1954.

Majuro residents giving farewell gifts to departing Rongelap friends, 1957.

in the United States, including endocrinologists, cardiologists, hematologists, internists, surgeons, ophthalmologists, and parasitologists (see Appendix 2). The variety of specialists indicates the diversity of the examinations conducted over the years.

The BNL group has been fortunate in maintaining a close liaison with the Atomic Bomb Casualty Commission in Japan, and several physicians formerly associated with it have participated in our surveys. We have also had a close relationship with the people studying the Japanese fishermen exposed to the Bravo fallout; in 1964 exchange visits were arranged for Dr. Kumatori and the author to attend surveys both of these fishermen and of the Marshallese.

In May 1954, upon completion of the initial examinations (conducted at Kwajalein), the 82 Rongelap people, because of the contamination of their home islands, were moved to Majuro Atoll, and they were examined there in September 1954 and in March 1955, 1956, and 1957. They returned to Rongelap, to live in a newly built village, in July 1957, after radiological surveys had shown the island to be safe for habitation. The 157 Utirik people were taken home after the initial examinations, since their island was only slightly contaminated and was considered safe for habitation. The 28 military personnel exposed on Rongerik Atoll were taken to Tripler Army Hospital for further examinations.

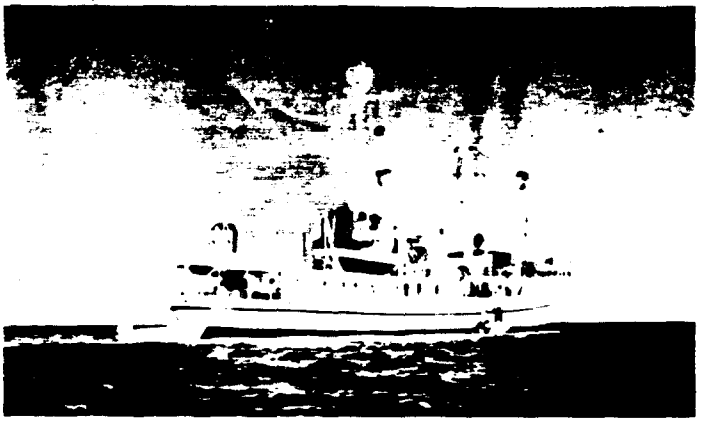
Following the initial acute effects of radiation on the Rongelap people, the examinations over the next 9 years revealed few significant findings. An increase in miscarriages and stillbirths among the exposed women during the first 5 years was thought to be related to radiation exposure, but general health and disease incidence in the exposed people were about the same as in the unexposed Rongelap people who had returned from other islands and served as a comparison population. During this uneventful period, suggestions were made for reducing the scope or frequency of the surveys, but these were never seriously considered since it was realized that little is known about the late effects of radiation on humans and effects might still appear. In 1963 the unexpected apearance of growth retardation in some children and of thyroid abnormalities fully justified this reasoning. In 1972 a case of fatal acute myelogenous leukemia developed in a 19-year-old exposed Rongelap boy, indicating the need for additional surveillance. The surveys therefore were expanded to include a hematological check in the exposed Rongelap people every 6 months. At that time also difficulties were encountered in maintaining a strict thyroid treatment program in the exposed Rongelap people, with potential serious results, particularly in the people who had had thyroid surgery. The decision was therefore made to place a resident physician in the Marshall Islands to keep check on the treatment program and assist the Trust Territory in general health care of the Rongelap and Utirik people.

The medical findings on the Marshallese have been published in numerous BNL reports and journal articles.[2-23]

Some problems have been encountered in carrying out the medical surveys in the islands. Careful planning is required for all necessary equipment and supplies to be on hand at the remote location, and procedures must be adapted to field conditions with proper electronic support. Some technical procedures such as staining of blood smears and cell cultures present problems under tropical, humid conditions. Providing adequate transportation has been a major difficulty. While the Navy furnished LST's and amphibious planes, problems were minimal, but for most of the surveys we have had to rely on cargo ships chartered by the Trust Territory, and these have sometimes proved unreliable in meeting schedules or hazardous because of faulty life-saving devices and inadequate navigational equipment. Once the survey team was lost at sea for more than a day trying to find an island, and twice ships ran aground on reefs. Small-boat travel in rough waters was hazardous; the trip from ship to shore was sometimes in outrigger canoes and might result in wet passengers and baggage. To overcome these problems, an LCU was recently acquired for carrying out the AEC-sponsored radiological and medical surveys in the Marshall Islands.

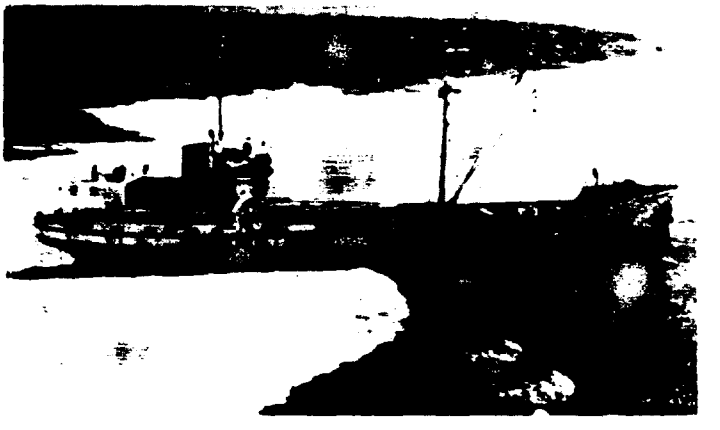
Other problems included the following: The language barrier hampered communication with the people, even though a limited number of reliable English-speaking people were usually available. The lack of adequate vital statistics resulted in uncertainty in the exact age of many of the people; and medical records were poorly kept, particularly in the outer islands (e.g., the health aide would often give the cause of death as "too old"). It was difficult to obtain consent for an autopsy, even at district medical centers, because of feelings against mutilation of the body after death. Some of the subjects thought that drawing blood made them feel weak; and some questioned the

People returning to Rongelap (1957) after 3 years at Majuro.



Trust Territory cargo vessel carrying personnel and supplies for medical surveys, 1966.

Rebuilt village on Rongelap to which people returned in 1957.



Trust Territory cargo vessel on reef after departure from Rongelap, 1972.

Support from Navy amphibious planes during the early annual examinations facilitated visits to the outer islands.

Moving equipment ashore. In recent years this task has been facilitated by the acquisition of a jeep and a tractor.

need for continued examinations when no significant findings were reported. In spite of such problems, the people have always been friendly, and, except on one occasion (a case of political interference, described below), have generally cooperated in the examinations. Psychological reaction to the fallout has been reflected at various times in fears regarding fertility, a belief that fish poisoning was due to radioactivity rather than to the poisonous nature of the fish, feelings of weakness attributed to radiation, and fear of thyroid cancer and leukemia (following the death of the Rongelap boy).

A major concern for the past 5 years has been local political interference and questions concerning the medical examinations. The Trust Territory now has a Congress of Micronesia (not connected with the U.S. administration), which has appointed a Special Committee to investigate the health status of the Rongelap and Utirik people. Unfounded accusations were made by certain local politicians that the United States deliberately exposed the people to fallout so that they could be used as "guinea pigs" to study the effects, and that the medical team was concealing deaths from radiation and was not reporting all of its findings. In March 1972 the survey was interrupted by the politicians and had to be aborted at Rongelap. Afterwards the Congress of Micronesia appointed medical observers to accompany the team on a survey in September 1972. Two prominent physicians from Japan (Drs. H. Ezaki and T. Kumatori), one from England (Dr. E.E. Pochin), and one from the U.S. Public Health Service (Dr. W. Cole) accompanied the team and participated in the examinations. Their report to the Congress of Micronesia was most favorable and has done much to quiet the political accusations. The Special Committee on Rongelap and Utirik published a lengthy report[24] of its investigations, including the report of the observers, and this also has helped bring about a more favorable attitude to the medical examinations.

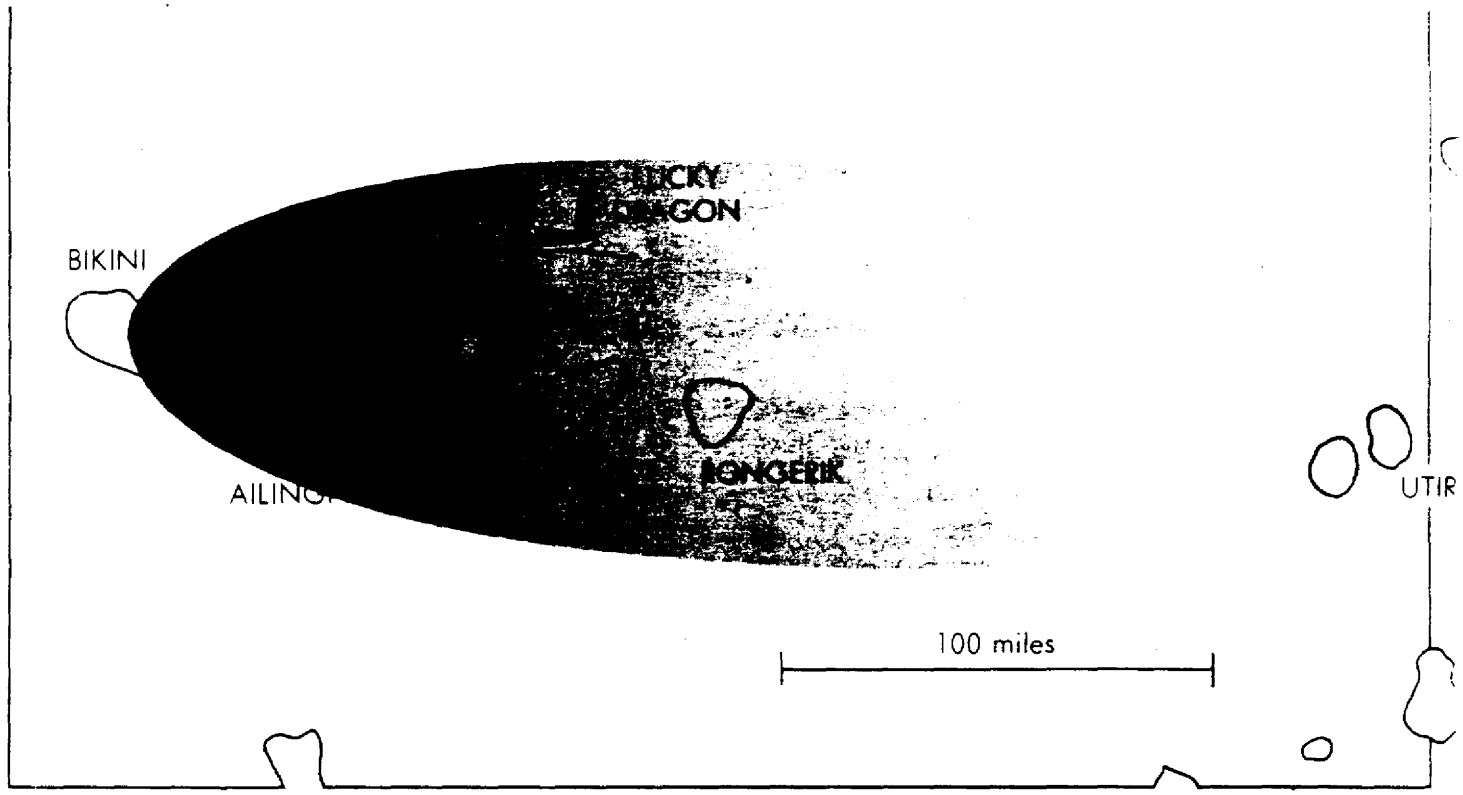
Two major bills have been initiated by the Congress of Micronesia. One concerns payment of travel and living expenses for any patient from Rongelap and Utirik on our examination list who needs hospitalization. This bill has been approved by U.S. authorities and is being implemented. The other concerns compensation for the Rongelap people for ill effects of fallout exposure and provides also for a payment to the Utirik people and for special compensation to the parents of the boy who died of leukemia. (See Appendix 1.) We believe that the Rongelap people are entitled to further compensation for injuries received, although, on the basis of the effects of their previous compensation in 1965, the ultimate benefits are doubtful, and another payment might push them further toward the state of welfare and perpetual dole. Dr. William Peck, the former Director of Health Services in the Trust Territory, has put this situation nicely: "To withhold further funds and thus deepen the grievance, or increase the compensation and thereby the dolor of dependence – either course of action seems only to compound their original injury, and our dilemma!"

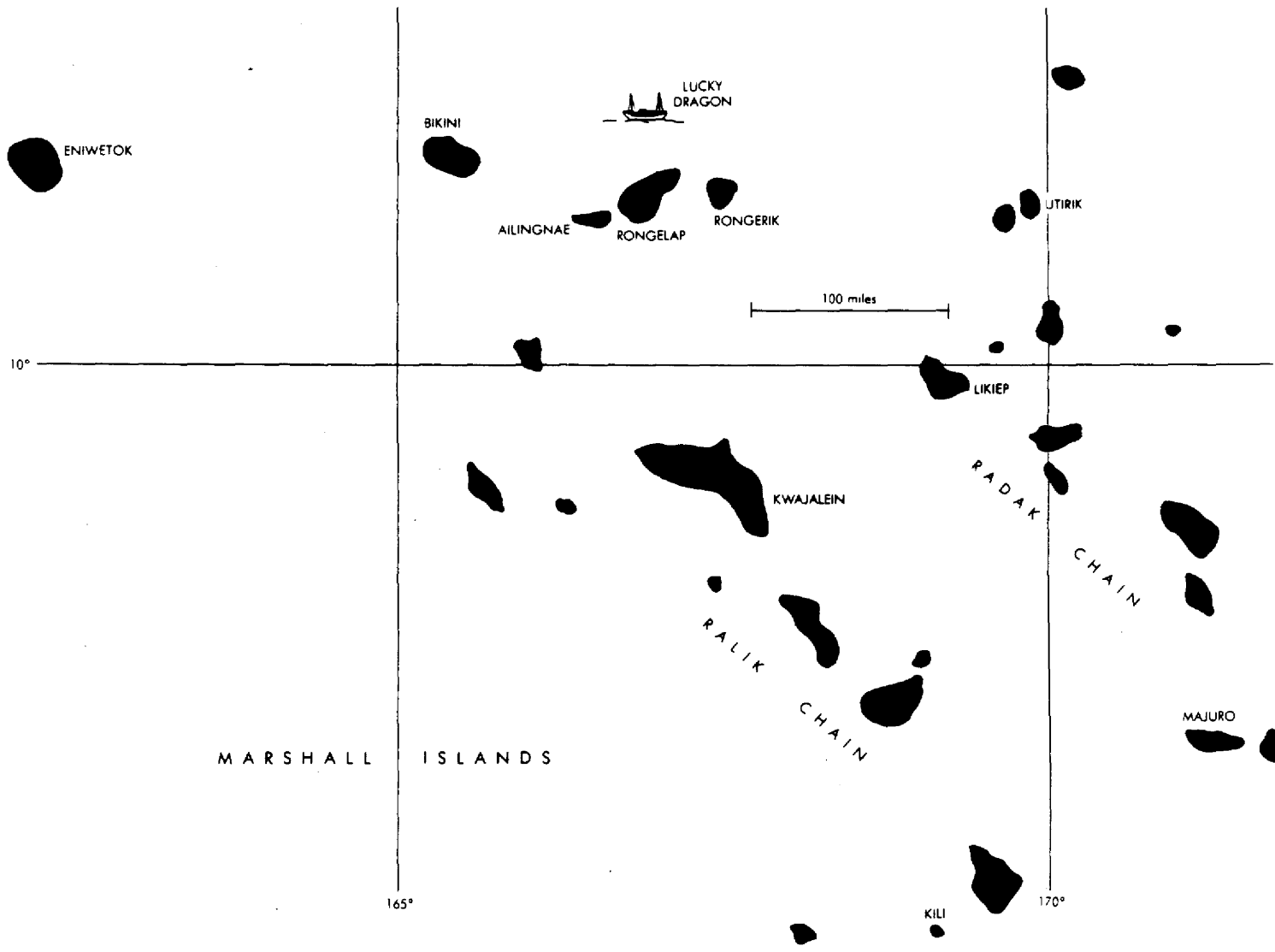
Further problems in the Marshall Islands are associated with the return of the former residents to live on Bikini and Eniwetok, which were contaminated during the testing program. Numerous radiological surveys have indicated that with certain restrictions these atolls can be habitable again. Since a number of concrete homes have been completed on Bikini, about 40 people have returned, but they must be subsidized because it will be several years before the coconut and other trees bear fruit. A number of people are expected to return to Eniwetok to live in the near future. The medical team as well as other radiological safety personnel at BNL have been assigned the responsibility of personnel and environmental monitoring to assure the radiological safety of the returning people. Personnel monitoring and environmental surveys at Bikini were instituted a few years ago, and only very low levels of internally absorbed radionuclides have been noted. ERDA is sponsoring continuing surveys of these atolls, being done by several institutions.

The medical surveys have been a rewarding experience for all the personnel who have participated. We have been fortunate in obtaining the services of talented specialists and technicians and in having the continued staunch support and assistance of many people at Brookhaven National Laboratory, ERDA, and the Trust Territory, and the authorities at Kwajalein, without which the success of the surveys would not have been possible. The medical team has developed a deep friendship for the Marshallese over the years. In spite of the injuries and hardships resulting from the 1954 accident, they have cooperated with us even through the period of political disturbances. With the improved political climate and attitude toward the examinations we look forward to continuing these important and challenging missions and to offering specialized health care to these Marshall Islanders in future years. R.A. Conard, M.D.

CONTENTS

CONTENTS


BIKINI
100 miles


LUCKY DRAGON
BIKINI
ENIWETOK
AILINGNAE
RONGELAP
RONGERIK
UTIRIK
100 miles
10°
LIKIEP
KWAJALEIN
RADAK CHAIN
RALIK CHAIN
MAJURO
MARSHALL ISLANDS
165°
170°
KILI

A TWENTY-YEAR REVIEW OF MEDICAL FINDINGS IN A MARSHALLESE POPULATION ACCIDENTALLY EXPOSED TO RADIOACTIVE FALLOUT

I. Background

A. THE ACCIDENT

The testing of nuclear devices in the Marshall Islands (see Hines[25]), beginning with Operation Crossroads at Bikini in 1946 and ending with the moratorium in 1958, did not result in significant radiation exposure to personnel or fallout contamination outside the test area except in one case. On March 1, 1954, the detonation from a tower of a thermonuclear device, Bravo, in the Castle Series of tests at Bikini resulted in a serious fallout accident. The yield was about 17 megatons, considerably greater than expected, and an unpredicted shift in winds in the upper atmosphere caused the radioactive cloud to drift over and deposit fallout on several inhabited atolls to the east: Rongelap with 64 people, Ailingnae with 18 people, Rongerik with 28 American servicemen, and Utirik with 157 people (see Figure 1). A Japanese fishing vessel in the area, the *Lucky Dragon*, with 23 fishermen aboard was also exposed (see Appendix 3). The fallout is thought to have commenced at Rongelap about 4 to 6 hr after the detonation, at Rongerik about 7 hr after it, and at Utirik about 22 hr after it. Its duration on the islands is uncertain but has been estimated as about 12 hr, the greater part of the fallout occurring early in the period.[2] The estimated dose of gamma radiation to the island populations is discussed in Section II. A. The American servicemen on Rongerik noted that the needle on a telemetering instrument suddenly began rising and went off-scale in 30 min, beginning about 6 to 7 hr after the detonation. An alarm was radioed to the task force, and a plane flying low confirmed that significant fallout had occurred.

The exposed people were evacuated by planes and Navy ships within about two days and taken to Kwajalein, 175 miles to the south. They were first examined by the medical group at the Naval Dispensary there. Eight days after the accident a medical team consisting of 21 doctors and technicians, largely from the Navy, which had been requested by the AEC, arrived at Kwajalein. For two months the team took medical histories, did repeated physical examinations including studies and photographs of beta burns of the skin, made numerous hematological tests, and monitored for external and internally absorbed radioisotopes. Complete removal of the radioactive contamination from the skin and hair required many cleansing procedures; the coconut oil used on the hair was particularly retentive. At the end of the examination period, most of the skin burns had healed and, although significant hematological depression had occurred, no serious illnesses were evident that could be related to radiation injury. The Marshallese people were taken to a tent encampment on Ebeye Island for a stay of several weeks. Since Utirik Atoll was only very slightly contaminated from the fallout, it was considered safe for habitation, and the Utirik people were returned there with fresh supplies, clothing, and livestock. Rongelap Atoll was too contaminated to allow immediate return and its people (along with the 18 from Ailingnae) were taken to a temporary village built for them on Ejet Island in Majuro Atoll, where they lived for 3 years until their return to Rongelap. The American servicemen were taken to Tripler Army Hospital for further examinations and later returned to duty.

B. ANNUAL EXAMINATIONS

Medical examinations of the Rongelap people were conducted at their temporary home on Majuro in September of 1954 and in March of 1955, 1956, and 1957. In 1954 an unexposed group of Marshallese living at Majuro was chosen as a comparison population for these examinations. This group, however, was composed of people from many of the Marshall Islands who were not easily located for subsequent examinations. Unexposed Rongelap people gradually moved to Ejet to live with their fellow islanders, and this group increased further on the return to Rongelap. These unexposed Rongelap people were included in the examinations and have served as an excellent comparison population since they are blood relatives of the exposed Rongelap people, match reasonably well for age and sex, and live under the same environmental conditions (see Section III. A.).

Medical survey team, March 1970.

Medical survey team, March 1971.

Medical survey team, March 1972.

Medical survey team, September 1972.

Medical survey team, March 1973.

Medical survey team, March 1974.

By 1957, radiological surveys indicated that, in spite of slight lingering radioactivity, Rongelap Atoll was safe for habitation. A completely new village was constructed, and in July the Rongelap people, more than doubled in number by the influx of relatives, were taken there on an LST with all their personal belongings, bed mats, and livestock. After arrival ceremonies they happily settled down to life in their new village. In March 1958 the survey was carried out for the first time at Rongelap; subsequent annual examinations have been done mostly at Rongelap with supplementary visits to other islands.

The Utirik people, after the initial examinations, were next examined on their home island in 1957. They have since been examined only about once every 3 years because their fallout exposure was minimal. The American servicemen have not been seen by our medical team since the initial examinations.

During recent years the Rongelap and Utirik people have moved in large numbers to the district centers at Majuro and particularly Ebeye (Kwajalein Atoll), where many are employed by the Kwajalein Missile Range. This had necessitated examinations at Majuro and Ebeye in addition to their home islands. (Table 1 shows the present geographical distribution of the people.)

The many studies that have been done on the Marshallese are directly or indirectly related to possible radiation effects. Often a single blood sample drawn for routine hematology has been used for many tests. Multiple blood sampling has been avoided whenever possible.

C. 1970-1974 SURVEYS

During the past 5 years a number of problems have arisen: thyroid abnormalities continued to develop in the exposed Rongelap people in spite of hormone treatment; an exposed Rongelap boy died from leukemia that may or may not have been related to radiation exposure; transportation deteriorated; and political interference was started which has been particularly frustrating and has threatened to put a stop to the medical supervision and care provided to the exposed people by the medical team.

In 1972, following the death from leukemia, the decision was made that more frequent hematological examinations of the exposed people were needed, and therefore such examinations were instituted every September in addition to the March survey.

Also in 1972, the decision was made to place a resident physician in the Marshall Islands. An increasing number of exposed Rongelap people were developing thyroid nodules, most of which required surgical intervention, and many of them were not adhering strictly to the thyroid treatment program, which is considered extremely important, especially after surgery. A resident physician would be able to monitor this program and, since the Trust Territory was short on medical personnel, could assist in general health care of the Rongelap and Utirik people. The plan is for such a physician to alternate between BNL and the Marshall Islands every two years. The first one has just completed a 2-year term, and another is taking his place soon.

Transportation has always been a major problem for the medical survey teams. Air transportation to the Pacific improved greatly with the advent of the jet plane, but travel by ship among the Marshall Islands (several hundred miles between atolls through rough seas) has remained difficult. The travel problem has been magnified by the need for the resident physician to visit Rongelap and Utirik quarterly, and by the added responsibility of the medical team, since 1971, for radiation monitoring of Bikini and more recently of Eniwetok, since the people displaced by the bomb testing program are

Table 1

Location of Rongelap and Utirik People, 1974

	Rongelap	Utirik	Majuro	Ebeye	Bikini	Other	Total
Rongelap exposed	31	–	5	24	–	6	66
Rongelap unexposed	37	–	14	65	7	8	131
Utirik exposed	–	41	43	22	–	10	116
Total	68	41	62	111	7	24	313

being returned to these islands. It became apparent that a special vessel was needed, and in 1973 the AEC arranged for the Army to provide an LCU (landing craft utility) based and maintained at Kwajalein (with AEC funding). This vessel, though small and slow, has the distinct advantage that it can be beached, so that ship-to-shore transfer of equipment and personnel by small boat is unnecessary. Three 30-ft air-conditioned trailers have been placed on the tank deck, one equipped as a "sleeper," one containing a whole-body counter (shadow-shield lead assembly and electronic equipment), and one set up for clinical examinations with a doctor's office, a small laboratory, and an x-ray facility. The LCU with these trailers is now in full use and affords tremendous improvements in the operations.

Examination facilities at Utirik consist of several Butler-type buildings left by a weather station group. On Rongelap several examination trailers have been installed so that the schoolhouse need no longer be used. Examinations trailers have also been installed on Ebeye and Majuro.

Major events in the Marshall Islands related to the surveys, including the political problems of the past few years, are summarized in Appendix 1.

Before and after each survey, village meetings have always been held on Rongelap and Utirik to inform the people of the objectives of the examinations, the findings, and recommended treatments. Because of the language barrier, the people did not always understand the need for the examinations, or their results. Every effort is now being made to correct this. Preparation of a question-and-answer booklet on the effects of the fallout and treatment on the Rongelap and Utirik people was recommended by the Congress of Micronesia; such a booklet has been printed at BNL for the Trust Territory Health Services and is being distributed; it should do much to correct many of the former misconceptions (see Appendix 4).

During the 20 years covered by these reports drastic changes have occurred in the Marshall Islands District.

The population more than doubled from 1948 to 1973, the total going from ~10,000 to ~25,000. The increases were most dramatic on Majuro (going from 1,200 to 10,300) and on Ebeye (going from 750 to 5,000). On the outer islands the total population changed less (Rongelap, 100 to 167; Utirik, 126 to 217), but the age distribution became abnormal. Young adults have gravitated toward the district centers, and the "home islands" are left with children and old people. A recent survey on Rongelap showed that of its 167 people, 115 were <15 years old and 30 were >50. The flux is great: during any given field trip up to 30 people may be arriving or leaving.

The district has opened up to foreign travel. Majuro has a jet airport, paved roads, electricity, and telephones. Expectations are rising, and when public services fail to keep pace with increasing demand, dissatisfaction is more vocal.

The atmospheric bomb tests ended in 1958. Kwajalein was abandoned as a Navy Base in 1960 and was made part of the Army's missile test range. For the visiting medical teams, loss of Navy assistance in transportation has increased the logistic problems, but the continuing support of the Army Base has been invaluable.

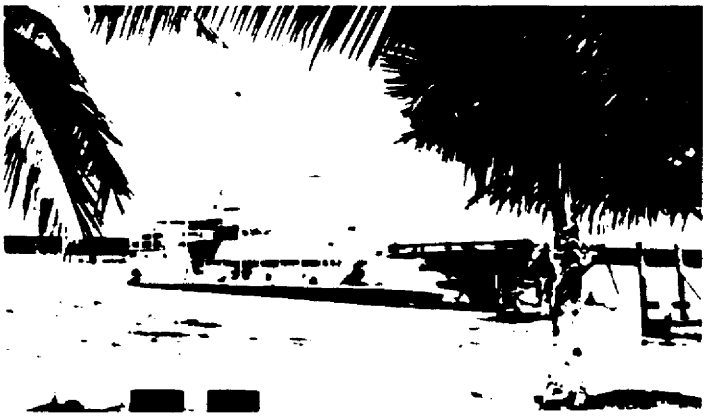
The people of Rongelap and Utirik have always been most friendly and cordial to the medical team. Except for political interference with the 1972 examinations, the people have always been cooperative.

II. Initial Findings

A. DOSE ASSESSMENT

1. Source

The radiation exposure of the Marshallese was due entirely to fallout, since the detonation site was too far away for thermal, blast, or direct irradiation effects. (In constrast, direct effects were responsible for all the injuries from the atomic bombs in Hiroshima and Nagasaki, with little or no fallout.) The fireball from the 1954 Bravo device, detonated from a tower, touched the surface of the earth at Bikini, and large amounts of material were drawn up and mixed with fission products in the bomb cloud. Because of an unpredicted shift in the winds in the upper atmosphere, fallout was deposited in a cigar-shaped area 20 to 40 miles wide extending ~200 miles to the east of Bikini (see Figure 1). The radioactivity was due to fission products and some neutron-induced isotopes; the fallout contained little fissile material. The radiation was therefore almost entirely from gamma and beta rays of varying energy from numerous fission radioisotopes. The time after detonation when fallout began was estimated as 4 to 6 hr at Rongelap, ~7 hr at Rongerik, and 22 hr at Utirik



LCU now used for travel between the islands, 1974.

Senator O.T. Borja (second from left) of the Congress of Micronesia, Chairman of the Committee Studying the Medical Status of the Rongelap and Utirik People, visiting BNL in 1973; shown with Drs. E.P. Cronkite (left), V.P. Bond, and R.A. Conard.

Examination trailer on Rongelap, 1965.

Thyroid examination on Rongelap, 1969.

Rongelap patients with interpreter in New York, visiting UN Building before undergoing thyroid surgery, 1968.

Hematological examinations, Rongelap, 1965.

Table 2

Estimation of Gamma Dose[2]

Group designation	No. in group	Approx. time of commencement of fallout	Time of evacuation	Instrument readings used in dose calculations	Best estimate of total gamma dose in air (R)
I. Rongelap	64	H + 4 to 6 hr	H + 50 hr (16 people) H + 51 hr (48 people)	375 mR/hr, H + 7 days	175
II. Ailingnae	18	H + 4 to 6 hr	H + 58 hr	100 mR/hr, H + 9 days	69
III. Rongerik	28	H + 6.8 hr	H + 28.5 hr (8 men) H + 34 hr (20 men)	280 mR/hr, H + 9 days	78
IV. Utirik	157	H + 22 hr	Started at H + 55 hr Completed at H + 78 hr	40 mR/hr, H + 8 days	14

and the fallout duration as ~12 hr, with most of the dose delivered early in that period. The times when people were evacuated from the islands are shown in Table 2.

2. Gamma (Whole-Body) Dose

The fallout (where seen) resembled snow or mist and was deposited relatively homogeneously so that the individuals on each island were considered to have received about the same estimated dose of gamma radiation. The children may have had a somewhat higher dose than that calculated for the adults because, being smaller, they were closer to the ground and received larger midline doses. This possibility is supported by the higher incidence of early nausea and vomiting and the greater depression of blood elements in the young children. The flimsy houses afforded little attenuation of the radiation. The whole-body doses were calculated from measurements with radiation field survey instruments held 3 ft above the ground, made within a week after the detonation, by extrapolation to the time of exposure with the energy spectrum and decay taken into consideration. Table 2 shows the calculated gamma doses for the different populations exposed. The degree of hematological depression that developed later is consistent with the doses calculated. In view of the 4π geometry of exposure, the midline doses to individuals were higher than those obtained with the usual bilateral exposure of x-radiation.[2]

3. Skin Dose

The dose to the skin surface was much greater than the whole-body gamma dose because of the large amount of beta radiation absorbed by the skin. The actual skin doses, although impossible to calculate, probably amounted to thousands of rads, and their range of values, due to different amounts of fallout sticking to different areas, accounted for the spotty nature and varying intensity of lesions. The extensiveness of the beta burns in each island group correlated roughly with the amount of fallout visible on their island (see Table 6). Most of the skin dose was due to fallout deposited directly on the skin, but some was due to beta radiation from fallout on the ground (estimated at Rongelap to be 2000 rads at the level of the dorsum of the feet, 600 rads at hip level, and 300 rads at the head, on the basis of continuous exposure and no shielding).[2] It was fortunate that the beta radiation had an average energy insufficient to penetrate deeply into the skin and therefore resulted for the most part in superficial beta burns. The average beta particle probably did not penetrate much beyond the basal layer of the skin (~100 μ). However, since epilation occurred in many people, the region of the hair follicles must have received a dose equivalent at least to the minimal epilating dose of 400 rads of 200-kVp x rays.

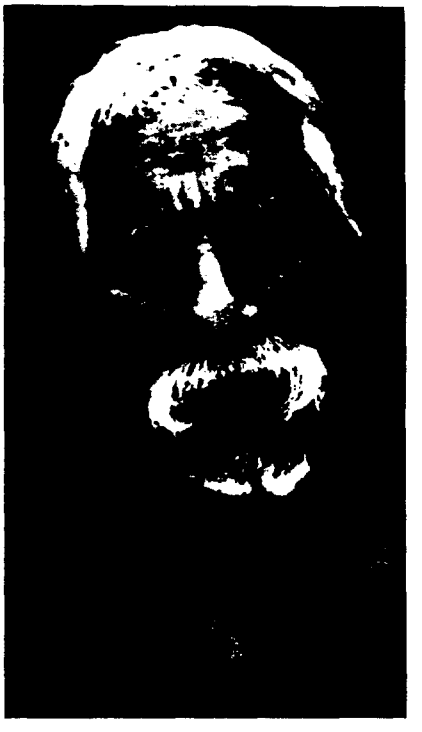
4. Internal Dose Calculations

Internal absorption of radioisotopes was due to inhalation as the radioactive cloud passed over and to ingestion of food and water contaminated with fallout, water probably being a major source. Drinking water is obtained by collecting rainfall from the roofs into catchments, and a slight rain was reported on Rongelap the night of the fallout. Since the cisterns were nearly empty, the dilution effect was minimal. Water was being rationed at that time, and it was drunk in spite of warnings from the health aide. On Rongerik food and water were better protected from fallout deposition.

Internal levels of radioisotopes absorbed from the fallout were assessed by numerous radiochemi-

Marshallese dental practitioner examining teeth as part of medical survey, Rongelap, 1960.

Net fishing at Rongelap, 1959.



Elderly Marshallese male, 1965.

Party given by medical team at conclusion of annual survey, Rongelap, 1970.

Rongelap people entertaining medical team at feast after examinations, Rongelap, 1971.

Copra being carried ashore. Copra production is the chief source of income in the Marshall Islands.

Table 3

Estimated Body Burden (μCi) of Rongelap People[2]

	Activity at day 1	Activity at day 82
^{89}Sr	1.6 - 2.2	0.19
^{140}Ba	0.34- 2.7	0.021
Rare earth group	0 - 1.2	0.03
^{131}I (in thyroid gland)	6.4 -11.2	0.0
^{103}Ru	0 - 0.013	-
^{45}Ca	0 - 0.019	0.0
Fissile material	0 - 0.016 (μg)	0.0

cal analyses of urine samples, beginning 15 days post exposure, for ^{89}Sr, ^{140}Ba, ^{131}I, the rare earth group, and fissile material. As expected, the Rongelap people had the highest body burdens. By 6 months, beta activity in the urine samples was barely detectable. Table 3 shows the main isotopes found at day 1 (extrapolated values) and at day 82. The agreement between the findings at the two laboratories is close considering the techniques available at that time. Levels in the Ailingnae group were about one-half and in the Americans about one-quarter the levels in the Rongelap group. Only isotopes of iodine, strontium, barium, and a few rare earth elements were absorbed to any significant degree. In the Rongelap group, at day 1, ^{89}Sr and ^{131}I were near the maximum permissible levels, and the estimated total amount of radioactive material in the gastrointestinal tract was about 3 mCi; whether this had any relation to the early gastrointestinal symptoms is not known.[2] Radiological monitoring of personnel and environment at Rongelap and other atolls in subsequent years is discussed in Section VI.

5. Thyroid Dosimetry

The fallout produced several possible sources of radiation exposure to the thyroid gland. The gamma radiation resulted in thyroid doses of 175 rads in the Rongelap people, 69 rads in the Ailingnae people, and 14 rads in the Utirik group. Iodine isotopes are produced in relatively high yields by the fission process. Some are too short-lived to be of consequence, but ^{131}I, ^{132}I, ^{133}I, and ^{135}I are sufficiently long-lived to cause a considerable dose to the thyroid following internal absorption and concentration in that gland, and these were absorbed both via inhalation and via ingestion in food and water (see Appendix 9C). Other internally absorbed isotopes (see Table 3) were not thought to be significantly absorbed by the thyroid and probably contributed little to the dose to that gland. Conversely, the radioiodines contributed only slightly to the whole-body radiation dose.

During the early period after the fallout, radioiodine was recognized as possibly its most hazardous constituent, but the estimated dose to the gland of 100 to 150 rads was not considered sufficient to cause later development of thyroid abnormalities. No acute effects were noted in any of the people that could be related to the internal absorption of radioiodines or other radioisotopes. Contamination of the skin resulting in extensive beta burns in the neck region in 70% of the people (see Figure 2) probably did not contribute to the thyroid dose because of the low energy of the beta radiation. Possibly slight absorption of radioiodines through the skin occurred.

When the people returned to Rongelap to live in 1957, no radioactive isotopes of iodine remained (except possibly very slight amounts of ^{129}I), and the principal remaining isotopes (^{137}Cs, ^{90}Sr), which were absorbed to low levels in the people, probably did not contribute to any significant degree to the thyroid dose.[6]

When thyroid lesions began developing in the Rongelap children in 1963, the dose to the thyroid of the Rongelap people was reevaluated by James.[26] His estimates of the gamma dose agreed approximately with the previous estimate of 175

Figure 2. Beta burns (March 29, 1954).

rads. Calculation of the dosage from radioiodines unfortunately had to be based on a single, pooled urine sample from Rongelap people collected 15 days post exposure. Harris[27] at Los Alamos had reported a low level of ^{131}I in this sample and had calculated a one-day thyroid content of 11.2 μCi based on the assumption of 0.1% urinary excretion of the maximum thyroid burden on the 15th day.

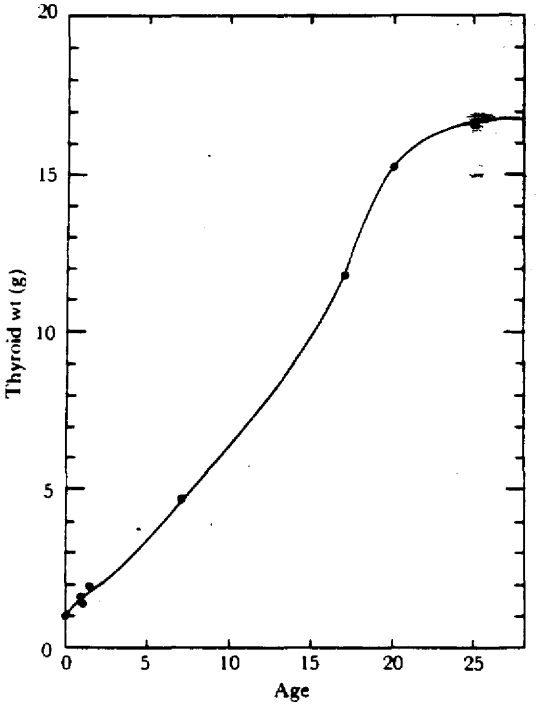


Figure 3. Weights of human thyroids in New York City. (From MOCHIZUKI ET AL.[28])

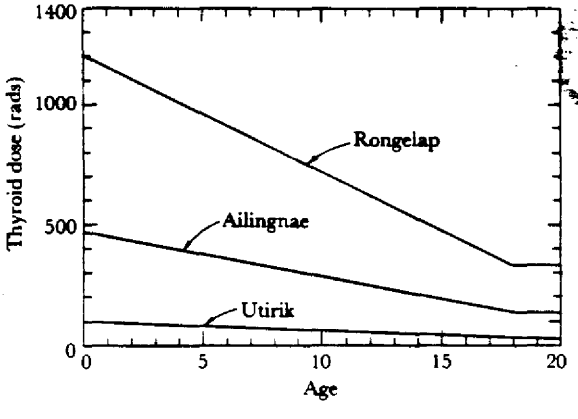


Figure 4. Thyroid dose versus age for children in exposed Marshallese groups.

James calculated doses for absorption from both inhalation and ingestion. He noted that the shorter-lived isotopes of iodine delivered 2 to 3 times the estimated dose delivered by ^{131}I alone. The importance of these shorter-lived isotopes in producing thyroid effects in the Marshallese will be referred to in Section IV. The dose to the thyroid of a Rongelap adult (including gamma) was calculated as about 335 rads (220 to 450 rads) and to that of a 3-year-old Rongelap child as 700 to 1400 rads. (The spread is due to uncertainties in estimating dose from absorption of radioiodines by inhalation versus ingestion.). With the assumption that the ratio of whole-body gamma doses to thyroid doses was the same as for the Rongelap people, James' calculations were used to estimate thyroid doses in the Ailingnae and Utirik groups; the results were 135 rads for the Ailingnae adults and 27 rads for the Utirik adults. The children's thyroid doses were based on the weight of the gland at various ages (Figure 3).[28] By using a linear relationship between the thyroid size and the dose calculated by James, the doses to individual children were taken from regression lines drawn for the three exposed populations (Figure 4); these are given in Section IV. In retrospect, the estimated average dose of 1050 rads to the thyroids of young children appears to be low, at least for two boys who developed atrophy and myxedema. The calculated doses are obviously rough estimates. The incompatability of the observed effects with the calculated doses based on ^{131}I must be related partly to the greater dose effect of short-lived iodine isotopes (see Appendix 9C).

B. ACUTE EFFECTS

1. Effects of Whole-Body Exposure

a. Early Symptoms. During the first two days, before evacuation, symptoms related to the skin and the gastrointestinal tract were noted in a large number of Rongelap people, in a lesser number among the groups exposed on Ailingnae and Rongerik, and in none of the Utirik group. The severity of the symptoms was correlated with the amount of fallout and the radiation dose. Skin symptoms (itching and burning of areas not covered by clothing) were noted in about one-fourth of the Rongelap people, and a few complained of irritation of the eyes and lachrymation. These symptoms were thought to be related to beta irra-

diation, but the caustic nature of the fallout may have contributed.

The G.I. symptoms in the Rongelap people consisted of anorexia and nausea (in about two-thirds of them) and to a lesser extent of vomiting and diarrhea (in one-tenth of them). Only a few in the Ailingnae group complained of these symptoms, and no one in the Rongerik and Utirik groups. The gamma exposure is thought to have been sufficient to account for the G.I. symptoms, but the ingested radioactive material (estimated at about 3 mCi) may have contributed. The G.I. symptoms were more prevalent in the Rongelap children (see Table 4). These symptoms lasted only about two days and had largely disappeared by the time the people arrived at Kwajalein.

b. Depression of Blood Elements. The early hematological findings and the findings in subsequent years are presented in tabular form in Appendix 6 and graphically in Section III. C. The early findings are summarized here and the later ones in Section III. C.

One of the earliest findings in the Marshallese indicative of significant exposure was lowering of leukocyte and platelet levels in the peripheral blood. This was most marked in the 64 people on Rongelap who had received 175 rads and less so in other groups receiving less exposure. The hemopoietic depression was roughly proportional to the gamma dose of radiation received. Even in the 157 Utirik people who received only an estimated 14 rads, it was possible to distinguish slight platelet depression in the group as a whole. The smaller groups on Ailingnae and Rongerik showed peripheral blood levels between those of the high and low exposure groups. The hematological depression was thought to be due entirely to gamma radiation, since it was not considered likely that the dose from internally absorbed radionuclides contributed to it significantly. The hematological depression was greater in the children, particularly those exposed at <5 years of age (see Table 5). The changes are enumerated below in more detail.

Table 4

Nausea and Vomiting in Rongelap Group[22]

		Incidence, %	
Age at exposure, yr	No.	Nausea	Vomiting
$\leqslant 5$	13	85	38
$\geqslant 6$	51	44	4

Table 5

Mean Blood Counts at Peak Depression in Rongelap Group[22]

	Percent of control	
	Age <5	Age >5
Neutrophils	56	64
Lymphocytes	25	55
Platelets	23	34

Lymphopenia to about half the level of the comparison Marshallese population was evident when the Rongelap people were first examined 3 days after exposure. In children <5 years of age the lymphocytes dropped to 25% of the level of the comparison children but showed a slight rise during the following weeks. The lymphocyte levels showed a slight increase by one year.

Neutrophil levels showed considerable fluctuation during the first month, possibly related to the prevalence of beta burns of the skin during that period. Neutrophil depression became evident by 5 to 6 weeks post exposure, the level dropping to about half the control level in adults and slightly lower in children aged <5 (Table 5). Neutrophils recovered more rapidly than lymphocytes and reached near control levels by one year.

Early *platelet* counts showed less fluctuation than other blood counts and decreased to about 30% of control levels by the fourth week. A spurt of recovery to about 75% of control levels occurred during the next few weeks, followed by slower recovery with mean levels never exceeding 90 to 95% of control levels during the first year. The platelet count is probably the most sensitive index of the severity of radiation exposure in the sublethal range.

Erythropoietic depression has not been a consistent finding, and radiation effects on these cells have not been demonstrable by peripheral blood counts.

Morphological changes. During the period when neutropenia was most severe, some people were found to have peculiar monocytic leukocytes that were thought to be large lymphocytes. These cells were not found subsequently, although an occasional binucleated lymphocyte was seen.

c. Clinical Findings. The development of significant depression of peripheral blood leukocytes and platelets during the first 6 weeks was cause for considerable apprehension among the medical

examiners. However, even at the nadir of depression (platelets, 28 to 30 days; granulocytes, 42 to 46 days) no associated bleeding tendency or increase in infections was seen. Eleven individuals had platelet counts between 35,000 and 65,000 but without evidence of bleeding. Between days 23 and 42 post exposure, 10% of the Rongelap group developed granulocytopenia of $\leqslant 1000$ cells and 42% had leukocyte counts $\leqslant 4000$ or absolute granulocyte counts <2500. During this period an epidemic of upper respiratory infections occurred involving more than half the exposed population. The illness in the higher exposure Rongelap group was no more severe than in the less exposed groups, and individuals in all groups were able to show an upward trend in leukocytes during the illness. Prophylactic treatment with antibiotics was not instituted because it was considered wiser to withhold them until a real need might arise. Antibiotics were given in certain cases where such therapy is ordinarily indicated.

2. Effects of Skin Exposure

Beta burns of the skin are described in detail in the original report of the medical findings.[2] The fallout was ubiquitous on Rongelap, Ailingnae, and Rongerik. The gritty white flakes clung to the moist skin, got into the hair, were inhaled, and were ingested with contaminated food and water. Areas of the skin covered by clothing were largely protected, and some who bathed in the lagoon and children who waded at the water's edge unknowingly were protecting themselves since they were later found to have fewer skin burns. However, the majority of the people on Rongelap and Ailingnae had widespread contamination of exposed surfaces of the body; the skin dose was probably in the thousands of rads and was due mostly to the beta component.

Most of the people complained of itching and burning of the skin for the first 24 to 48 hr, and some complained also of burning and lachrymation of the eyes. This may have been due partly to the caustic nature of the fallout. These symptoms were less evident in the American servicemen on Rongerik and were absent in the Utirik people. Table 6 shows the amount of fallout and the resulting skin burns for different groups.

Personnel decontamination commenced on board the Navy ships used for evacuation, where the people were given saltwater showers and clothing donated by the crew. On arrival at Kwajalein the skin was still contaminated, and decontamination procedures were continued for several days. Clothing was laundered repeatedly, and was discarded if its radiation level was not sufficiently reduced.

The first skin burns appeared about 12 to 14 days post exposure in the Rongelap people; burns appeared somewhat later in the Ailingnae and Rongerik groups and were less severe and extensive. About 90% of the Rongelap and Ailingnae groups and 40% of the Rongerik groups developed lesions, but none of the Utirik people. The lesions were multiple and spotty (Figure 5) and were characterized by superficial hyperpigmented macules, patches, or raised plaques, which desquamated within a few days leaving thin, pink epithelium (Figure 6). No erythema was observed before or during development of the lesions: it may have been lacking because of insufficient dose to dermis, or it may have been obscured by the darkness of the skin. Deeper burns with ulceration were noted in about 15% of the Rongelap people. Epilation (Figure 7) usually accompanied scalp lesions and was prevalent in $>90\%$ of the exposed Rongelap children (Table 7); it occurred to less degrees in adults and Ailingnae groups. Lesions were most common on the anterior neck, axillae, antecubital fossae, and feet, but even the anal region in some young children was involved. Lesions of the dorsum of the feet (Figure 8) were

Table 6

Skin Lesions[2]

Group	Composition	Fallout observed	Skin lesions and epilation
Rongelap	64 Marshallese	Heavy (snowlike)	Extensive
Ailingnae	18 Marshallese	Moderate (mistlike)	Less extensive
Rongerik	23 White Americans 5 Negro Americans	Moderate (mistlike)	Slight
Utirik	157 Marshallese	None	None

Figure 5. Extensive skin lesions in 13-year-old boy.[2]

Figure 6. Neck lesions at 28 days, showing wet desquamation.[2]

Figure 7. Epilation at 28 days in 7-year-old girl.[2]

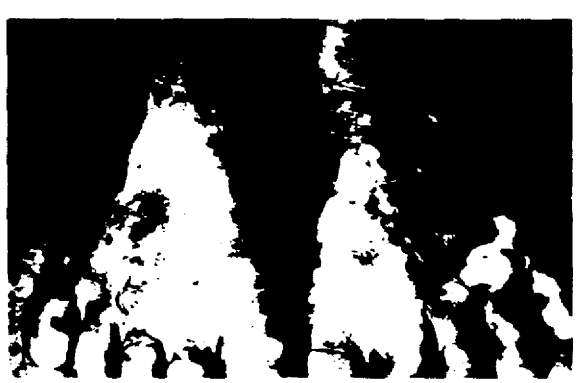


Figure 8. Foot lesions at 29 days, showing deep involvement between first and second toes.[2]

Figure 9. Early beta burns of skin ($\times 100$), showing extensive transepidermal damage, with milder damage to dermis showing cellular infiltration.[2]

Table 7

Epilation in Rongelap Group[22]

Severity of epilation	Incidence, %		
	Age 0 to 5 (13)	Age 6 to 15 (13)	Age >16 (38)
Slight (1+)	7.6	38.4	13.8
Moderate (2+)	38.6	30.7	5.5
Severe (3+)	53.8	23.0	8.3
Total	100.0	92.1	27.6

usually more severe, often with bullae formation; they made walking painful and in some cases involved secondary infections. Most of the lesions healed rapidly, with repigmentation.

Microscopic studies and numerous skin biopsies during the acute stages of the lesions revealed marked epidermal injury with considerable atrophy and flattening of rete pegs, cells with pleomorphic nuclei, pyknosis and cytoplasmic halos, and giant cells (see Figure 9). Cells laden with pigment were frequently present throughout the epidermis, probably accounting for the gross pigmentation of the lesions. Severe damage to the dermis and blood vessels was not observed.

An unusual observation was the development of transient bluish-brown pigmentation of the semilunar areas of the fingernails and toenails, first noted about 3 weeks post exposure in most of the Rongelap and Ailingnae people and seen, among the American group, only in Negroes. The cause of this pigmentation remains obscure.

Since no specific treatment is known for beta burns, the lesions were treated by cleansing and use of ointments and lotions for symptomatic re-

lief of itching and pain. Antibiotics were used in a few cases of secondary infections. The lesions healed within a few weeks with only minimal sequelae (described below).

These observations make it apparent that beta burns of the skin having acute symptoms associated with the lesions may be a serious consequence of fallout exposure. The Marshallese could have avoided many of the lesions if they had known to take the proper precautions such as protecting the skin from fallout and early cleansing. Had the whole-body radiation dose been greater, the reduction in immuno-hematological defenses might have resulted in more serious skin infections considerably complicating the skin symptoms.

3. Effects of Internally Absorbed Radionuclides

As pointed out in Section II. A., radiochemical urine analyses beginning 15 days after the accident indicated that the Rongelap people and to a lesser extent the other exposed groups had absorbed considerable amounts of radionuclides by inhalation and by ingestion of contaminated food and water[2] (see Table 3). Although serious late effects have developed due to radioiodine exposure to the thyroid, and near maximum permissible doses of several other radioisotopes were absorbed, no acute symptoms associated with absorbed isotopes appeared, the only exception being a possible role of the estimated 3 mCi of radioisotopes in the G.I. tract during the first two days in causing the early G.I. symptoms.

These observations indicate that lack of early symptoms associated with ingestion of radioactive material in an acute fallout situation does not preclude development of late effects from such ingestion.

III. Findings on Follow-Up Examinations (Late Effects)

During the first 10 years, after the early effects and before the development of thyroid abnormalities, few medical findings could be definitely associated with radiation exposure. The general health of the exposed Marshallese appeared to be about the same as that of the unexposed Marshallese comparison population, with the same types and incidence of diseases. The deaths that occurred were not related to radiation exposure. In the Rongelapese a few findings were considered to be connected with radiation exposure, such as residual skin effects; an increase in miscarriages and stillbirths during the first 5 years; suggestive evidence of a lag in complete recovery of peripheral blood elements; evidence of slight chromosome aberrations of the peripheral blood, and some degree of growth retardation in a few children. Other studies on possible late effects during this period were negative (cataracts, aging, mortality, longevity, malignancy).

During the second decade following exposure, effects developed in the Rongelap people which had not been heralded during the first decade. The most important were the widespread development of thyroid abnormalities, associated with growth retardation in some children. A fatal case of leukemia and a possible increased incidence of malignancies may or may not have been radiation related; the numbers are too small to permit any definite conclusions.

These findings are reviewed below.

A. GENERAL MEDICAL FINDINGS

The health situation in the Marshall Islands is in many respects improving. Most vector-borne diseases of the tropics do not occur, leprosy is rare, and tuberculosis is not a serious problem. Cardiovascular diseases are rare (except for diabetic gangrene of the feet).

The public and personal hygiene leave much to be desired. Skin infections (impetigo and fungus) and intestinal infestations (amoebae and worms) are endemic. In 1958, one year after their resettlement on Rongelap Atoll, stools were examined from 69 exposed and 112 unexposed people.[7]* Infestation was widespread with many individuals showing multiple types of parasites, the main pathogens being *Entamoeba histolytica* (found in 18.2%), hookworm (5.5%), *Trichuris trichiura* (34.3%), and *Giardia lamblia* (7.7%). Flagellates also included *Trichomonas hominis*. No significant differences were noted between the incidences in the exposed and unexposed populations. Recently *Ascaris* has been introduced into the Marshalls from other districts and infestation with it has become prevalent. Education and constant surveillance will be needed to overcome the parasite problem. Eosinophilia is common but is not always associated with helminthic infestations.

*These studies were done by Dr. M. Goldman and Mr. R.K. Carver, Communicable Disease Center, Chamblee, Ga.

With the growing dependence on imported foods and on money as the medium of exchange, cases of malnutrition are encountered. Night blindness due to vitamin A deficiency has been observed on several occasions, mainly in children.

The most serious epidemic during the 20 years covered by this report was the poliomyelitis epidemic in 1963. The "polio ward" at the Armer Ishoda Memorial Hospital in Majuro (which now operates as a physical rehabilitation center and orthopedic surgical ward) was created to care for the victims of this disaster. Rongelap Atoll was stricken in January/February of 1963 with 23 children and 3 adults taken ill. One of the adults died and two of the children had severe residual paresis.

Venereal disease appears to be on the increase.

Dental caries has been a notable finding during physical examinations of the Marshallese; it is attributed to poor oral hygiene, inadequate dental care, and possibly nutritional deficiency. Some of the younger generation are now showing the benefits of improved dental care. Dentists have participated in several surveys, and their general conclusion is that poor oral hygiene is showing its usual results: a high caries rate in teen-age children, severe periodontal lesions in adults (heavy calculus, loss of alveolar bone), and edentulous mouths in the aging. No significant difference between exposed and unexposed Marshallese has been seen except for a slightly greater incidence and severity of periodontal disease in the exposed group. It is not known whether this finding is related to radiation exposure.

Fish poisoning is common in the Marshall Islands. Symptoms are usually minor but occasionally severe or even fatal. Inflammation of mu-

Table 8

Physical Findings* in Rongelap and Utirik Adults, 1970–1974

	Rongelap exposed (68 examined)		Rongelap unexposed (135 examined)		Utirik exposed (117 examined)	
	No.	%	No.	%	No.	%
Acne	2	2.9	1	0.7	–	0
Anemia	3	4.4	1	0.7	–	0
Arteriosclerosis (mild)	7	10.2	2	1.5	8	6.8
Arteriosclerosis (mod.-sev.)	7	10.2	11	8.1	6	5.1
Asthma	–	0	2	1.5	2	1.7
Atrial fibrillation	1	1.5	2	1.5	1	0.8
Bradycardia	2	2.9	–	0	–	0
Cardiac enlargement	3	4.4	5	3.7	1	0.8
Cervical erosion	11	16.2	5	3.7	3	2.6
Cystourethro-rectocele	8	11.8	–	0	–	0
Deafness	2	2.9	3	2.2	–	0
Emphysema	–	0	–	0	1	0.8
Epilepsy	–	0	1	0.7	–	0
Hernia	2	2.9	2	1.5	2	1.7
Hypertension	8	11.8	16	11.9	17	14.5
Leprosy, arrested	1	1.5	–	0	–	0
Migraine	1	1.5	–	0	–	0
Obesity (gross)	7	10.2	20	14.8	14	12.0
Osteoarthritis	3	4.4	12	8.9	1	0.8
Pelvic inflammatory disease	–	0	1	0.7	–	0
Prostatic hypertrophy	3	4.4	1	0.7	–	0
Rheumatic heart disease	1	1.5	1	0.7	–	0
Rheumatoid arthritis	1	1.5	2	1.5	1	0.8
Spermatocele	1	1.5	2	1.5	–	0
Syphilis (?) (arrested)	2	2.9	4	2.9	–	0
Tumor, benign (except thyroid)	2	2.9	2	1.5	1	0.8
Tumor, malignant	1	1.5	1	0.7	–	0
Varicocele	–	0	2	1.5	–	0

*Findings on ophthalmology and diabetes are reported separately.

Table 9

Pregnancy Terminations (Abortions, Miscarriages, and Neonatal Deaths) Summarized by Five-Year Intervals, 1954-1973

	Terminations/total pregnancies							
	1954-1958		1959-1963		1964-1968		1969-1973	
	No.	%	No.	%	No.	%	No.	%
Exposed females	10/30	33.3	5/30	16.6	10/46	21.7	10/47	21.3
+ Exposed males	4/19	21.1	3/10	30.0	2/15	13.3	0/5	0
+ Unexposed males	6/11	54.5	2/20	10.0	8/31	25.8	10/42	23.8
Unexposed females	6/43	14.0	7/47	14.9	9/53	17.0	18/87	20.7
+ Exposed males	1/4	25.0	0/6	0	1/4	25.0	1/15	6.7
+ Unexposed males	5/39	12.8	7/41	17.1	8/48	16.3	17/72	23.6

Table 10

Adult Mortality, Rongelap

Exposed (Av. age at death: 64±5)				Unexposed (Av. age at death: 65±2.5)			
Year	Subject No.	Age & sex	Probable cause	Year	Subject No.	Age & sex	Probable cause
1956	25	46 M	Heart disease	1958	857	65 M	Cerebral thrombosis
1957	38	78 M	Heart disease, diabetes	1959	854	55 F	Infection in 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 of cervix (?)
1962	46	84 M	Heart disease	1962	953	48 M	Status asthmaticus
1962	26	21 M	Accident	1962	848	41 F	Neurosyphilis (?)
1962	56	75 F	Accident	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	964	90 M	Probably cardiovascular (?)
1966	29*	77 M	Asthma, heart failure	1967	967	24 M	Accident
1966	55	88 M	Heart disease	1967	936	76 F	Infection complicating diabetes
1966	13	71 F	Cancer of uterus**	1967	853	62 M	Diabetes
1968	59*	58 F	Influenza-pneumonia	1968	860	78 M	Congestive heart failure
1971	50*	51 M	Acute asphyxiation, cause unknown	1969	852	65 F	Hypertension, diabetes
				1970	884	76 M	
1972	54	19 M	Acute myelogenous leukemia	1970	916	46 F	Asthma, diabetes
1972	60	74 F	Heart disease	1970	1515	50 M	
1974	68	64 M	Cancer, stomach	1970	918	72 M	Heart disease, diabetes
				1970	875	53 M	? (epileptic)
				1971	899	76 M	
				1972	947	64 M	Hypertensive heart disease
				1972	957	64 F	
				1972	858	78 F	Heart disease (?)
				1972	961	79 M	Heart disease (?)
				1973	856	74 M	Hemiplegia, pneumonia
				1973	898	75 F	
				1973	1506	65 M	Asthma, heart disease
				1974	948	66 M	Heart disease, diabetes
				1974	908	84 F	Heart disease, diabetes

*Ailingnae group.
**Not confirmed by autopsy or biopsy.

cous membranes from improperly prepared arrowroot flour is not uncommon. Both have erroneously been associated with radiation effects in the minds of the Rongelap people.

Diabetes mellitus is a major problem and is discussed separately in Section III. G.

A program is under way to build new dispensaries on the outer islands. On Ebeye a new hospital building has provided improved conditions for health care.

It is against this general background that our examinations take place. We are privileged to get generous help from the local health authorities in our task, and conversely when we are on the scene we cannot avoid getting involved in problems outside the scope of fallout complications.

Major physical findings during the past 5 years are listed in Table 8.

B. VITAL STATISTICS: FERTILITY AND MORTALITY

The number of live births during the last 5-year period was 37 among the exposed and 69 among the unexposed. Calculated as live births per year per 1000 population these numbers give a birthrate of 112 for the exposed and 106 for the unexposed. In addition, there were 10 miscarriages among the exposed and 18 among the unexposed, so that, in both groups, one pregnancy out of five ended in miscarriage. This is approximately the same frequency as observed in the past (see Table 9).

Legal abortions are not performed in the Marshall Islands, and there is no reason to believe that any of the pregnancy terminations on record were provoked. Family planning has not been practiced in the past although it is slowly gaining ground. We therefore believe that the fertility and fetus viability indicated by these statistics are the natural ones, and, although the exposed females showed an apparent increase in miscarriages during the first 5 years, there is no evidence suggesting that the history of exposure to radiation has had any permanent effect on either.

The people who have died are listed by year in Table 10, with their age and probable cause of death (such death certificates as are available are not always specific). The overall mortality rate for the 20-year period is ~12 per 1000 per year for the exposed Rongelap group and ~13 for the unexposed; for the last 5-year period the rates are ~9 and ~21 respectively. These differences are not statistically significant; in such small groups

Table 11

Mortality, Utirik
(Av. age at death: 61±3)

Year	Subject No.	Age & sex	Year	Subject No.	Age & sex
1956	2118	24 M	1965	2183	67 M
1957	2184	63 M	1965	2204	71 F
1957	2219	57 F	1965	2238	65 F
1957	2222	63 F	1965	2253	56 M
1958	2243	50 M	1967	2181	78 M
1959	2122	87 M	1967	2202	72 F
1959	2127	73 M	1967	2223	79 F
1959	2170	46 M	1968	2101	62 M
1959	2187	61 F	1968	2112	70 M
1960	2116	27 F	1968	2141	67 F
1960	2131	35 F	1968	2259	36 F
1960	2180	76 M	1969	2191	90 F
1961	2177	11 M	1969	2214	80 M
1961	2199	49 F	1970	2175	73 M
1963	2203	71 F	1970	2211	65 M
1964	2163	75 M	1971	2258	64 M
1964	2190	85 F	1971	2246	25 F
1964	2192	84 F	1972	2178	37 M
1965	2121	68 M	1972	2252	57 M
1965	2154	51 F	1973	2186	67 F
			1974	2201	68 F

observed over such short periods, differences this large or larger could occur by chance ($p<0.05$). Even so, had the trend been in the opposite direction, we would have had reason to be concerned; as an example, we are keeping a careful watch on the apparent increase in malignancies (see Section V).

The mortality rate among the exposed Utirik people was ~13 for the 20-year period and ~14 for the last 5 years. The deaths are listed in Table 11, but causes are not given because of insufficient data.

Recent misconceptions make it necessary to clarify comparisons with district-wide statistics. The vital statistics of the Trust Territory have improved greatly over the 20 years covered by these reports, but they are still not published in sufficient detail to permit valid comparisons with ours. The age distributions are too different: district-wide the median age is 16 years, whereas in our group it is between 30 and 40. The difference between the mortality in the general population (~7 per 1000 per year) and that in our groups (~13) reflects this difference in age distribution and is not related to the history of exposure. A similar effect can be seen on the birthrate (~40 per 1000 per year district-wide and ~110 in our groups).

C. HEMATOLOGICAL FINDINGS

Since blood-forming cells are among the most radiosensitive cells in the body, hematological examinations have been emphasized in both exposed and unexposed populations. Evidence of such sensitivity in the exposed Rongelap people was provided by the early acute depression of leukocytes and platelets and by possible residual effects on hemopoiesis indicated by a lag in recovery of blood elements to the levels of the unexposed population. The development of a fatal case of leukemia may also be evidence of residual effect.

1. Procedures

Until 1972 yearly measurements were made of peripheral blood elements, including WBC, differential white counts, RBC and hematocrit and/or hemoglobin, and platelet counts. Since a case of leukemia was found in 1972, such measurements have been made twice a year. The counts were done under a microscope until electronic means came into use (Coulter counter and General Science Haema-Count MK 3 and MK 4 instruments). Reticulocyte and bone marrow examinations have been done on several occasions by standard procedures.*

A number of special hematological examinations over the years have included tests for leukemia (i.e., special scanning of blood smears for immature forms, basophil counts in 4000 cells, alkaline phosphatase staining of neutrophils,** analysis for Australia antigens),† studies of red cell and plasma volume, chromosome analysis, etc. Plasma and red cell samples have been brought back for special studies in laboratories in the United States. These studies are further discussed in later sections, where relevant.

2. Recovery of Hemopoiesis

In Figures 10 to 12 the mean blood counts of the combined (Rongelap and Ailingnae) exposed group are compared with those of the unexposed group over the 20-year period. Data are listed in Table 12 for the past 5 years, and in Appendix 6 for the entire 20 years. Figure 13 shows the cu-

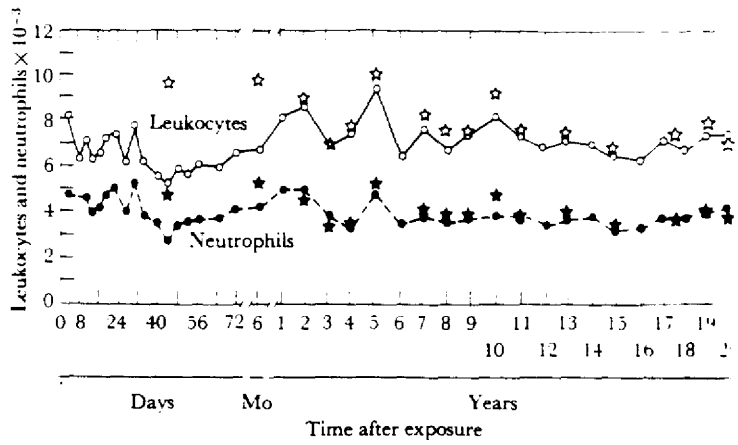


Figure 10. Mean neutrophil and white cell counts of Rongelap people from the time of exposure through 20 years post exposure. Stars represent mean values of comparison population.

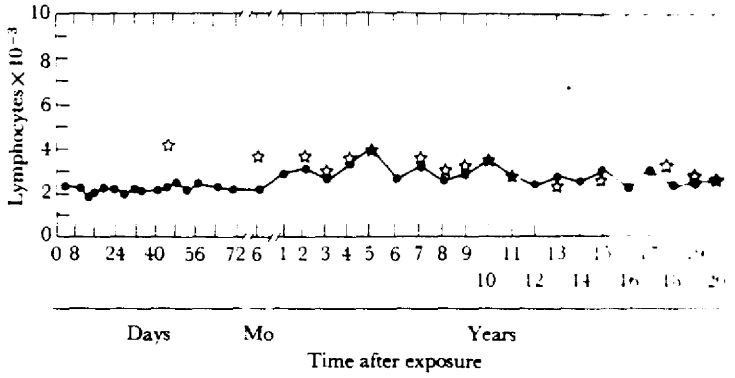


Figure 11. Mean lymphocyte counts of Rongelap people from time of exposure through 20 years post exposure. Stars represent mean values of comparison population.

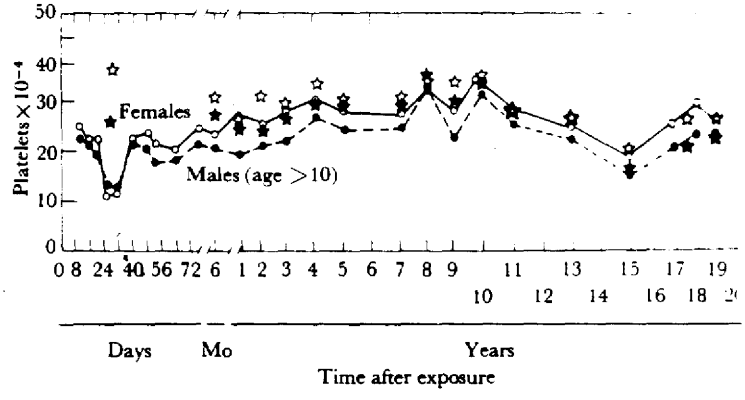


Figure 12. Mean platelet counts of Rongelap people from time of exposure through 20 years post exposure. Stars represent mean counts of unexposed comparison populations.

*Mr. N. McDaniel of SUNY at Stony Brook has done the differential counts for the past several years.

**Dr. W. Moloney and Mrs. L. Tullin at Harvard Medical School did the alkaline phosphatase staining of neutrophils in peripheral blood smears.

†Dr. B.S. Blumberg, Institute for Cancer Research, Philadelphia, did the studies for Australia antigens.

Table 12

Summary of Hematological Findings by Population, 1970-1974
(Mean ± standard deviation)

Group	Year	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
Rongelap	1970		6.40±1.34	3.50±1.34	2.41±0.81	0.13	0.36	0.22	40.5±4.7			7.4±0.6
Ailingnae	1970		6.44±1.20	4.00±1.35	1.98±0.66	0.15	0.24	0.35	38.8±5.5			7.5±0.6
Rongelap	1971	220±63	7.34±1.72	3.86±1.11	3.09±0.89	0.10	0.22	0.19	41.0±4.6	411±55		7.1±0.7
Ailingnae	1971	246±81	6.41±1.51	3.46±1.15	3.00±0.87	0.10	0.17	0.16	38.7±6.5	399±46		7.1±1.0
Unexposed	1971	233±69	7.21±1.70	3.78±0.98	3.18±0.91	0.16	0.24	0.22	40.4±6.2	402±73		7.5±0.5
Rongelap	1972	272±97	6.69±1.90	3.94±1.66	2.34±0.87	0.19	0.33	0.33	40.7±6.3	429±56		7.6±0.5
Ailingnae	1972	258±77	7.50±1.53	4.62±1.53	2.18±0.41	0.28	0.35	0.59	37.8±5.0	448±54		7.5±0.6
Unexposed	1972	267±86	8.09±1.50	4.38±1.34	3.03±0.93	0.26	0.39	0.25	42.0±5.5	445±54		7.4±0.7
Utirik	1972	280±76	7.77±2.00	4.33±1.72	2.48±0.56	0.36	0.56	0.34	40.0±3.8	435±40		7.8±0.5
Rongelap	Mar 73	247±81	7.43±1.41	3.83±1.15	2.62±0.81	0.38	0.54	0.52	40.7±5.8	445±61		7.4±0.4
Ailingnae	Mar 73	247±56	7.08±1.74	3.72±1.02	2.27±0.79	0.36	0.65	0.78	38.4±5.6	425±67		7.5±0.4
Unexposed	Mar 73	253±72	7.91±1.73	4.25±1.62	2.69±0.84	0.40	0.53	0.60	39.5±6.2	440±58		7.4±0.9
Utirik	Mar 73	281±67	9.41±1.97	5.63±1.87	2.69±0.65	0.54	0.66	0.70	41.7±4.5	462±58		7.7±0.6
Rongelap	Sep 73	264±77	7.62±1.84	4.17±1.60	2.51±0.76	0.39	0.48	0.34	38.9±6.4	454±78	13.6±2.1	
Ailingnae	Sep 73	257±50	7.05±2.14	3.92±1.53	2.21±0.39	0.30	0.56	0.62	39.9±4.2	467±44	14.0±1.1	
Unexposed	Sep 73	262±63	7.17±1.51	4.08±1.34	2.31±0.75	0.35	0.41	0.50	38.5±5.3	486±73		
Rongelap	Mar 74	256±69	7.45±1.67	4.25±1.44	2.42±0.62	0.41	0.41	0.31	38.4±6.0	507±63	13.3±2.9	7.8±0.7
Ailingnae	Mar 74	283±70	7.73±2.67	4.60±1.91	2.05±0.61	0.47	0.58	0.28	38.2±6.1	499±114	13.9±1.3	8.3±0.4
Unexposed	Mar 74	245±63	7.46±1.59	4.06±1.31	2.59±0.93	0.43	0.48	0.43	39.1±7.6	512±72	13.6±1.8	7.6±0.7
Rongelap	Sep 74	193±56	8.32±1.69	4.28±1.45	2.86±0.79	0.42	0.60	0.67	45.2±6.4	511±72	13.7±1.7	
Ailingnae	Sep 74	186±37	6.57±2.40	3.41±1.66	2.36±1.09	0.39	0.38	0.35	44.5±7.2	478±87	14.3±1.6	

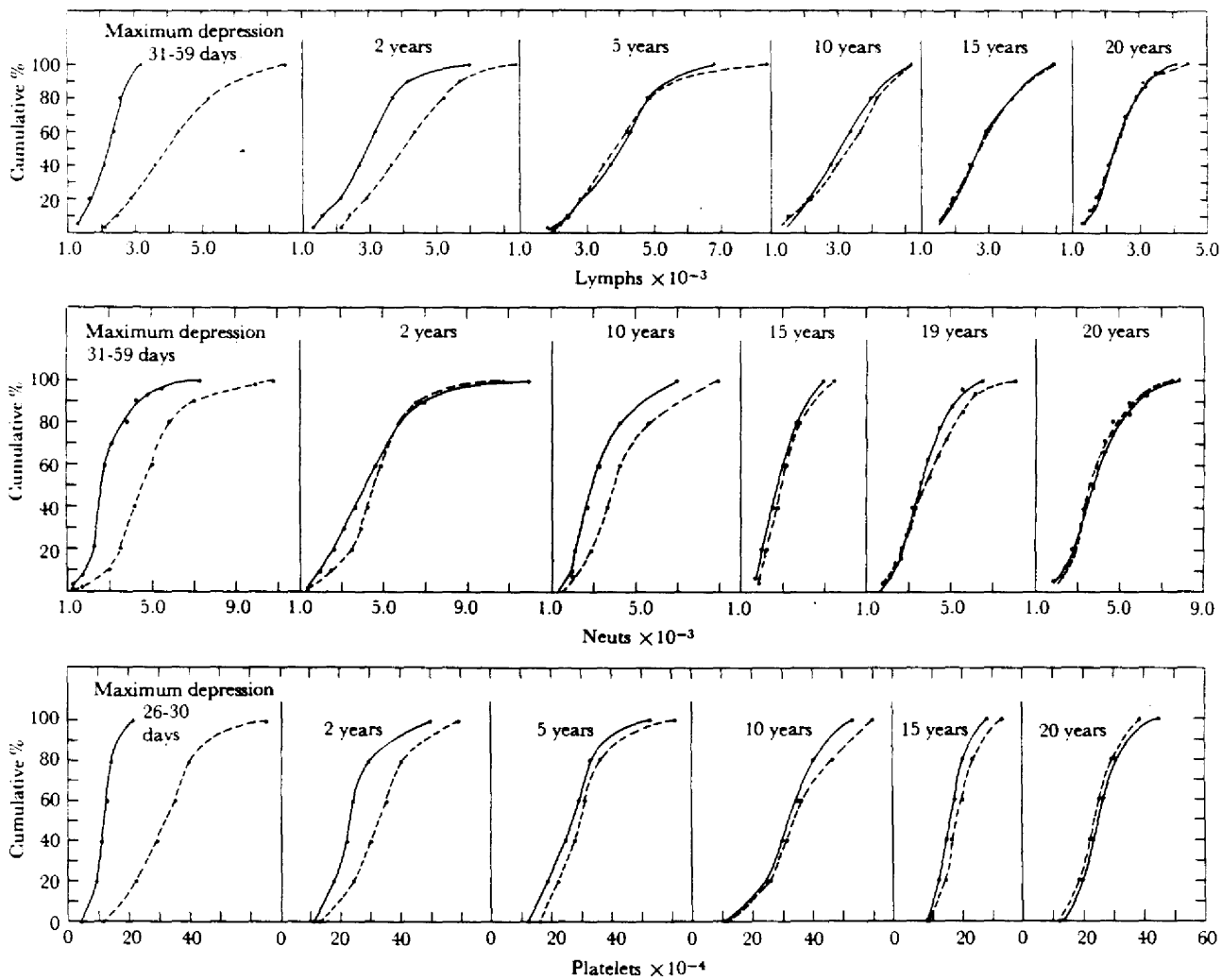


Figure 13. Cumulative percent distribution curves for blood elements of Rongelap people at various times (—, exposed; ---, unexposed).

Figure 14. Bizarre mitosis in a myelocyte.

Figure 15. Binucleated normoblast.

mulative percentage of neutrophils, lymphocytes, and platelets at various times. From these data it appears that hemopoietic recovery in the exposed groups was incomplete during the first 15 years after exposure. Since that time the levels in the exposed group have been about the same as in the unexposed group. On the basis of RBC, hematocrit, and hemoglobin determinations, erythropoietic function has been about the same in the exposed and in the comparison population. The lag in recovery of leukocytes and platelets may represent residual bone marrow injury. The results of bone marrow and chromosome studies and the development of a case of leukemia tend to support this contention.

3. Bone Marrow

Although bone marrow examinations at 6 months post exposure showed no gross abnormalities, smears on four exposed Rongelap people 10 years later showed alteration in the myeloid-erythroid ratio in three of them, manifested by an increased number of red cell precursors. In addition to hyperplasia, the findings included abnormalities of chromatin material with double nuclei and an increased number of mitotic figures in the normoblastic series (see Figures 14 and 15).[11] Occasionally, bilobed lymphocytes have been noted in the peripheral blood of some exposed people.

4. Other Hematological Findings

Total blood volume and red cell volume were studied 10 years after exposure.[11]* No differences were found between exposed and unexposed Rongelap groups, but it was noted that blood volume and red cell volume tended to be reduced in many Marshallese compared with Americans[29] (see Figure 16).[12] Several other hematological observations, not related to radiation exposure, were noted. Eosinophilia >5% in more than half the people has been a consistent finding. This could be accounted for only partly on the basis of intestinal parasitism and may be related also to numerous fungus infections of the skin and other chronic infections. Other findings possibly related to chronic infections are above-normal sedimentation rates and high gamma globulin levels (both tending to increase with age). Varying degrees of anemia have been seen occasionally, particularly in women of childbearing age. Price Jones sizing of red cells in these cases showed a slight microcytic tendency.[8] Iron deficiency apparently was not the cause since serum iron usually was in the normal range.[8] Reticulocyte counts have not been significantly increased in either the exposed or unexposed population. Macrocytic anemia has not been seen. Vitamin B_{12} levels have been unusually high in the Marshallese[8]; the cause of this is unknown but does not appear to be related to liver disease or leukemia. The sickling trait has not been seen in the Marshallese examined.

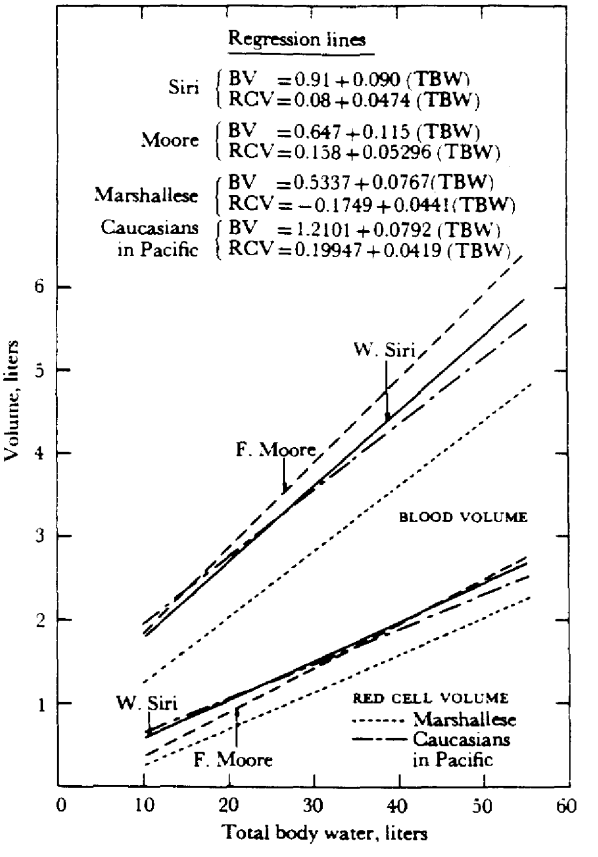


Figure 16.

D. SUBSEQUENT COURSE OF SKIN LESIONS

The residual skin lesions noted in the Rongelap and Ailingnae people since the initial lesions are listed in Table 13. During the first year many of the healed areas, particularly on the back of the neck, showed a roughening (rugosity) and bluish-black pigmentation, which became less noticeable with time (see Figures 17 and 18). Deeper lesions exhibited early residual scarring and lack of pig-

*These studies were done by Dr. L.M. Meyer, Veterans Administration Hospital, Brooklyn, N.Y., and Dr. W.E. Siri, University of California at Berkeley.

Table 13

Residual Beta Burns

Subject No. & sex		Subject No. & sex	
2 M	Roughening and pigment variation on front of neck. Several pigmented macules ACF.* Perianal depigmentation.	39 F	Slight roughening and pigmentation back of neck; pigment variations and slight hyperpigmentation dorsum right foot.
3 M	Mottled pigmentation both axillae. Pigmented area behind left ear.	49 F	Numerous pigmented macules both sides of neck and a few on arms and ACF.
11 M	Pigment changes left ACF, dorsum first right toe; pigmented nevi axilla.	54 M	Mottled pigmentation and depigmentation on front of neck. Died 1972.
17 F	Scarring and pigmentation left ACF.	59 F	Mottled pigmentation and depigmentation on back of neck. Died 1968.
20 M	Pigmented patch back of neck.	63 F	Slight rugosity and pigmented ridges on back of neck.
23 M	Pigmented macules left axilla, front of neck and chest. Depigmented spots shaft penis.	64 F	Mole back of neck; slight pigment variation and a few macules front of neck. Mole back of neck.
24 F	Slight pigment variation on front of neck; several pigmented macules dorsum left foot.	65 F	Pigment variation and roughening front of neck. Not apparent now.
25 M	Scarring dorsum left foot. Biopsy at 2 years hyalinization of connective tissue and thickening of blood vessel walls of cutis. Died 1956.	67 F	Depigmented scars dorsum left foot.
26 M	Scarring and depigmentation of dorsum right foot. Died 1962.	75 F	Slight pigmented area dorsum right first toe.
34 F	Slight roughening and pigmentation back of neck. Moles on front of neck.	78 F	Numerous pedunculated moles on sides and front of neck.
		79 M	Pigmented and depigmented scar posterior surface left ear.

*ACF = antecubital fossa.

mentation, but the scars have faded and repigmentation has tended to occur in most cases. No evidence has been seen for the development of chronic radiation dermatitis or premalignant or malignant changes in any case, and most people now show little residual evidence of lesions. Hair of normal color and texture regrew by 6 to 12 months in all cases except for one man who still has spotty epilation on the back of his head.

Follow-up skin biopsies of residual lesions, studied at 6 months[3] and at 3 and 10 years[6,11] post exposure, histologically showed excellent healing, but in most cases residual evidence of exposure was noted, such as epidermal atrophy and thickening of the corium with collagen bands, although little evidence of significant vascular changes was seen in the corium (see Figure 19).

Though no malignant changes in the skin have thus far been noted in the Marshallese, the development of skin cancer due to radiation exposure is still a distinct possibility, since its latent period may be as long as 48 years and may, on the average, be longer than for some other types of radiation-induced malignancy. Many of the younger exposed Marshallese still have a long life expectancy.

There are several reasons, however, why skin cancer may not occur in the Marshallese. The low average energy of the beta rays resulted in much less damage to the dermis than to the epidermis. This would make the prognosis favorable if, as many investigators believe, the development of skin cancer requires sufficient damage to the dermis to impair nutrition of the epidermal cells. In the Marshallese the superficial nature of the skin burns, rapid healing, and absence of chronic radiation dermatitis are factors against the development of skin cancer[30]; nevertheless, persisting cellular changes are seen in their residual skin lesions. If, as Teloh et al.[31] believe, skin cancer results from direct irradiation of the epidermis without necessarily severe dermal injury, its occurrence would be more likely. The appearance of nevi in

Figure 17. Persisting depigmented areas, 6 months after exposure, in areas of most severe lesions.[2]

Figure 18. Ear lesion healed, 6 months after exposure, with considerable scarring.[2]

Figure 19. Section of skin from beta-burn area on back of neck of 56-year-old woman (No. 34) at 10 years after exposure (100×). Note atrophy of epidermis with narrowing of stratum granulosum and fingerlike projections of rete pegs. Slight atrophy of the sweat gland ducts is also present.[11]

Figure 20. Pigmented nevuslike lesions in previous beta-burn area of neck (subject No. 78).[11]

the neck regions that sustained severe beta burns suggests that the development of benign lesions may have been enhanced by the skin exposure (see Figure 20). Possible cocarcinogenic factors such as the ultraviolet radiation in tropical sunlight and skin irritation from coral, trauma, and infections may render the development of malignant changes in the skin more likely, but the dark pigmentation should offer some protection, as it has been shown that skin cancer is less prevalent among Negroes.

E. OPHTHALMOLOGICAL FINDINGS

Routine ophthalmological examinations (including funduscopic) have been included in the regular physical examinations of both the exposed and unexposed populations. In addition, special examinations for possible effects of radiation on the eyes have been done at intervals by ophthalmologists accompanying the medical team, first in May 1954 (2 months after the accident) and then in 1955, 1956, 1957, 1958, 1959, 1962, 1964, 1967, 1971, and 1972. The routine examinations include testing of vision, accommodation, muscle balance, and pupillary reactions; external examination; and funduscopy. Vision is tested with the Snellen chart and the E-chart when necessary. Intraocular tension has been measured when indicated, and color vision has been tested on several occasions. Special examinations have included slit-lamp and fundus photography. As part of the aging studies quantitative changes in certain eye characteristics

(visual acuity, arcus senilis, accommodation, retinal arteriosclerosis) have been recorded.[8,13,32]

1. General Findings

Some general observations on the eyes of the Marshallese have indicated differences from the eyes of U.S. Caucasians which may be related to ethnic background and possibly to environmental factors. The major eye findings for the past 5 years are listed in Table 14.

The high incidence of pingueculae and pterygia is the most notable finding listed in the table. This has been found by others to be characteristic of people living on low coral atolls in the Pacific and is thought to be related to irritation of the eyes by coral dust, salt water, and bright sunlight. The other major findings listed were related to general aging effects (loss of visual acuity, cataract formation, loss of accommodation, loss of ocular muscle balance, etc.) and these appeared to occur to a lesser degree in the Marshallese than in Americans, although the incidence of arcus senilis and cataracts seemed higher. The Marshallese had less myopia, aberrations of color vision, and ambly-

Table 14

Ophthalmological Findings, 1970-1974

Ocular condition	Rongelap and Ailingnae exposed (70*)		Rongelap unexposed (133*)		Utirik exposed (100*)	
	No.	%	No.	%	No.	%
Anisocoria	1	1.4	0	0	0	0
Arcus senilis	22	31.5	34	25.5	21	21
Argyll-Robertson pupil	1	1.4	0	0	0	0
Chalazion	3	4.3	1	0.75	0	0
Choroiditis	2	3.0	2	1.50	1	1
Congenital defects	2	3.0	0	0	0	0
Conjunctivitis	0	0	2	1.50	0	0
Corneal pigment	0	0	0	0	0	0
Corneal scar	2	3.0	5	3.75	1	1
Drüsen	0	0	2	1.50	0	0
Enophthalmos	0	0	0	0	1	1
Enucleation	1	1.4	0	0	0	0
Glaucoma, primary	0	0	0	0	0	0
Glaucoma, secondary	0	0	1	0.75	0	0
Lens:						
Cataracts	12	17.0	26	19.5	20	20
Lenticular opacities	9	13.0	14	10.5	17	17
Polychromatic sheen	7	10.0	13	10.0	17	17
Macular degeneration	2	3.0	1	0.75	1	1
Night vision impairment	0	0	0	0	0	0
Nystagmus	1	1.4	2	1.5	0	0
Phthisis bulbi	1	1.4	0	0	0	0
Pinguecula	7	10.0	6	4.5	7	7
Positive Romberg	1	1.4	0	0	0	0
Proptosis	1	1.4	1	0.75	0	0
Pterygium	17	24.0	41	31.0	29	29
Ptosis	2	3.0	0	0	0	0
Retinal arteriosclerosis	11	15.7	19	14.0	1	1
Retinal hemorrhages	2	3.0	0	0	0	0
Retinal scars	2	3.0	2	1.5	3	3
Retinopathy, diabetic	0	0	0	0	2	2
Strabismus	2	3.0	2	1.5	3	3
Uveitis	1	1.4	0	0	0	0
Vitreous opacities	2	3.0	1	0.75	1	1
Xanthoma	1	1.4	0	0	0	0

*Number of people examined.

opia. Also notable was their lack of retinoblastomas, malignant melanomas of the choroid, and basal cell carcinoma of the eyelids. Ocular pathology caused by herpes simplex or zoster viruses was not seen. No cases of retinal detachment occurred except in two people as a sequela of ocular surgery.

Corneal scars, macular degeneration, vitreous floaters, and other degenerative intraocular conditions were seen in about the numbers expected in any part of the world. No *Toxocara* infestation or other intraocular parasites have been encountered, and no ocular involvement by the parasites *Onchocerca* or filaria has been seen. In the one case of inactive leprosy on Rongelap no ocular lesions have been noted. Some people have residual scars of yaws, but no active lesions have been seen during the 20-year period.

Most of the congenital malformations and familial ocular diseases found in Americans were not seen in the Marshallese, such as ocular dystrophias, retinitis pigmentosa (congenital night blindness), congenital cataracts or glaucoma in the young or familial glaucoma in older people, and coloboma of the iris or posterior segment of the eye. On the other hand, certain congenital traits apparently peculiar to this race were noted. The average size of the corneas is greater than in the U.S., more than 60% of the people examined having corneas >14 mm in diameter. In $\sim 5\%$ of the people the retinal vessels showed increased tortuosity (corkscrew-like) and dilatation. This phenomenon has sometimes been found associated with the sickle cell trait, but the latter has not been noted in the Marshallese.

Dietetic and hygienic conditions in the Marshall Islands have improved over the 20 years of this study. In earlier years, poor night vision in children due to vitamin A deficiency was occasionally found, and molluscum contagiosum was seen more frequently; also lesions involving the skin of the eyelids, forehead, and face. It has been several years since night vision impairment or molluscum has been seen.

2. Possible Radiation Effects

Both the detonation in March 1954 and the resulting fallout gave rise to several types of radiation that might cause injury to the eyes.

Longwave radiation. Direct observation with the naked eye of the fireball of an atomic bomb detonation is known to produce retinal damage with scarring and possible blindness.[33,34] The Bravo fireball was observed by a small number of people on Rongelap, and, though its distance from them precluded thermal injury to the skin from longwave (visible and infrared) light, injury to the retina must be considered. The fireball was described by the Rongelap people as appearing "like the sun rising in the west." They probably did not see it at its greatest luminescence, when the damaging ocular effects occur, because this comes a fraction of a microsecond after detonation, so that at a distance of 100 miles the fireball would not yet have risen above the horizon. Also, at that distance the size of the image on the retina would be small. These factors probably explain why no one complained of blindness from seeing the fireball and no retinal scars have been found in any of the people.

Ionizing radiation. The fallout on Rongelap resulted in a gamma dose of 175 rads to the whole body including the eyes. Beta rays from fallout deposited on the ground and contaminating the eyes resulted in some superficial exposure of the lids, conjunctiva, and cornea, but their low penetration makes it unlikely that the lens or retina received any significant dose from this source. During the two days before evacuation, many of the people complained of some itching and burning of the eyes with some lachrymation. These symptoms may have been related to radiation from fallout in the eyes, but physical and chemical irritation by the caustic fallout dust may have played an important part. Internal absorption of radionuclides probably did not contribute to the radiation dose to the eyes.

Conjunctivitis. The burning and lachrymation disappeared by the time the people were evacuated to Kwajalein Island. However, at the time of development of beta burns, about 2 weeks after exposure, two men developed conjunctivitis and inflammation of the eyelids which may have been due to beta irradiation. These lesions healed within several days.

Corneal pigmentation. Beginning at about 2 months post exposure, in some people a pigmented line was noted in the cornea at the limbus in the horizontal axis of the palpebral fissure, which appeared to be below the surface of the epithelium. It varied from 0.5 to 2 mm in length. This may have been due to beta radiation and was possibly related to the bluish pigmentation noted in the nail beds. The latter disappeared when the nail bed grew out several months later, but the corneal

pigmentation persisted for several years before it finally disappeared. These pigmented streaks are not to be confused with conjunctival and corneal pigmentation of genetic origin noted in some of these people, which is in the superficial epithelium and is permanent.

Pterygia and pingueculae. During the early surveys the incidence of pterygia and pingueculae was thought to be greater in the exposed Rongelap people than in the unexposed comparison population. However, as the latter group has increased, this no longer appears to be the case (see Table 14).

Opacities of the lens. Opacities of the lens have been a common finding in the Marshallese (see Table 14). They are scored in order of decreasing size as cataracts (arbitrarily considered as a lens opacity impairing visual acuity by $\geqslant 20/30$), lens opacities, polychromatic sheens, and flecks. Special microscopic examinations for lens flecks were made in 1969.[13]

Irradiation of the lens in sufficient dosages may result in a spectrum of opacities ranging from polychromatic sheens to full-blown cataracts.[35-38] Only in the early stages of cataract formation can changes characteristic of radiation generally be recognized; more mature cataracts due to radiation usually cannot be differentiated from those due to aging or disease. The development of lens opacities due to radiation depends on the characteristics of the radiation, the age of the individual at exposure, and the interval after irradiation. Neutrons are known to be several times as cataractogenic as gamma or beta rays and played an important role in cataract development in the Japanese exposed to the atomic bombs and also in cyclotron workers.[37-39] In the Marshallese only gamma radiation need be considered because no neutrons were involved in the fallout and the beta radiation did not have sufficient energy to impart a significant dose to the lens. The gamma radiation had a fairly energetic spectrum (100 to 1500 keV). The 175-rad dose was delivered at a decreasing rate over the two days of exposure. The minimum cataractogenic dose of x rays or gamma rays is considered to be $\sim$200 rads and the dose to produce progressive lesions, $\sim$500 rads.[40] The latent period averages about 2 to 3 years. With larger doses the latent period may be shorter, and with lower doses it may be $\geqslant 8$ years.

The smallest lens opacities observed with slit-lamp microscopy were lens flecks, which are thought to be defects in single lens fibers. These discrete opacities were at most only a few microns in size and could in no way impair vision. The number of flecks increased with age in all groups, but they developed more rapidly in adolescent females.[13]* A higher score in females aged 13 to 20 at exposure may have been related to the radiation. The higher incidence of flecks in adolescent females was thought to be associated with their high estrogen levels.

Other lens opacities observed in the exposed Marshallese did not fit the descriptions of radiation-induced types and were similar to those seen in the unexposed population. The absence of radiation-induced cataracts is not unexpected, since the dose to the Marshallese was below the minimum dose of x rays or gamma rays needed. The lapse of 20 years is well beyond the usual latent period and it seems unlikely that any such lesions will develop. All the cataracts seen have been of the senile or pre-senile type and no juvenile cataracts have been noted. Possible correlation with diabetes is discussed in Section III. G. 6. The slightly higher incidence of cataracts in the exposed Rongelap people over the years may be related to a slight preponderance of older people in the exposed population.

Polychromatic sheens, yellowish or "beaten brass" to blue-green in color, were noted in some Marshallese, both exposed and nonexposed. These were not associated with any lenticular opacities characteristic of radiation exposure.

F. GROWTH AND DEVELOPMENT STUDIES OF EXPOSED CHILDREN

1. Data Taken

During each medical survey of the Marshallese people, systematic pediatric examinations have been conducted on subjects under the chronological age of 20 years (see Table 15) with the exception of adolescent girls who were pregnant or who had had babies. The studies consisted of a brief interval history, routine physical examination, palpation of the thyroid gland, and assessment of growth and development.

The growth status of the children exposed to fallout has been followed regularly since the initial examination. From 1954 to 1958 the growth data consisted of routine measurements of stature and weight. In 1959, roentgenographic evaluation of

*These studies were done by Dr. J. Bateman, BNL.

Table 15

Composition of Pediatric Groups, 1972

Age at exposure, yr	No. males	No. females
Rongelap group		
<10	10	9
10-19	5	9
In utero	3	1
Ailingnae group		
<10	2	4
Unexposed group		
Born 1945-1954	22	19
Born 1935-1944	11	7

skeletal development (left hand and wrist) was initiated,[41] and systematic documentation of anthropometric data was started. The documentation was continued during the succeeding examinations on subjects considered to be in the growth phase (generally through age 20). The data recorded regularly included standing height (stature), body weight, head circumference, head width, head length, chest circumference, biacromial width, and calf circumference, and sporadically included sitting height, chest width, chest depth, and buttocks circumference. (See Appendix 7.)

The development of secondary sex characteristics was qualitatively assessed according to the standard criteria generally accepted in growth studies (Greulich et al.,[42] Shuttleworth[43]). Such procedures included the staging of breast development in girls; estimation of the type, pattern, and amount of body hair (facial, pubic, axillary, etc.), and staging of the growth of penis and testes in boys. During several examinations, photographic documentation was done as part of the assessment of growth status and physique.

The roentgenographic evaluation of skeletal development was done by the inspectional technique of Greulich and Pyle,[41] and the American standards were used for comparison.

2. Physical Findings

The early post-exposure examinations indicated that the younger children, in general, manifested more extensive and more severe radiation injury than the older children and adults. Following recovery from the acute effects of radiation, the overall physical status of the children appeared to be normal except for the subsequent development of thyroid abnormalities, which are discussed below in Section IV. The other physical findings, which have been carefully tabulated in the individual reports for each survey, indicate no definite pattern of abnormalities that would differentiate the exposed children from the unexposed comparison children. The usual spectrum of infectious and noninfectious diseases, congenital anomalies, and physiological variations has been noted in both exposed and unexposed children.

3. Growth Status

Interpretation of the anthropometric data and assessment of the growth status of the exposed Rongelap children have been complicated by (a) radiation injury to the thyroid gland, (b) partial or total thyroidectomies in the children who developed thyroid neoplasia, and (c) the administration of thyroid hormone to the entire exposed Rongelap population since September 1965. If the data on the three children (Nos. 3, 5, and 65) who were obviously hypothyroid and had markedly small body measurements are excluded, comparisons of mean stature and skeletal age indicated no statistically significant difference between the exposed and unexposed children for either boys or girls.

Compared with American norms, the mean skeletal ages at given chronological ages for each group were, for unexposed girls, quite similar, but for unexposed boys, they appeared to be delayed by 6 to 7 months at each chronological age.

Earlier preliminary comparisons had suggested that boys exposed at age <5 years were retarded in statural growth compared with unexposed boys. The current analysis excludes those with a specific diagnosis of hypothyroidism. The individual growth and development curves for 13 children exposed at age <5 and their controls are shown in Figures 21 and 22. The effect of thyroid treatment on the two boys (Nos. 3 and 5) is evident. Several other children (Nos. 2, 19, 65, 42) had a tendency toward retarded development, which may have been improved by thyroid medication.

On the 4 children exposed *in utero*, careful examinations have resulted in no unusual physical findings. The growth progress of 3 of them has been similar to that of age peers (see Table 16). One boy (No. 85) has had a head circumference smaller than the average for unexposed males and a slightly retarded pattern of skeletal maturation, but his statural growth curve was comparable with that of unexposed males.

Final evaluation of stature and skeletal age will be postponed until all the exposed children have reached the age of 21 to ensure that all are fully mature.

Height and weight data on survivors of the atomic explosions at Hiroshima and Nagasaki have been recently reexamined by the Atomic Bomb Casualty Commission.[44] Analysis of the final

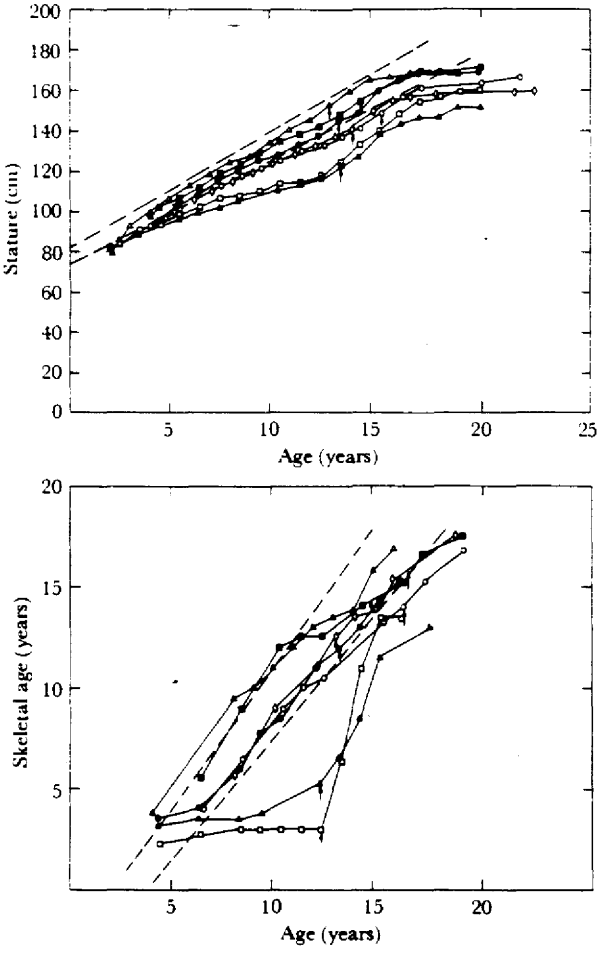


Figure 21. Statural growth and skeletal growth data for boys exposed at age <5 years. Dashed lines show minimum to maximum spread for unexposed peer group. Arrow indicates start of thyroxine treatment. Subjects: ●, No. 2; ◇, No. 19; ■, No. 23; ○, No. 32; △, No. 54; □, No. 3; ▲, No. 5.

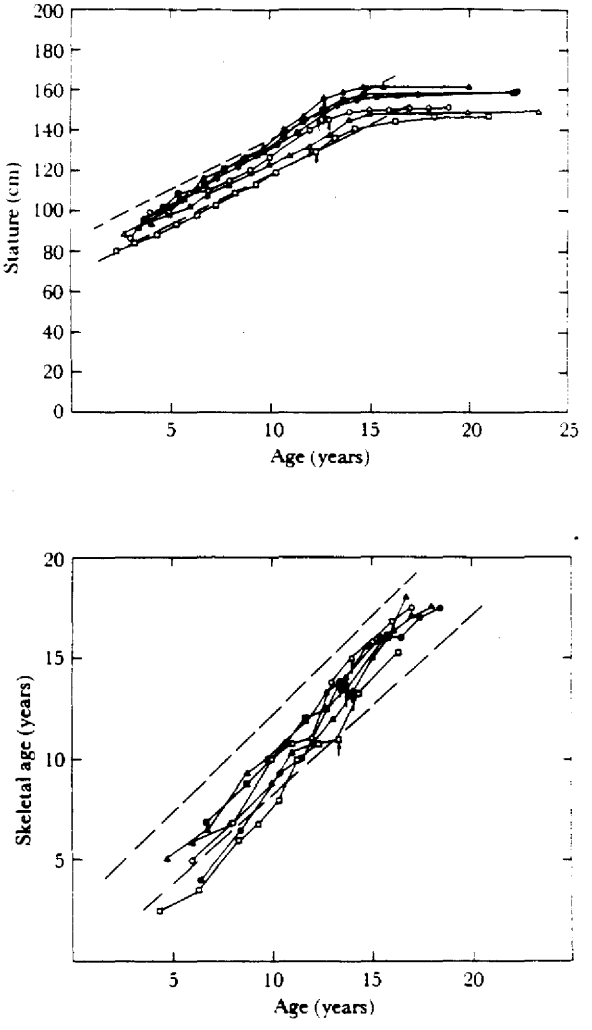


Figure 22. Same data as in Figure 22, for girls. Subjects: ●, No. 17; ○, No. 21; ▲, No. 33; △, No. 42; ■, No. 69; □, No. 65.

Table 16

Summary Data on Children Exposed *In Utero*

Subject No. & Sex	Trimester at exposure	Stature	Head circumference	Skeletal maturation
83 M	third	slightly tall	large	slightly advanced
84 M	third	slightly small	normal	normal
85 M	second	normal	small	slightly retarded
86 F	first	normal	normal	normal

(adult) statures and weights, measured at age 21 or older, of subjects exposed to radiation at age $\leqslant 17$ indicated that those exposed in Hiroshima attained a mean height markedly lower than normal and that the degree of retardation diminished with increasing age at exposure, being greatest in those exposed at age $\leqslant 5$. The effect was more prominent in boys. The preliminary tabulations of growth data in the Marshallese children[16,45] showed similar trends. Subsequently, the extent of the thyroid injury documented in the Marshallese children provided the basis for the present assumption that growth retardation reflected primarily the results of impaired thyroid function. It is anticipated that when adult (final) anthropometric measurements become available, further evaluation of the growth pattern of the Marshallese children (including further assessment of the effect of thyroid hormone administration) will be possible.

G. DEGENERATIVE DISEASES

The aging process is accompanied by an increased incidence of degenerative diseases, and irradiated animals have shown increases both in general aging phenomena and in such diseases; therefore, in these studies careful consideration has been given to signs of aging and to the incidence of cardiovascular diseases, arthritis, and other degenerative diseases.

1. Cardiovascular Diseases

Cardiovascular diseases appear to be no more prevalent among the Marshallese than in Americans or other populations, and no difference was found between their incidences in the exposed and unexposed groups. Hypertension appears to be somewhat less prevalent among the Marshallese than among Americans. Blood pressures $>140/90$ have been recorded in only about 9% of the people, and the increasing percentage of older people in the study groups does not seem to have raised the incidence of hypertension. Many of the younger people, particularly females, have notably lower blood pressures (90-100/55-65). The generally lower blood pressures in the Marshallese have been thought to be related to lower dietary salt intake,[7,46] but the gradually increasing use of imported foods of greater salt content has caused no apparent increase in hypertension. Serum sodium and potassium levels have usually been in the normal range. Routine chest x rays and ECG's on individuals >40 years old have revealed the cardiac changes expected with aging: arteriosclerosis heart disease with occasional cardiac decompensation. Auricular fibrillation has been noted in a few people; rheumatic heart disease in only two. Peripheral vascular diseases (thrombophlebitis, peripheral venous disease, hemorrhoids) appear to be less common than in Americans. Oscillometric studies showed peripheral pulses to be exceptionally good, even in older people and in diabetics (see below). No luetic, metabolic, or nutritional form of heart disease has been seen.

2. Arthritis

Many Marshallese >40 years of age complain of joint symptoms in the arms and legs and to a lesser extent in the lower back. Many, particularly the older ones, have complained also of vague muscle pains in the arms and legs. Some older women develop marked kyphosis in the lower thoracic-lumbar region which may be related partly to the squatting or stooping position necessary for cooking over open fires. X rays show many of the above complaints to be associated with osteoarthritic changes; but many people with complaints do not have definite bone changes. Arthritis seems no more common in the exposed than in the unexposed population, and its general incidence does not differ greatly from that among Americans. Rheumatoid arthritis has rarely been seen. Examination of the serum for "rheumatic factor" gave positive results in only a few cases (3 of 184 tested in 1959).[8]

3. Nephrosclerosis

Primary kidney disease has not been noted frequently, but nephrosclerosis has occasionally been seen complicating cardiovascular disease.

4. Aging

Radiation in sublethal doses may reduce longevity in animals and man. Induction of malignant transformation is well documented and accounts for most of the early deaths. However, development of premature senescence with associated degenerative diseases and immunodeficiency, as a late effect of exposure, is also believed to account for some degree of life shortening in animals.[47-49] For humans, definite proof of such effects is lacking, the only documented life-shortening effect being related to malignancies.[50,51] In the Japanese bomb survivors, the correlation of life-shortening with

malignancies is well documented,[52-55] but that with other less well-defined aging criteria is not clear-cut. Beebe et al.,[55] reporting on 1300 deaths from 1950 to 1966 among 8200 exposed Japanese, stated: "Once cancer is removed from the list of natural causes, mortality appears to bear no relation to radiation dosage. In none of the 4 time periods is there evidence of general increase in mortality that one might expect from the hypothesis of accelerated aging."

Since the underlying mechanisms of ordinary aging are not clearly defined, it is difficult to compare radiation effects with the aging process and to recognize their interaction. Studies of longevity in the relatively small Marshallese population under observation do not provide any evidence of a possible life-shortening effect of radiation. At this time the average age at death in the Rongelap exposed group is 64 years compared with 65 in the unexposed population, and 61 among the unexposed Utirik people. The differences are not significant.

Numerous empirical studies concerned with possible radiation-induced aging effects have been carried out on the Japanese survivors[52-56] and on the Marshallese.[8,12,13,57-61] On several occasions Rongelap people were given a battery of nonspecific tests for aging similar to those used in the Japanese studies.[8,12,13] Some of these tests were based on subjective assessment, on a 0 to 4+ scale, of items such as greyness of hair, arcus senilis, senile changes in the skin, balding, etc., but most involved direct measurements of items such as skin looseness, skin elasticity (skin caliper), visual accommodation, visual acuity, hearing (audiometric), blood pressure, neuromuscular function (light extinction test), hand strength (dynamometer), vibratory sense (vibrometer), and lean body mass (whole-body potassium by gamma spectrographic analysis). Comparison of these values in the exposed and unexposed Marshallese (Table 17) showed no significant differences. The biological age scores (average percent score), plotted in Figure 23 for both groups, are about the same.

5. Immunological Studies

Radiation is known to impair the immunological status of individuals soon after exposure if the dose is sufficient to produce significant leukopenia. During the early period, though the acute effects on the Rongelap people included considerable depression of peripheral blood elements, comparison of the incidence of infections with that in the Utirik group gave no evidence of impaired immunity. Reduced immunological reserve may likely be a late effect of radiation exposure,[62] but it has not been observed conclusively in man. The development of leukemia and other malignancies following exposure may quite possibly be related to re-

Table 17

Correlation of Criteria With Age and Radiation Exposure[12]

	Correlation with age (r value)	Correlation with radiation	
		Percent	Significance* (p)
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.

duced immunological surveillance. Our interest in immunological capacity is related also to the consideration that impairment of immunity is thought to be associated with the aging process.

Antibody response. Three years after exposure a number of exposed and unexposed Rongelap people were tested for antibody response to primary and secondary tetanus toxoid inoculations, with a mouse used for toxin-antitoxin assay of serum.[6*] The difference between the exposed and unexposed groups was not significant.

Blood cell changes. The persistent lag in complete recovery of leukocytes was believed to reflect reduced bone marrow reserve and therefore reduced immunological reserve. The apparent recent recovery of hemopoiesis in the exposed people to control levels may indicate an improvement in their immunological status. Table 18 shows that reductions in lymphocytes and platelets and an increase in sedimentation rates were correlated with increasing age in the Marshallese.

Immunoproteins. Immunoelectrophoretic analysis of serum proteins in 1962 showed neither a paraproteinemia nor a typical picture of antibody deficiency syndrome, but a high frequency of some of the immunoglobulins was noted.[10**] Complement fixation studies showed the Marshallese to have antibodies to most viruses except Asian influenza (an epidemic of this disease later occurred in 1972). The antibody titers appeared somewhat lower in the exposed people.[10]

The older people had higher levels of gamma globulins but slightly decreased albumin levels (1969).[13,59,60] Immunodiffusion studies showed that the rise in gamma globulin levels was paralleled by an increase in immunoglobulin.[†] The increase in IgG moiety was the most pronounced, and it showed significant correlation with age ($r=0.78$). The increase in K light chains was also significantly correlated with age. The immunoglobulin levels were more depressed in the exposed group, particularly those of IgG, IgA, and L light chains (Table 18). However, by 1974 the gamma globulin levels in the exposed people were nearly the same as in the unexposed (Table 19).

Lymphocyte function. Tests on the phytohemagglutinin (PHA) stimulation of lymphocytes cultured from peripheral blood showed a definite decrease of responsiveness with increasing age of the individual (Table 18 and Figure 24) but no definite difference between exposed and unexposed groups.[59,60] In a more recent study, acetylation of nuclei of PHA-treated lymphocytes was measured at various times as a function of lymphocyte transformation and of aging.[61] Decreased acetylation of nuclei is associated with increasing age and is roughly parallel to the decrease in lymphocyte transformation. Total acetate incorporation in the nuclei during the first hour of culture was correlated with acetylation of histones, but by 20 hr acetylation of other nuclear materials had also occurred.

Chromosome counts. In 1969 chromosome counts were made on PHA-stimulated lymphocytes cultured from peripheral blood from 78 unexposed and 27 exposed Rongelap people.[63] Both hypodiploid and polyploid levels were found to be related to the subject's age. Females >50 and exposed males >50 had ~1.5 times as many hypodiploids as did the younger subjects; polyploid levels were sharply reduced in all subjects >50. Hy-

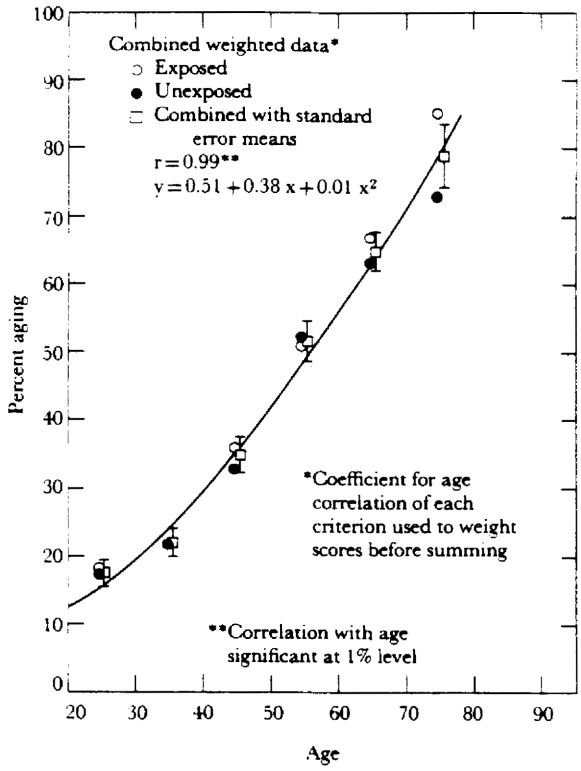


Figure 23. Biological age scores.[12]

*Dr. R.D. Stoner at BNL did these analyses.

**Drs. R. Bütler and A. Hassig at the Swiss Red Cross Laboratory did these analyses.

†These studies were done by Drs. J.L. Fahey and R. Woods of the National Cancer Immunoglobulin Center.

Table 18

Correlation of Immunoelectrophoretic and Peripheral Blood Findings With Age and Radiation Exposure[13] (D = decrease; I = increase)

Criterion	Unexposed group		Exposed group	
	Change with age	Correlation with age (r value)	Percent dif. from unexposed	Significance (p value)
Lymphocyte transformation	D	0.89	− 1.1	0.68
Serum proteins				
Total serum proteins	I	.35	− 1.5	.24
Albumen	D	.45	+15.0	.01
Total globulins	I	.58	−17.1	.01
Alpha-1	I	.37	−31.0	.01
Alpha-2	I	.43	−20.0	.01
Beta	I	.32	− 6.0	.03
Gamma	I	.75	−18.3	.01
Immunoglobulins				
A (IgA)	I	.49	−17.0	.05
D (IgD)	I	.20	− 3.0	.98
M (IgM)	I	.20	− 4.0	.74
G (IgG)	I	.78	− 8.0	.22
Kappa light chains	I	.96	− 3.0	.69
Lambda light chains	I	.24	−14.0	.15
K/L ratio	I	.41	+ 0.4	.74
Blood findings				
Hematocrit	D	.57	+ 2.9	.07
Sedimentation rate	I	.72	+11.4	.08
Total leukocytes	D	.43	− 2.5	.59
Lymphocytes	D	.91	− 0.1	.51
Neutrophils	I	.44	−13.8	.04
Platelets	D	.65	− 8.4	.04

Table 19

Serum Proteins, 1973-1974

Group	Total protein	Albumen	Alpha 1	Alpha 2	Beta	Gamma	Total globulin
Rongelap	7.76±0.70	4.22±0.50	0.20±0.05	0.69±0.12	1.08±0.24	1.59±0.41	3.54±0.58
Ailingnae	7.99±0.51	4.30±0.37	0.19±0.07	0.72±0.15	1.05±0.20	1.76±0.38	3.60±0.56
Utirik	7.60±0.50	4.29±0.43	0.16±0.06	0.69±0.30	0.89±0.23	1.67±0.45	3.33±0.50
Unexposed	7.60±0.71	4.11±0.45	0.19±0.06	0.75±0.14	1.00±0.24	1.57±0.48	3.52±0.54

podiploid levels were related also to radiation; this was more pronounced in the males, with the exposed having 2.8 times as many hypodiploid cells as the unexposed, whereas the exposed females had 1.3 times as many as the unexposed. Polyploid levels were not found to be related to radiation. Both sex and chromosome size appeared as factors possibly related to hypodiploid levels. In all subjects, regardless of sex or exposure, the largest and most frequent loss of chromosomes was in the G(Y) group (2.3 times expected loss). In the C(X) group, females lost 15.2% more chromosomes than expected and males 12.6% less. No sex or radiation effect was apparent in the other five chromosome groups. A series of additional cultures indicated the presence of chromosome breakage factor in the plasma of the exposed subjects. In cultures of the latter, chromosome aberrations

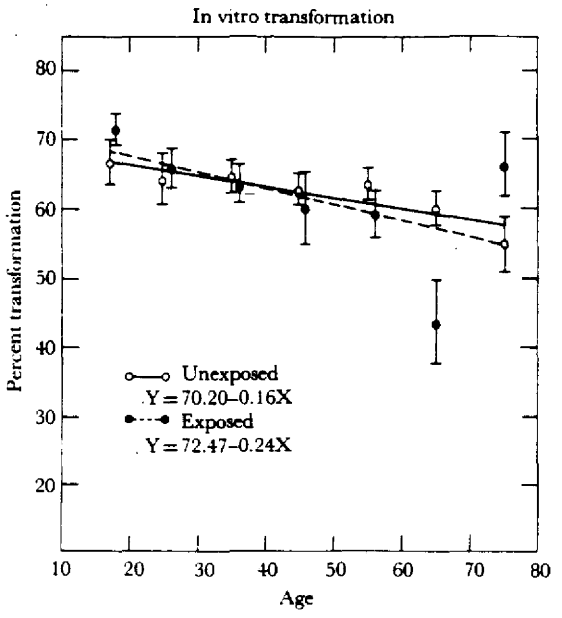


Figure 24. Age-related change in lymphocyte transformation in peripheral blood cultures showing the mean percent transformation for each decade with standard deviation.[13]

were nearly doubled but aneuploid levels were not affected.

In summary, several indications have been seen of reduction or borderline deficiency of the immunological status in the exposed Rongelap people in earlier years, but no evidence that such deficiency was related to disease incidence, with the possible exception that the increased development of malignancy in the exposed Rongelap people may be an indication of reduced immunological surveillance. Recent rises of leukocyte and gamma globulin levels to control values indicate some degree of recovery, but better tests for immunological status are needed.

6. Diabetes Survey

Diabetes is a major problem in all the Marshall Islands, and investigation of it is important for two reasons. First, the disease is difficult to manage; rules developed for U.S. and European conditions may not apply. Choice of diet is limited. The hygienic conditions favor infections and make foot care a major consideration; also, home use of insulin is precluded in all but a few cases. It would be a significant contribution to the welfare of the people to diagnose the condition early and to define the optimum rules for management, and advice on this problem has been requested by Trust Territory medical personnel. Second, the situation of scattered, sequestered population groups and the large contrast in living conditions between home atolls and district centers make it an ideal area for investigating the relative importance of pathogenetic elements. In this respect, the situation may not be matched anywhere.

During early 1974, 375 people from Utirik and Rongelap Atolls, some of whom are now residents of Majuro and Ebeye, were examined in order to establish the incidence and nature of diabetes in the Marshall Islands.* The 28 previously diagnosed diabetic patients from other atolls were also examined but are not included in this analysis. On Majuro, 120 subjects were surveyed; on Ebeye, 116; on Utirik, 81; and on Rongelap, 58. Blood and urine glucose determinations were made, a questionnaire was filled out, and pertinent physical examination was done to assess the presence or absence of degenerative complications of diabetes. Blood was also obtained for uric acid, cholesterol, and triglyceride determinations. Plasma glucose was measured in the fasting state and/or 2 hr after the ingestion of 75 g carbohydrate (Glucola). In this survey a plasma glucose level, either fasting or post-prandial, >120 mg % was considered indicative of abnormal glucose tolerance. In some cases glycosuria was accompanied by normal plasma glucose and these were not included in the group with diabetes. Some subjects with glycosuria did not have plasma glucose determinations (for a variety of reasons); these are categorized as possible diabetics. Although attempts were made to obtain complete data on all subjects aged >15, these were not always successful. The failure to supply requested information on some questionnaires accounts for the discrepancies in the numbers given in Table 20. The results are also categorized on the basis of whether the subject previously knew of the abnormality in glucose tolerance. Obesity is evaluated on the basis of height and weight but frequently this information is not complete. The data are being analyzed in their present form, but the missing information will be obtained, if at all possible, as the program continues.

Preliminary evaluation of the data (see Table 20), with the limitations mentioned, strongly suggests that the incidence of diabetes mellitus in the

*We are grateful to Drs. James B. Field and Catherine Detre at the University of Pittsburgh School of Medicine for analysis of the diabetes data.

Table 20

Diabetes Survey: Preliminary Results, 1974

Group	No. people*	Diabetes		Glycosuria with normal blood sugar	
		No. cases	%	No. cases	%
Rongelap and Utirik	375	103	27.5	29	7.5
Male	180	48	26.7	17	9.4
Female	195	51	25.9	10	5.1
Obese	196	58	29.6	18	9.2
Non-obese	136	22	16.2	10	7.4
Utirik	201	57	28.4		
Rongelap	174	46	26.4		
Residing at**					
Majuro	120	37	30.8		
Ebeye	116	29	25.0		
Rongelap	58	16	27.6		
Utirik	81	21	25.9		

*Includes 10 subjects from other atolls, married to Rongelap and Utirik people .

**Includes both Rongelap and Utirik people residing at these locations.

Marshall Islands is considerable and is probably greater than in any other population groups except possibly certain American Indian groups.[64] Although some previous reports have indicated a high incidence of diabetes in some Polynesian populations,[65-69] others have suggested a low incidence.[68-70]

The overall incidence of diabetics was 26%, and ~ ⅓ of these patients were unaware of their abnormality. The average age of the diabetics was considerably greater than that of the nondiabetics. The disease appears to be as common in males as in females, but a definite statement must await completion of the data analysis. Obesity was much more common in women than in men, and the data indicate an increased incidence among obese subjects. Cataracts seemed to be much more common in the diabetics than in the nondiabetics, but this would certainly be influenced by the older age of the diabetic population. No definite difference was seen in the incidence of cataracts among individuals known to have diabetes compared with those in whom the diagnosis was made during this survey. A significantly higher percent of the diabetics than of the nondiabetics had neuropathy, but, because of age differences and other factors previously stated, covariance must be eliminated from the data before these observations can be evaluated. (Neuropathy was evaluated on the basis of history of paresthesias and/or objective abnormalities in reflexes and sensory perception.) The limited data available did not suggest evidence for increased retinopathy or peripheral vascular disease among the diabetics. The disease more closely resembles maturity onset diabetes with absence of acute symptoms, ketosis, and absolute dependence on insulin treatment.

The data do not suggest any differences between Rongelap and Utirik people (genetics) or between places of residence (environmental influence), and there is no evidence that radiation exposure has played a part in the pathogenesis. The differences in diet and general living conditions between the "outer islands" and the district centers are considerable and might well play a significant role in pathogenesis. However, analysis of this factor is difficult because of the mobility of the people, who constantly move between home island and the centers; the place where an individual is examined may not be where he has spent the major part of, say, the last 10 years.

Despite the limitations, the preliminary results are considered to be of sufficient interest to warrant a more careful and complete investigation. Additional observations will be made to provide the missing information and to obtain more objective data regarding the incidence of neuropathy, cataracts, and retinopathy in these patients. The insulin response to a glucose challenge would also be of interest in assessing some of the factors that might be important in the etiology of diabetes in the Marshall Islands.

The examinations are by necessity restricted to Rongelap and Utirik people. When the Eniwetok and Bikini people return to their home islands in sufficient numbers, the diabetes investigations may be expanded to cover these groups.

H. CHROMOSOME AND GENETIC STUDIES

1. Studies of Chromosomes for Radiation Effects

In 1964 chromosome preparations were obtained from lymphocytes cultured from the peripheral blood of 43 exposed (21, age <20; 22, age

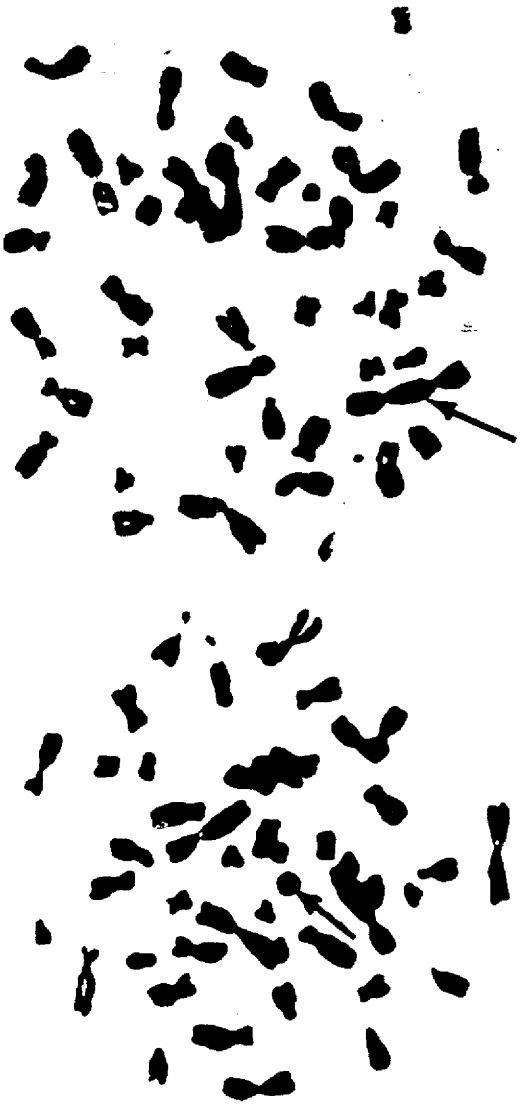


Figure 25. Two-hit chromosome aberrations in exposed Marshallese. Top: arrow points to dicentric form; bottom: arrow points to ring form.[12]

>20) and 8 unexposed Rongelap people.* Chromosome aberrations were noted in 23 of the exposed and in 5 of the unexposed Marshallese, but the exposed group had a number of two-break aberrations (represented by dicentric chromosomes, translocations, and a ring form) that are thought to be associated with radiation exposure (Figure 25).[71] No two-hit aberrations were found in the unexposed group, but both groups had an unusual number of acentric fragments, the cause of which is not known. Paradoxically, Rongelap people with the lower exposure had more aberrations than those with the higher exposure. These studies indicate that a small but significant number of chromosome aberrations persisted in blood lymphocytes in some Marshallese as late as 10 years after exposure. The results are consistent with those of similar studies on the exposed Japanese fishermen,[72] on victims of other radiation accidents,[73] and on Japanese bomb survivors.[74]

*These analyses were done by Drs. H. Lisco, New England Deaconess Hospital, and R.A. Conard, BNL.

2. Somatic Mutations

In 1974, studies* were made of the frequency of amino acid substitution in the hemoglobin of the Marshallese on blood samples sent to Oak Ridge.[75]

Since there is no coded isoleucine[76] in adult human hemoglobin A, its presence must be due to errors in transcription or translation or to somatic mutations arising during DNA replication. Errors in transcription, which occur infrequently, form altered mRNA, tRNA, and rRNA; they change the coding in the mRNA and may reduce the fidelity of the tRNA with regard to both the kind of amino acid it accepts and the mRNA codons it recognizes.[77] Errors in translation[77] arise through the attachment of wrong amino acids to tRNA (aminoacyl synthetase errors) and the imprecise recognition of mRNA codons by tRNA anticodons (translational variation). Somatic mutations result from mistakes in replication of DNA; many single-base-substitution mutations change nonisoleucine into isoleucine codons,[78] and the resulting mutant cells could have hemoglobin mRNA with isoleucine codons. For this reason, an increase in the isoleucine content of hemoglobin A would be expected in humans exposed to agents causing base-substitution mutations. Possibly radiation may cause base-substitution mutations in human somatic cells, but this has never been established.

The isoleucine content of the hemoglobin A was determined in blood from 13 exposed Marshallese and 12 unexposed. The frequency of isoleucine substitution for other amino acids in hemoglobin was calculated by dividing the nanomoles of isoleucine by the total nanomoles of all other amino acids in each sample. The frequencies are listed in Table 21.

*These studies were done by Drs. R.A. Popp, G.P. Hirsch, and E.G. Bailiff at Oak Ridge National Laboratory.

Table 21

Frequency of Substitution of Isoleucine for Other Amino Acids in Human Hemoglobin From 25 Marshallese

Subject No. and sex	Age at exposure, yr	Age at present, yr	Substitution frequency ($\times 10^{-5}$)	Average ± SEM ($\times 10^{-5}$)
Exposed, 175 R				8.81±1.96
3 M	1½	21	19.79	
10 M	30	50	3.58	
18 F	24	44	5.06	
24 F	13	33	13.45	
33 F	1	21	4.74	
35 F	12	32	5.19	
42 F	2	22	10.40	
71 F	27	47	8.29	
Exposed, 69 R				5.94±1.92
6 M	1	21	6.98	
8 F	1½	21	12.93	
44 M	3	23	4.04	
45 F	31	51	3.65	
81 F	7	27	2.12	
Unexposed				3.20±1.52
813 M		20	3.37	
815 M		24	2.17	
929 F		35	3.47	
836 M		41	2.45	
839 F		46	1.89	
841 F		41	3.56	
846 F		51	2.41	
867 F		46	2.12	
868 F		51	4.35	
944 M		49	3.93	
1547 F		60	7.15	
1549 M		21	1.57	

A slight, but insignificant, increase in the isoleucine substitution frequency was found in controls aged between 20 and 51; the linear regression has a positive slope of 0.0234×10^{-5}/year. Except for subject No. 1547, the higher frequencies were found in samples from exposed persons, but some of the exposed had values in the control range (Table 21). The higher frequencies were observed more often in individuals exposed at younger ages, although the globin from subject No. 33, exposed at 1 year, had a low isoleucine content. The findings are consistent with the higher leukemia induction, among persons exposed to x rays[79] and to atom bomb irradiation,[80] in those exposed prenatally and at young ages.

Studies in progress strongly suggest that the increased isoleucine content in the hemoglobin of exposed Marshallese is due to base-substitution somatic mutations. The supporting data[75] include analyses showing (1) that higher isoleucine substitution frequencies occur in both the alpha and beta chains of hemoglobin from exposed persons, and (2) that contamination by fetal hemoglobin, which does contain isoleucine, could contribute no more than 7 parts per million amino acid residues to the values reported in Table 21.

3. Genetic Studies

a. Possible Radiation Effects. The inheritance of radiation-induced mutations has been amply demonstrated in genetic studies on animals, but it has not been unequivocally seen in man. Large numbers of animals are necessary to demonstrate such an effect, and the size of the irradiated human populations studied is probably too small for it to be readily detected. The largest-scale human study was made by Neel et al. on the children of parents exposed in Hiroshima and Nagasaki,[81] and it showed no clear-cut genetic effects. Examinations of the much smaller group of Marshallese

children born to an exposed parent or parents have shown, on the basis of incidence of gross anomalies, no evidence of inherited radiation-induced mutations.

The principal aberrations noted in Marshallese adults and children have been bilateral shortening of the fifth finger, prominent head of ulna, dislocated wrist, enlarged cornea, tortuosity of retinal vessels, asymmetry of face, congenital nystagmus, and pigmentation of the cornea. Studies of familial patterns of such defects have not been done; they are not related to exposure.

The possible increase in miscarriages and stillbirths among the exposed women during the first 5 years may lead to speculations that radiation induced lethal mutations in germ cells of the ovary or, less likely, in sperm cells.* The presence of chromosome aberrations in peripheral blood lymphocytes in the Marshallese 10 years post exposure, and the possible somatic mutation in the hemoglobin, described above, support speculations that mutations in the germ plasm occurred and might be detected in the offspring by more sensitive tests. However, in view of the relatively small dose of radiation and the small size of the population, on *a priori* grounds a clear-cut demonstration of a genetic effect of radiation in the children born to exposed parents would seem unlikely. Nevertheless, given the nature of the circumstances, it has been deemed desirable to make every effort to collect such data. Since recessive combinations may take several generations to affect the phenotype, recognition of their radiation etiology may be extremely difficult. Neel et al. are at present carrying out more sophisticated studies in the Japanese, concerned with the occurrence of variant forms of 24 serum proteins and erythrocyte enzymes. He and Dr. R.E. Ferrell kindly consented to examine blood samples collected in 1974 from 187 Marshallese children and parents (exposed and unexposed). The results, summarized below, included no significant findings indicating radiation-induced mutations.

b. The Frequency of "Rare" Protein Variants. The blood samples mentioned above were subjected to electrophoretic analyses for polymorphisms and rare variants of proteins and erythrocyte enzymes.[82] The findings were summarized by Neel et al.[82] as follows.

"Blood specimens from a sample of 187 Marshall Islanders were studied with reference to variants of 24 serum proteins and erythrocyte enzymes. Six of the traits studied exhibited genetic polymorphisms (adenosine deaminase, phosphoglucomutase$_1$, acid phosphatase, 6-phosphogluconate dehydrogenase, haptoglobin, group specific component). There was in addition one 'rare' variant (of albumin) in 4047 determinations. These results on rare variants have been combined with those of others on Micronesians, and the frequency of rare variants in Micronesians compared with the frequencies in West European Caucasians, Japanese, and Amerindians. There are many difficulties in such comparisons, and, although the observed values for the four ethnic groups differ by a factor of

*About one-half the miscarriages occurred in exposed women married to unexposed men.

Table 22

Gene Frequencies for Six Genetic Polymorphisms in the Marshall Islands[82]

System	Phenotype 1	2-1	2	Total	Gene frequency
Adenosine deaminase	167	18	0	185	ADA^1 = 0.951
Group specific component	116	50	2	168	Gc^1 = 0.839
Haptoglobin*	56	93	32	177	Hp^1 = 0.579
Phosphoglucomutase$_1$**	156	26	1	184	PGM_1^1 = 0.912
	A	AB	B		
Acid phosphatase	101	73	11	185	AP^A = 0.743
6-Phosphogluconate dehydrogenase	164	21	0	185	$6\text{-}PGD^A$ = 0.943

*The Hp^0 type was observed in 4 individuals.
**Two examples of the PGM phenotype 2-7 and a single phenotype 1-7 were observed (PGM_1^7 = 0.008).

3, the Micronesians exhibiting the lowest frequency, it is felt that no conclusions concerning differences between ethnic groups can be drawn at this time."

Table 22 shows the gene frequencies for the six genetic polymorphisms found.

c. Blood Studies for Genetically Inherited Traits in the Marshallese.* A large body of data has been collected from genetic studies on the Marshallese people. The results are not only of great anthropological interest but also may show in time some possible genetic effects of radiation exposure in future generations. Table 23 lists the frequency of the various traits tested.

Blood grouping studies show that the Marshallese have a relatively high B gene frequency, a high N gene frequency, an extremely high R^1 gene frequency, and total absence of Kell and Diego factors. These characteristics differ from those of Polynesians and suggest a relationship of the Marshallese people with Southeast Asians and Indonesians. Haptoglobin studies showed a frequency of the Hp^1 gene higher than in European 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. *Transferrins* in all sera were type CC, the common European type. β-Aminoisobutyric acid urinary levels showed the Marshallese to be the highest excreters of this acid of any population thus far reported. Levels in the exposed group were about the same as in the unexposed group, and no correlation was found with body burden levels of radionuclides; this indicates that there is probably no correlation with radiation exposure. *Hemoglobin types* were considered normal (all had type AA_2). *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). 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. The level of BAIB (β-amino-*iso*-butyric acid) was the highest yet reported for any population.

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 closest kinship with people of Southeast Asia.

Table 23

Genetically Inherited Traits in Marshallese

Trait	Frequency (%)	Gene frequency	
ABO (310 people)			
O	60.7	R_e	0.768
A	19.7	P_c	0.128
B	15.1	Q_c	0.103
AB	4.5		
MN (310 people)			
M	8.0	M	0.194
MN	22.0	N	0.806
N	70.0		
Kell (310 people)	0.0		
Diego (310 people)	0.0		
Rh (310 people)			
Rh_1Rh_1	90.9	R^1	0.950
Rh_1rh_2	4.2	R^2	0.020
Rh_1Rh_2	3.9	R^0	0.030
Rh_2	0.3		
Rh_0	0.6		
Haptoglobins (176 people)			
Types 1-1	33.5		
Types 2-2	18.2		
Types 2-1	47.2		
Types 0 & rare	1.1		
Ag system (187 people)			
C.deB.	98.0		
New York	38.0		
Hemoglobin (171 people)			
AA_2	100.0		
BAIB (188 people)			
High excretors	90.0		
Australia antigen (474 people)			

*We are grateful to the following persons for analyses: blood groupings – Dr. L.N. Sussman, Beth Israel Hospital; haptoglobins, transferrins, AG antigens – Dr. B.S. Blumberg, Institute for Cancer Research, Philadelphia; hemoglobin types – Drs. R.L. Engle, Jr. and G. Castillo, Cornell University Medical Center, and Dr. S.H. Bayer, Johns Hopkins Hospital; glucose-6-phosphate dehydrogenase studies – Dr. Bayer; BAIB studies – Dr. S.M. Gartler, University of Washington, and Dr. Blumberg.

These data also may be useful as a base line, should genetic changes appear in later generations, possibly related to radiation exposure.

IV. Thyroid Findings

A. EARLY FINDINGS

Examinations for possible thyroid abnormalities were an important part of the program from the beginning. At the time of the accident it was not considered likely that the thyroid had received a sufficient dose of radioiodine to result in abnormalities. In retrospect this proved to be quite wrong, since thyroid injury and its sequelae have been the most serious late result of the fallout exposure in the Marshallese people. A chronological review of events leading to the development of thyroid abnormalities follows.

Beginning several years after exposure it was noted that 5 of 19 children exposed at <10 years of age showed retardation of growth.[8,16] This was particularly notable in the boys exposed at <5 years of age (Figure 26). The cause of this retardation was not immediately apparent. It was recognized that thyroid hormone deficiency from thyroid injury could result in such growth retardation. However, examinations during this early period did not reveal any thyroid abnormalities, and the PBI levels in these children as well as in all Marshallese were in the normal to high range. The growth retardation gradually became more apparent, and at

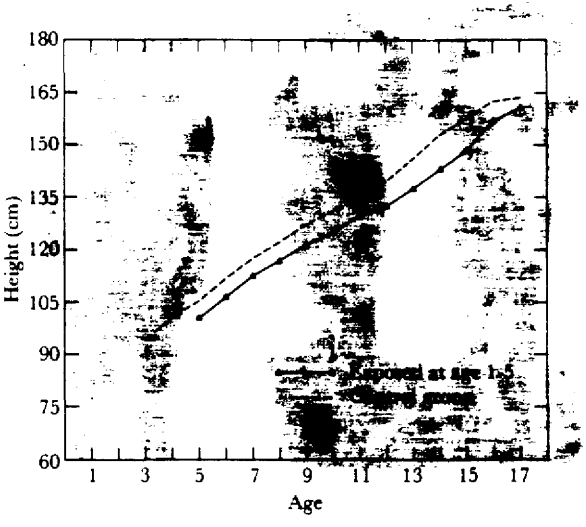


Figure 26. Statural growth in Rongelap boys exposed at <5 years of age, 1958-1967.

Figure 27. Growth-retarded boys exposed at age 1 year (No. 3, left, and No. 5), 6 months after start of treatment at age 11 (1966).

Figure 28. Bone dysgenesis of heads of humeri in subject No. 5, typical of hypothyroid disease (1965).

Table 24

Thyroid Findings, September 1974

Subject No. and sex	Est. thyroid dose,* rads	Age at exposure	Age lesions developed	Diagnosis** (S = surgery)
Rongelap exposed (175 rads), 64 people (3 *in utero* listed separately)				
3 M	1150	1	12 (?)	Myxedema (thyroid atrophy)
5 M	"	1	12 (?)	Myxedema (thyroid atrophy)
33 F	"	1	13	(S) Adenomas
54 M	"	1	13	(S) Adenomas (leukemia, died age 19)
65 F	"	1	13	(S) Adenomas
2 M	1100	2	12	(S) Adenomas
17 F	1050	3	12	(S) Adenomas
19 M	"	3	14	(S) Adenomas
21 F	"	3	12	(S) Adenomas
32 M	"	3	–	–
42 F	"	3	15	(S) Adenomas
23 M	1000	4	16	(S) Adenomas
69 F	"	4	14	(S) Adenomas
72 F	905	6	17	(S) Carcinoma (papillary-follicular), adenomas
15 F	855	7	21	(S) Adenomas
20 M	"	7	18	(S) Adenomas
36 M	"	7	19	(S) Adenomas
47 M	810	8	–	–
61 F	"	8	19	(S) Adenomas
76 M	655	11	–	Hypertrophy, lobular
75 F	655	12	30	(S) Adenomas
24 F	570	13	–	–
35 M	"	13	–	–
26 M	"	13	–	Died 1962
67 F	520	14	34	Nodule 0.5 cm, rt.
39 F	475	15	–	Hypertrophy rt. lobe
49 F	"	15	–	Lobular hypertrophy
74 F	425	16	–	–
22 F	380	17	–	–
12 F	335	18	–	–
73 M	"	18	–	–
37 M	"	20	–	–
18 F	"	21	34	(S) Carcinoma (follicular)
9 M	"	22	–	Thickened upper lt. lobe 1974
10 M	"	24	–	–
14 F	"	25	–	–
27 M	"	26	–	–
77 M	"	26	–	(Leprosy)
71 F	"	28	–	–
40 M	"	29	40	(S) Adenomas
64 F	"	30	41	(S) Carcinoma (papillary-follicular)
66 F	"	30	–	–
7 M	"	36	–	–
63 F	"	36	–	–
78 F	"	37	–	–
4 M	"	38	–	–
79 M	"	39	–	–
68 M	"	44	–	Died 1974
25 M	"	44	–	Died 1956
34 F	"	45	–	–
52 F	"	46	–	Died 1963
80 M	"	46	–	–
11 M	"	50	–	–
82 M	"	50	–	–
30 F	"	52	–	Died 1968
62 F	"	55	–	Died 1959
60 F	"	56	–	Died 1959
13 F	"	59	–	Died 1966
58 F	"	59	–	–
56 F	"	66	–	Adenomas found at 1962 autopsy†
38 M	"	75	–	Died 1957
46 M	"	76	–	Died 1956
55 M	"	76	–	Died 1966
57 F	"	98	–	Died 1963

Table 24 (Continued)

Thyroid Findings, September 1974

Subject No. and sex	Est. thyroid dose,* rads	Age at exposure	Age lesions developed	Diagnosis** (S = surgery)
Ailingnae exposed (69 rads), 18 people (1 *in utero* listed separately)				
6 M	450	1	–	–
8 F	"	1	17	(S) Adenomas (benign?)
44 M	395	4	–	–
48 F	360	6	–	Hypertrophy rt. 1974
53 F	320	8	27	Mass 0.5 cm, lt. lobe '73; neg. '74
81 F	"	8	–	–
70 F	190	15	–	Neurofibroma neck removed (1968)
51 F	135	25	45	(S) Adenomas
31 M	"	31	–	Died 1958
45 F	"	32	51	(S) Adenomas
50 M	"	34	–	Died 1971
16 M	"	39	–	–
41 M	"	44	63	Nodule lt. lobe
59 F	"	44	56	(S) Adenoma, died 1968
1 F	"	54	–	–
29 M	"	65	–	Died 1966
43 F	"	67	–	Died 1964
28 F	"	68	–	Died 1965
In utero exposed, 4 people				
83 M	175+?	2nd tri	19	(S) Adenomas
84 M	69+?	" "	–	–
85 M	175+?	1st tri	–	–
86 F	175+?	" "	–	–
Utirik exposed (14 rads), 157 people††				
2229 F	31	20	36	(S) Carcinoma (follicular)
2208 F	"	35	54	(S) Adenomas
2212 F	"	35	54	(S) Adenomas
2194 F	"	37	53	Lobular gland
2258 M	"	47	63	Nodule, died 1970
2182 F	"	52	72	Nodule
2221 F	"	54	71	(S) Adenoma
Rongelap unexposed, 196 people (~80% of group)††				
938 F	–	–	32	(S) Adenoma
829 F	–	–	34	(S) Adenoma
841 F	–	–	54	(S) Adenoma
845 M	–	–	50	Possible nodule
910 M	–	–	70	Nodule 0.5 cm, rt.
912 M	–	–	20	Firm area, nodule?
1007 M	–	–	58	Small nodule, lt. lobe
858 F	–	–	60 (?)	Large goiter, soft & movable, died 1973
898 F	–	–	75 (?)	Nodule 0.5 cm, lt., died 1973
Likiep unexposed, 137 people, 1970 (~50% of group)††				
– F	–	–	75	Nodular thyroid
– F	–	–	48	Nodule, lt.
– M	–	–	63	Nodule, rt.
– M	–	–	80	Two small nodules, rt.
– M	–	–	74	Mass 5 cm, rt.

*Includes gamma dose. Method of calculation[26] is described in Section II of this report.

**Includes adenomatous lesions (without distinct capsules) and Hürthle cell adenomas.

† This case was not included in the statistics because, if it had been possible to do more autopsies, other thyroid tumors might have been found in both exposed and unexposed people.

††Only subjects with thyroid lesions are listed here.

8 years post exposure it was noted that two boys were particularly stunted in growth (Figure 27).[10,16] They had been exposed at one year of age and gradually developed atrophy of the thyroid gland and signs of myxedema with puffy faces, dry skin, sluggish reflexes, and bony dysgenesis of the humerus and femur (see Figure 28). These two boys (Nos. 3 and 5) were considerably shorter than their brothers (Nos. 83 and 84) who were younger and had been exposed *in utero*. In 1965, a satisfactory method for serum thyroxine analysis by ion exchange column became available. Studies by this method showed that some of the children did indeed have low serum thyroxine levels. Control studies on normal Marshallese revealed that many of them had unusually high iodoprotein levels, leading to a false interpretation of protein-bound iodine (PBI) determinations. It became apparent only then that low thyroxine (T_4) levels in some of the children had probably been masked by high levels of iodoprotein.[18-23] Several children with slight growth retardation had lowered T_4 levels. The hypothyroidism appeared to be from primary thyroid damage and not secondary to pituitary damage, since tests for growth hormone in several growth-retarded children were normal, and their serum thyroid-stimulating hormone (TSH) levels were elevated.

B. DEVELOPMENT OF THYROID NODULES

In 1963, 9 years after exposure, a 12-year-old girl was found to have an asymptomatic nodule of the thyroid gland. Development of thyroid abnormalities in other subjects continued during subsequent years.[18-23] At present (1974) 29 of 86 exposed people of Rongelap are affected, including the two stunted boys who developed thyroid atrophy without nodularity. One of 4 children exposed *in utero* developed thyroid nodules in 1974. Figure 29 shows that, considering the population remaining at risk (persons living in 1964, at the time of appearance of the thyroid abnormalities), the trend is for continued development of lesions with recent increases among the lower-exposure Ailingnae group. Table 24 lists the thyroid status of all exposed Rongelap people and of people with positive findings in certain other populations.

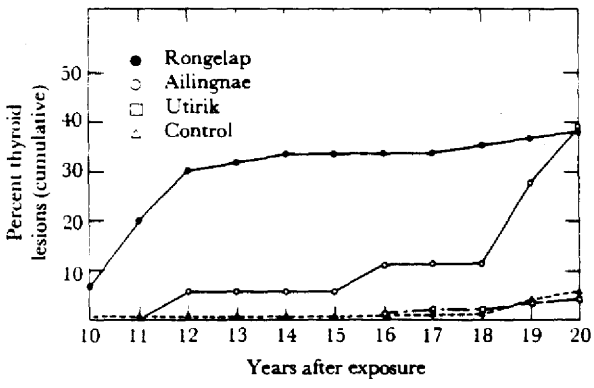


Figure 29. Cumulative percentages of persons with thyroid lesions in exposed Rongelap, Ailingnae, and Utirik groups and in unexposed Rongelap control group since 1964, based on numbers of persons in the groups in 1964, the time when radiation-induced lesions were first noted.

The thyroid nodules were usually multiple, were not tender, and varied in size from several millimeters to several centimeters in diameter. In some of the children nodular glands were associated with low thyroxine levels and slight growth retardation. The two stunted boys (Nos. 3 and 5) showed markedly reduced thyroxine levels. None of the adults with nodularity had low thyroxine levels prior to surgical exploration. The growth retardation with reduced thyroxine levels appears to be the result of radiation injury to the thyroid in the children (see Section E, below).

Table 25 lists the incidence of benign and malignant lesions and the estimated dose of radiation to the thyroid glands in the various populations. The highest incidence of lesions (89.5%) has been noted among those in the heavily exposed group who were <10 years old at the time of the accident. The absence of lesions among those of corresponding ages in the less exposed Utirik group and the unexposed groups is notable, but 2 of 6 exposed children in the Ailingnae group have recently developed lesions. The incidence of thyroid lesions among the exposed Rongelap adults is considerably lower than that among the children but higher than among the Utirik or unexposed groups. The Utirik group does not appear to have had a higher incidence of thyroid lesions than the unexposed groups, but one Utirik individual developed cancer of the thyroid.

C. SURGICAL EXPLORATION

In view of the possibly malignant nature of the thyroid nodules in the exposed population, surgical exploration of the affected thyroids, with removal of nodules, was considered necessary. Thy-

Table 25

Thyroid Lesions in Marshallese, December 1974

Group	Age at exposure	Est. thyroid dose,[a] rads	% Subjects[b] with thyroid lesions	No. subjects with surgery	% Subjects[b] with malignant lesions
Rongelap exposed (175 rads)	<10	810–1150	89.5 (17/19)	15	5.3 (1/19)
	10–18[c]	335–810	16.6 (2/12)	1	
	>18	335	9.1 (3/33)[d]	3	6.1 (2/33)
	All	556[e]	34.4 (22/64)	19	4.7 (3/64)
Ailingnae exposed (69 rads)	<10	275–450	33.3 (2/6)	1[f]	
	10–18	190	0.0 (0/1)	0	
	>18	135	36.3 (4/11)	3	
	All	217[e]	33.3 (6/18)	4	
In utero exposed		175+?	33.3 (1/3)	1	
		69+?	0.0 (0/1)	0	
Utirik exposed (14 rads)	<10	60–95[g]	0.0 (0/58)	0	
	10–18	27–60	4.8 (1/21)	1	4.8 (1/21)
	>18	27	6.4 (5/78)	3	
	All	50[e]	3.8 (6/157)	4	0.6 (1/157)
Rongelap unexposed	<10		1.6 (1/61)	0	
	10–18		7.7 (1/13)	1	
	>18		5.8 (7/120)	2	
	All		4.6 (9/194)	3	
Likiep unexposed	<10		0.0 (0/31)	0	
	>10		4.7 (5/106)	0	
	All		3.6 (5/137)	0	

[a] Dose from ^{131}I, ^{132}I, ^{133}I, and ^{135}I plus gamma; mean dose extrapolated from calculations[26] for adults and 3-year-olds (see Section II).

[b] Based on number of people exposed, excluding those *in utero* (number of cases/total number in group).

[c] The thyroid is considered to be fully developed by about age 18.

[d] One additional case of adenoma, found at autopsy, not included here.

[e] Weighted mean dose.

[f] Pathologists differed as to whether this lesion was malignant; it was scored as benign.

[g] The more energetic, shorter-lived isotopes of iodine contributed less to the total thyroid dose in the Utirik people because the fallout occurred later there. It might be surmised also that the biological effectiveness of the thyroid dose per rad would be less in the Utirik group.

roid surgery has been carried out on 24 of the exposed Rongelap people with removal of varying amounts of thyroid tissue depending on the extent of the lesions (20 were subtotal and 4 total thyroidectomies with parathyroidectomy inadvertently in one of the latter who was thought to have cancer at the time of surgery). Of these cases, 3 were found to have carcinoma of the thyroid and 21 to have benign lesions of the thyroid.

The first case of carcinoma of the thyroid occurred in a 40-year-old woman (29 at the time of exposure) in the heavily exposed group. The interval between exposure and diagnosis was 11 years.[20,23] The second and third cases in this group occurred 15 years after exposure, in a 36-year-old woman (age 21 at exposure) and in a 22-year-old woman (age 6 at exposure). A fourth case of cancer was found, 14 years after exposure, in a 36-year-old Utirik woman who had received considerably less exposure.[23] Three of 196 unexposed Rongelap people have had surgery with removal of benign adenomas. All three were women. Hospital summaries of cases admitted at BNL are presented in Appendix 10.

1. Gross Appearance

Since the discovery of the first thyroid lesion 9 years after radiation exposure, clinicians particularly interested in the thyroid have participated in the annual examinations. As a result, any slight irregularities discovered by palpation drew special attention. This high degree of scrutiny and suspicion led to the detection and removal of nodules

when they were much smaller than nodules usually encountered in general clinical practice. Few of the lesions were visible from the exterior, and none was accompanied by symptoms.

At the time of surgical exploration most of the thyroids in the exposed Rongelap people were lobulated and contained small discrete masses which were not of sufficient size to cause very significant enlargement or to distort the symmetry of the gland.[22] Most of the glands in the exposed Rongelap people with only one palpable nodule proved to have multiple nodules. Often the palpated nodule was not the lesion that ultimately prompted the most concern on histological examination.

The gland in some cases showed many tortuous hair-like vessels on the surface, reminiscent of thyroids that had previously been treated with radioactive iodine for hyperthyroidism. The cut surface of the thyroids revealed some nodules which appeared to be discrete with distinct capsules (Figure 30). In some instances these discrete lesions were very firm, pale brown or whitish. In some there were hemorrhagic or degenerative cysts. The margins of some other nodules were indistinct, producing a lobular character which comprised most of the thyroid in such a manner that the entire gland appeared to be responding to a diffuse pathologic process, not unlike the type of gland observed in chronic iodine deficiency but in miniature proportions.

Figure 30. Gross serial sections of an irradiated Marshallese thyroid, showing multiple discrete adenomata developing throughout both lobes of the thyroid. Scarring is evident between these nodules.

2. Microscopic Appearance

On microscopic examination all the thyroids of exposed Rongelap people showed varying degrees of adenomatous change. Many of the lesions were completely surrounded by a distinct capsule and, unlike the remainder of the thyroid, had a distinct histological pattern which ranged from microfollicular to fetal, solid, or embryonal types. Unexpectedly many of the adenomas were papillary (Figure 31), but all except two of those that were papillary were considered benign. Most of the individuals operated on later in the series were given a small tracer dose of ^{131}I so that the functional nature of the adenomatous areas could be studied for radioiodine uptake.[83,84] Multiple autoradiographs prepared from tissues from the last 15 patients have shown that essentially all the discrete lesions took up significantly less radioiodine than the non-nodular thyroid tissue and in many cases took up none at all (Figure 32). Only in one individual a single lesion, which was papillary in character, took up more radioiodine than the surrounding normal thyroid tissue. Although reduced radioiodine uptake does not necessarily indicate a malignant lesion, it is commonly observed that lesions having a capacity to metastasize take up far less radioiodine than the extranodular tissue (usually the ratio is $< 1/100$).

Most of the thyroids have been found to contain an unusual number of minute encapsulated lesions, some of them composed of solid cellular masses of cells (Figure 33*A*, *B*, and *D*), in contrast to lesions found in most adenomatous goiters, which are composed of follicular structures similar to but not identical to normal or hyperplastic glands. On careful gross examination of the glands, these minute lesions appeared as tiny whitish dots ~ 1 mm in diameter (pinhead size). The atypicality of these lesions and the presence of mitoses in the cells of some of them give rise to speculation regarding their ultimate malignant potential (Figure 34*A* and *B* and Figure 33*D*), especially since several obviously malignant lesions have been found in this exposed population. The lesions shown are from thyroids not harboring frankly malignant lesions elsewhere, except the lesion in Figure 33*B*, which was found in a thyroid that also had a highly malignant lesion in a distant part.

Of the four malignant lesions found (Figure 35), two were papillary adenocarcinomas displaying some areas that were less well differentiated, con-

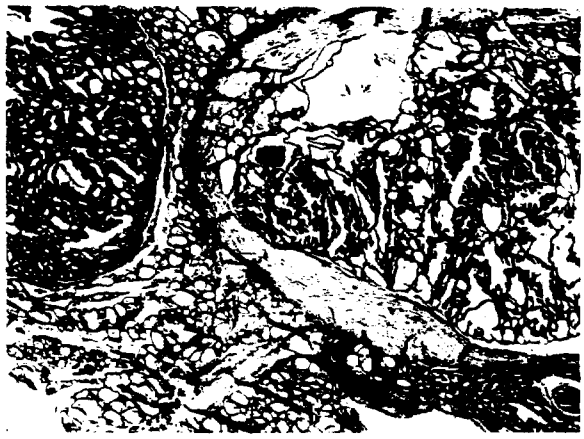


Figure 31. Two proliferative papillary adenomata that were considered benign on the basis of histological examination (×49, subject No. 15, 1969). Other minute adenomata from the same subject are shown in Figure 33*A*.

sisting of a solid cellular pattern. Both lesions were accompanied by metastases in the cervical region. In one (Figure 35*A*) the cervical metastases were extensive although the primary lesion was relatively small. A total thyroidectomy and unilateral radical cervical lymph-node and upper anterior and posterior mediastinal dissection were performed in this case, and there has been no evidence of recurrence in 5 years. The second case (*B*) was also treated by total thyroidectomy and regional lymph-node dissection. Only a single lymph node adjacent to the thyroid contained metastatic tumor although invasion to contiguous blood vessels was noted. This subject has remained free of recurrent disease for 10 years.

The third malignant lesion (*C*) was a follicular adenocarcinoma which showed considerable infiltration of adjacent normal thyroid tissue but was confined to the region of one superior pole and was not accompanied by positive lymph nodes. A total thyroidectomy with regional lymph-node dissection was done. The regional lymph nodes did not contain metastases. There has been no evidence of recurrence in 5 years. The above 3 patients with carcinoma were from Rongelap, where the exposure to fallout was greatest.

The fourth carcinoma (*D*) was a relatively undifferentiated adenocarcinoma ~2.5 cm in diameter. In many areas it was solid cellular in character. The pleomorphic cells had breeched the capsule in many places. The tumor was observed in vascular spaces but not in lymph nodes, and no distant metastases could be identified. The patient had presumably received minimal radiation exposure on Utirik, an outlying island quite remote

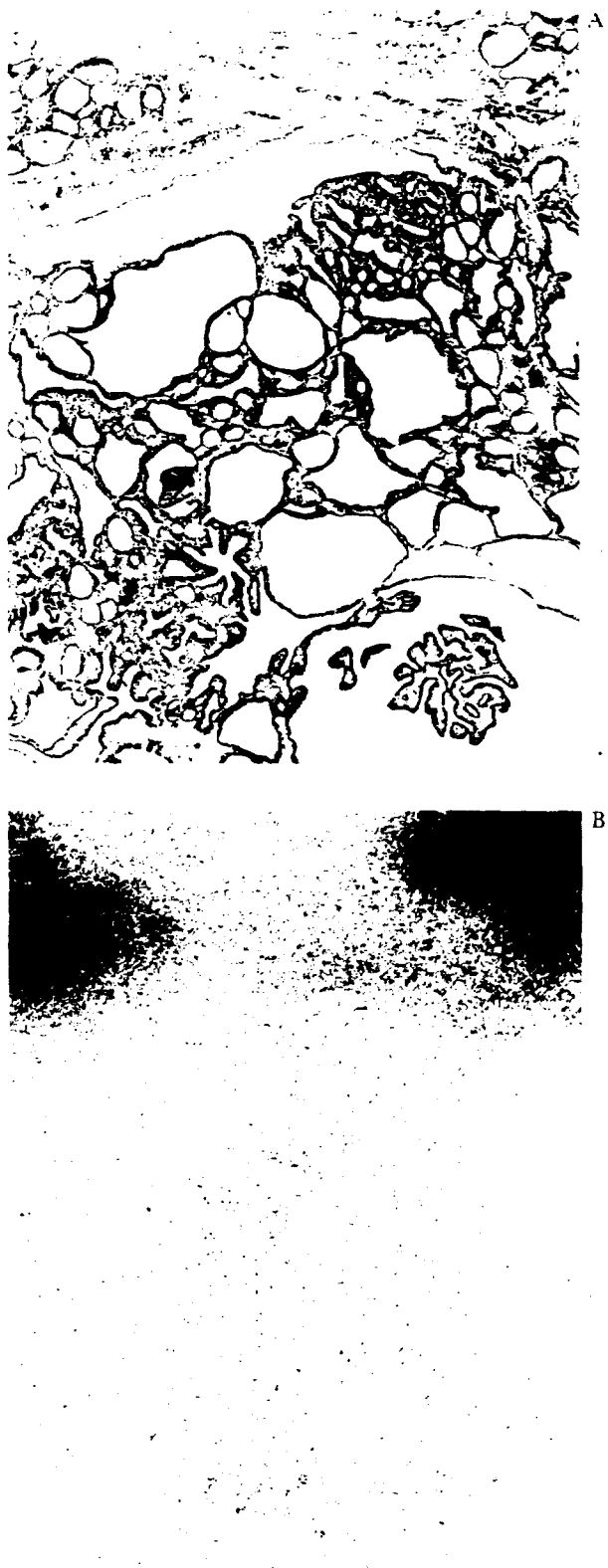


Figure 32. *A*: A histologic preparation of a lesion that developed in an irradiated Marshallese thyroid (×14). *B*: Autoradiograph, showing area of significant ^{131}I uptake to be in the "normal" extranodular tissue, in both upper corners of the section, and no uptake in the neoplasm (diffuse stippling is background). This lesion was not considered malignant. (Same subject as Figure 31.)

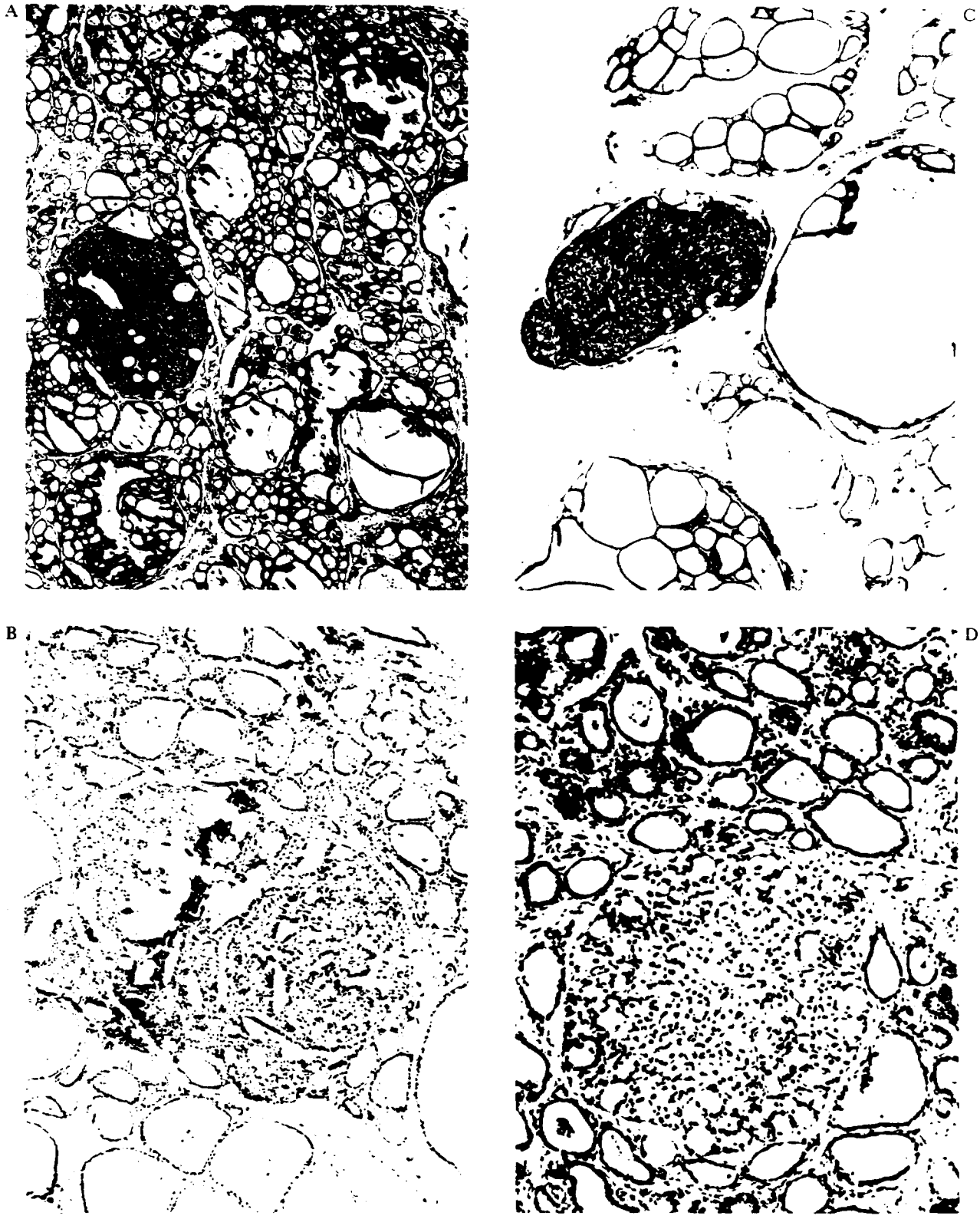


Figure 33. Examples of very minute discrete lesions of various histologic patterns scattered throughout the thyroids of many irradiated Marshallese. Many of these lesions are solid cellular or papillary; sections *A* (×15), *C* (×23), and *D* (×40) are from subjects (No. 15, 1969; No. 8, 1972; No. 40, 1973) whose thyroids contained no frankly malignant lesions. Section *B* (×52) is from subject No. 2229, 1969. Estimated doses to the thyroid (in rads) were (*A*) 855, (*B*) 31, (*C*) 450, and (*D*) 335.

from the areas of the more heavily exposed groups. Three other subjects from this island with the same exposure have had surgical excision of nodules which proved to be benign adenomas.

A fifth lesion that was papillary (the size of a match head) was found in a subject from an island where the exposure was intermediate (Ailingnae); several other individuals exposed there have developed benign lesions after a latent period somewhat longer than for those exposed on Rongelap. After review of this papillary lesion by eleven pathologists,* opinion is divided as to whether it should be considered malignant (Figure 34*A*); we have recorded it in our statistics as benign in view of the majority opinion.

A few lesions of the thyroid fall into an uncertain category in which even the most experienced pathologists are unable to reach a firm opinion as to a diagnosis of malignancy. Some of these lesions ultimately proved to have been malignant as evidenced by a distant metastasis some years after removal of the primary lesion. It seems that a neoplasm must reach some significant size before it can unequivocally satisfy the criteria for a diagnosis of malignancy even though smaller lesions may have cellular characteristics that suggest malignant capabilities. In clinical practice many lesions are of sufficient size that the criteria for malignancy are readily fulfilled. Among the Marshallese patients most of the thyroid nodules either were detected by palpation when scarcely 1 cm in diameter or were found coincidentally to removal of palpable adenoma. Many lesions showing atypical cellularity were so small that perhaps they had not yet had an opportunity to manifest all the usual criteria upon which a diagnosis of malignancy may be based. Figures 33*C* and 34*A* show one such minute lesion. This emphasizes the importance of the early diagnosis and treatment of Marshallese lesions.

*Drs. S. Warren, W. Meissner, and M.A. Legg, New England Deaconess Hospital; J.D. Reid, Cleveland Metropolitan General Hospital; T. Winship (deceased); L.B. Woolner, Mayo Clinic; L.V. Ackerman, SUNY at Stony Brook; R.V. Rosvoll, Emory University; and S. Robbins, A. Vickery, and B. Castleman, Massachusetts General Hospital.

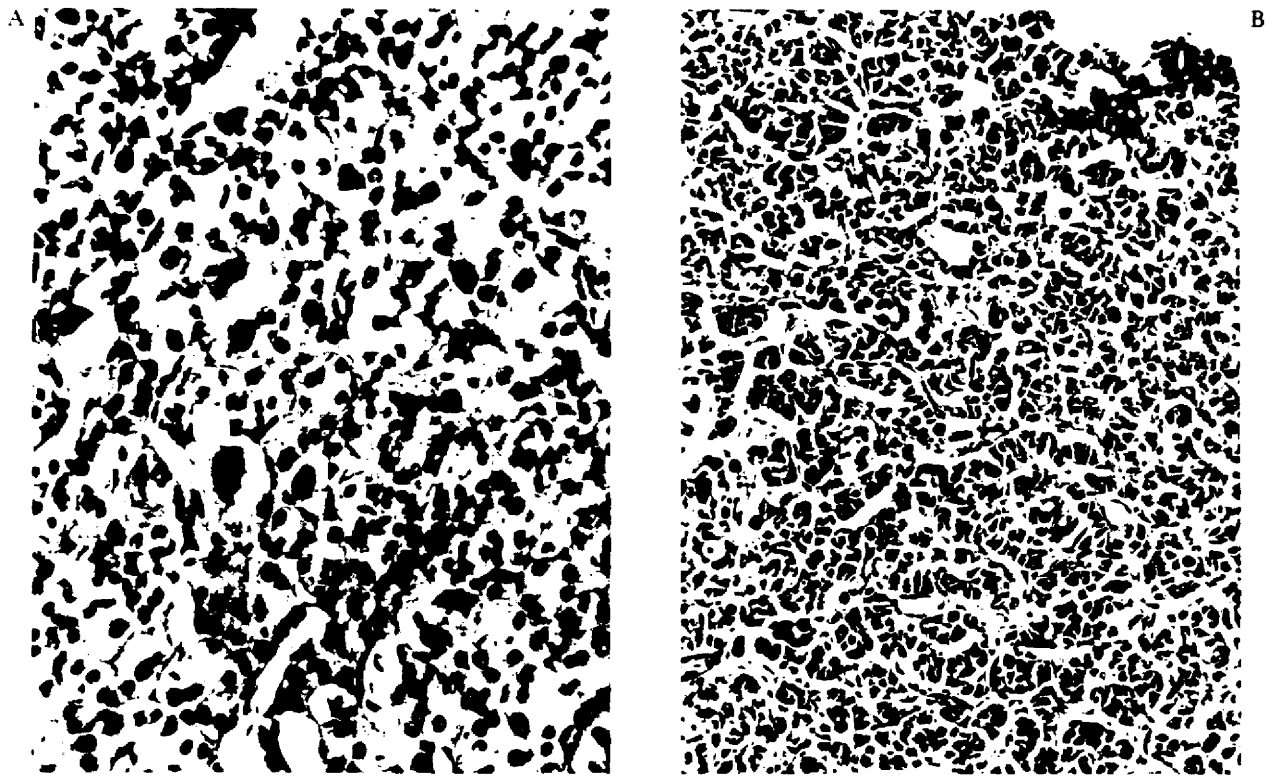


Figure 34. Examples of very minute neoplasms, only a few mm in diameter and completely encapsulated but composed of cells whose pattern suggests that if the lesions were larger they might display features prompting a suspicion of malignancy. In both examples shown (*A*, ×162, subject No. 8, 1972; *B*, × 91, No. 36, 1969) evidence of blood vessel or lymphatic invasion is lacking, and the capsule, which was very thin, was not breeched. The entire lesion of *A* is shown in Figure 33*C*, where its size can be compared with that of surrounding normal follicles. Both these examples are from thyroids that had no frankly malignant lesions elsewhere in them.

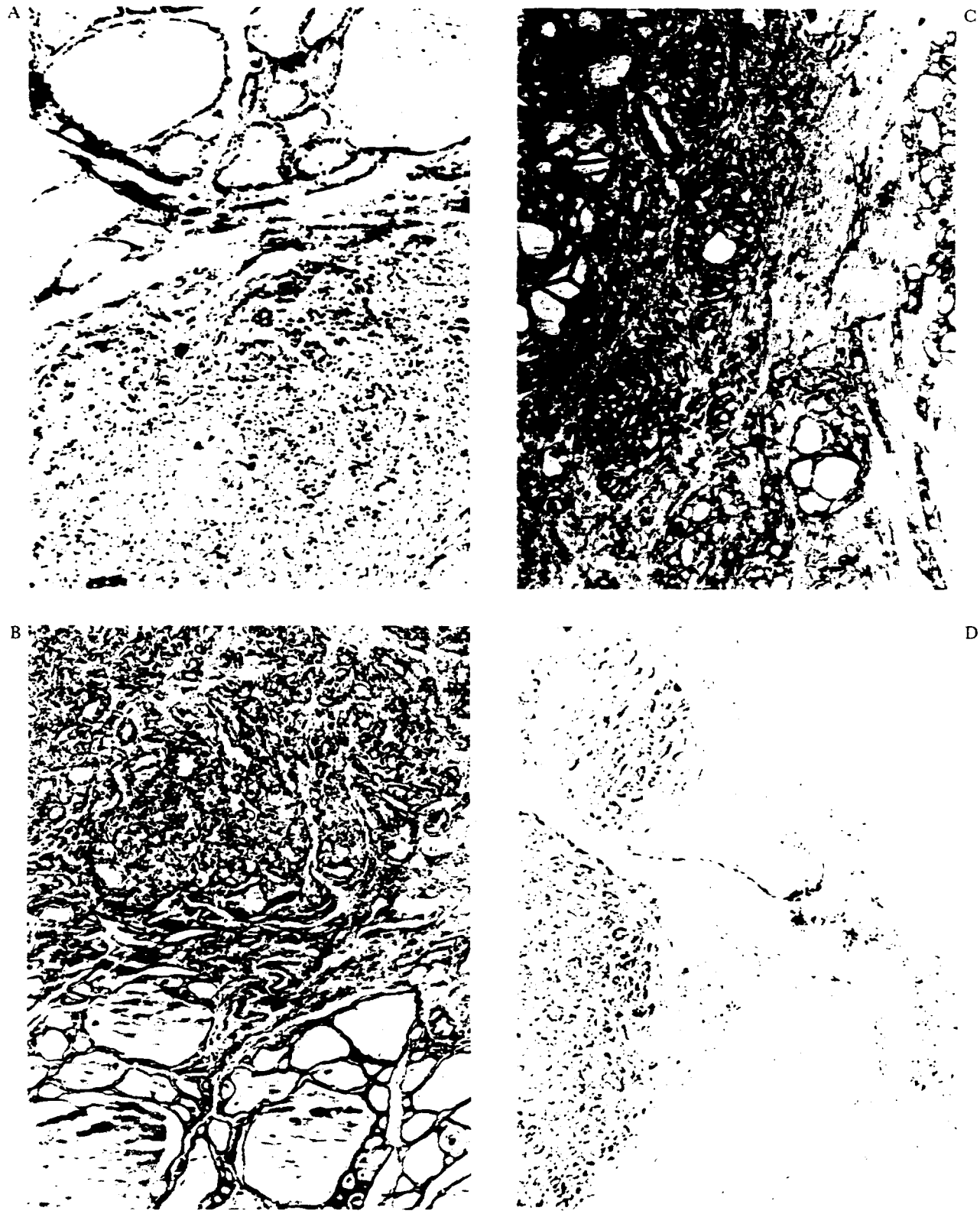


Figure 35. Four carcinomas of the thyroid found in Marshallese subjects. *A*: Papillary adenocarcinoma with many cervical lymph nodes containing metastases (×80, subject No. 72, 1969). *B*; Papillary adenocarcinoma with a single lymph node containing metastatic site (×21, No. 64, 1965). *C*: Infiltrating follicular adenocarcinoma with known metastases (×26, No. 18, 1969). *D*: Relatively undifferentiated follicular adenocarcinoma (×46, No. 2229, 1969).

D. HORMONE TREATMENT

In 1965, the seriousness of the development of thyroid lesions in the Rongelap people was recognized, and a panel of experts was called together to review the findings and make recommendations regarding the possibility of initiating preventive therapy.[19-23] The concensus was that the more heavily exposed Rongelap group should be placed on replacement thyroxine for life in order to block TSH secretion by the pituitary gland. It was hoped that nullifying the stimulating activity of this hormone on the thyroid would inhibit development of benign and malignant nodules, and that growth and development in the hormone-deficient children would be improved. The possibility of development of pituitary tumors, which has been noted in hypothyroid animals[85] and human beings,[86] might be prevented. Ethical considerations ruled out randomized clinical trials of therapy.

Synthetic L-thyroxine (Synthroid, Flint Drug Co.), which might be more stable than dessicated thyroid under tropical conditions, was recommended at a dose of 0.3 mg/day for people <50 years of age and 0.2 mg/day for those >50. Treatment was supervised by the health aide, but difficulties in maintaining a strictly regular treatment regimen soon became apparent. In an effort to overcome this problem it was found that giving the entire weekly dose at one time was effective and safe, and resulted in maintenance of normal levels of thyroxine.[87] Even on this simplified treatment schedule a few of the people, including some who had undergone thyroidectomy, showed low thyroxine levels indicating that they were not consistently taking the medication. (This was one of the reasons for establishing the post of resident physician in the Islands in 1972 to monitor the treatment program as well as offer health care.) For 4 people on this regimen who developed above-normal T_4 levels, associated with complaints of nervousness and palpitation, the dose was reduced. In view of the recent appearance of thyroid nodules among the Ailingnae group and in a subject exposed *in utero*, all persons exposed on the atoll are now included in the treatment program.

Figure 36. *Left*: Subject No. 5 (shorter) and his younger brother (No. 85) in 1963. *Right*: Same two boys in 1973 after No. 5 had been given thyroid hormone for 8 years.

Figure 37. Same comparison as in Figure 36, for subject No. 3 and his younger brother (No. 83).

The hormone therapy has unquestionably enhanced growth and development in the growth-retarded Rongelap children (Figures 21, 36, and 37 show the improvement in the two most stunted boys). However, the benefit of such treatment regarding development of nodularities in the thyroid is uncertain. Of the children exposed at age <10 years in the more highly exposed Rongelap group, only two have not developed lesions. The less exposed Ailingnae group, in which development of

thyroid lesions is more recent, has not been on treatment long enough for evaluation. The *in utero* case was not placed on thyroxine treatment until after nodules had been detected. Thyroid lesions developed in several persons who were presumably taking their thyroid medication regularly and who appeared to be euthyroid with normal thyroxine levels; on the other hand, thyroid nodules disappeared in two cases (Nos. 53 and 40) during thyroxine treatment (in the latter case, they recurred and were surgically removed).

1. Follow-up

Careful follow-up studies on the subjects who had cancer, including whole-body scans at Tripler Army Medical Center, have shown no signs of recurrence. No clear-cut evidence has been seen of further development of malignancies in the thyroid remnants in the benign cases. Because papillary thyroid carcinoma progresses very slowly, long continued follow-up observation is necessary.

No deaths or acute illnesses have been associated with the thyroid abnormalities. Morbidity has been related to the development of reduced thyroid function resulting in varying degrees of hypothyroidism and in one case of hypoparathyroidism following thyroidectomy. The lack of strict compliance with the thyroid treatment program in the operated cases involves the potential danger that serious hypothyroidism may develop, particularly in patients living on the outer islands and therefore less frequently seen.

E. STUDIES OF THYROID FUNCTION*

1. Procedures

Measurement of circulating thyroid hormone has been an important part of the evaluation of thyroid function in these surveys. During the first 10 years it was done by PBI analysis and subsequently by ion-exchange chromatography (thyroxine by column). Since 1972 evaluation of thyroid function has been greatly improved by the use of radioimmunoassay techniques for measuring T_4, T_3 (triiodothyronine), and TSH.[88,89] Studies of serum iodoproteins, which had resulted in artifactual elevations in the serum PBI in the Marshallese, are discussed below, as are more recent measurements of thyroxine-binding proteins and serum thyroglobulin.

Studies of thyroid function have also included tests of radioiodine uptake and excretion on several occasions. In the field these were done with a somewhat primitive apparatus (Figure 38). ^{132}I was used to minimize the dose to the thyroid. More extensive and sophisticated tests were done on patients brought to BNL for evaluation prior to surgery elsewhere. These included tests of thyroid radioiodine uptake and scans (technetium-99*m*) before and after TSH stimulation; determination of basal metabolism rate, cholesterol, antithyroglobulin antibody levels; and a variety of clinical chemistry tests. In a number of cases a small amount of ^{131}I was administered prior to surgery and the function of excised thyroid lesions and surrounding tissues was studied by autoradiography. One 48-year-old man (No. 40) briefly developed acute thyroiditis after TSH administration.

2. Studies of Exposed Rongelap People With Thyroid Abnormalities

Evidence of thyroid hypofunction and reduced reserve was seen in a few of the children prior to surgery (Nos. 2, 20, 33, and 65) and to a greater degree in the two boys who developed myxedema

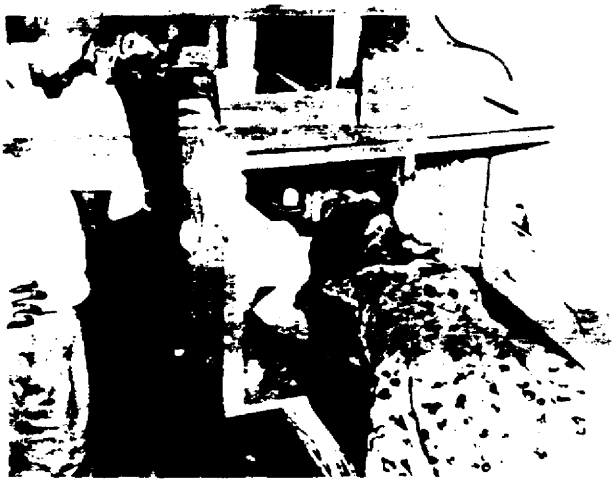


Figure 38. Thyroid function testing under field conditions, 1966.

*Thyroid uptake studies were done at Rongelap in 1965 and 1971 by Dr. J.E. Rall and in 1966 and 1972 by Dr. J. Robbins. At BNL thyroid function studies were done by Dr. H.L. Atkins. PBI determinations were made by the Clinical Chemistry Section at BNL and by Bio Science Laboratories, Van Nuys, Calif., who also did other serum iodine analyses. T_4 and T_3 by RIA, and dialyzable T_4, TSH, and TBG by reverse-flow electrophoresis were analyzed by Dr. P.R. Larsen at the University of Pittsburgh and more recently at the Peter Bent Brigham Hospital, Boston. TGB analysis by RIA was done by Drs. M. Gershengorn and J. Robbins, and TG analysis by Drs. M. Izuma and J.-L. Baulieu.

Table 26

Kinetic Analysis of $^{132}I^-$ Accumulation and Excretion in Rongelap Subjects Without Thyroid Abnormality

Group	Fraction of extrathyroid I^- per day		Thyroid fraction, $\lambda_{21}/(\lambda_{21}+\lambda_{61})$
	Urine* λ_{61}	Thyroid** λ_{21}	
9 Unexposed adults, 1965	0.81 (0.17–1.99)	0.79 (0.23–1.47)	0.49 (0.26–0.77)
12 Exposed adults, 1965	1.10 (0.34–2.57)	0.67 (0.33–1.27)	0.38 (0.24–0.65)
12 People exposed at age 10–20, 1972	1.18 (0.72–1.49)	0.90 (0.57–2.06)	0.43 (0.19–0.58)

*λ_{61} is the fraction of iodide outside the thyroid excreted in the urine per day.
**λ_{21} is the fraction of iodide outside the thyroid that enters the thyroid per day. Means and ranges given.

Table 27

Results of Follow-Up Evaluation of Plasma Thyroxine and TSH in Marshallese With Known Thyroid Lesions (Normal values: T_4 by RIA, 5.0 to 10.2 μg/dl; TSH, <1 to 5 μU/ml; abnormal values are given in boldface)

Subject No. and sex	Age at exposure	Diagnosis	1972		1973		1974		TBG-binding capacity, μg T_4 dl
			T_4, μg/dl	TSH, μU/ml	T_4, μg/dl	TSH, μU/ml	T_4, μg/dl	TSH, μU/ml	
Rongelap exposed									
3 M	1	Myxedema	6.7	<2.5	8.9	–	**2.7**	**13**	
5 M	1	"	**2.5**	**79**	**0**	**376**	3.6	1.7	
33 F	1	Adenomas	**3.7**	**6.8**	7.4	–	**10.1**	**32**	
54 M	1	"	**6.5**	**8.2**	–	–	–	–	
65 F	1	"	–	–	**1.8**	**>120**	**1.7**	**118**	
2 M	2	"	14.9	<2.5	15.3	–	–	–	
17 F	3	"	23.2	2.0	15.1	–	13.8	<1.0	20.4
19 M	3	"	13.4	3.4	**3.6**	**32**	6.9	<1.0	
21 F	3	"	12.6	<2.5	11.9	–	12.8	1.1	26.2
42 F	3	"	15.8	<2.5	10.1	–	17.4	<1.0	
23 M	4	"	**2.8**	**81**	**5.1**	**62**	**7.8**	**6.3**	
69 F	4	"	9.8	<2.5	6.6	–	**11.1**	**11**	
72 F	6	Carcinoma	**<1.0**	**460**	**0**	**149**	**<0.4**	**116**	
15 F	7	Adenomas	**7.7**	**9.2**	11.3	–	9.8	<1.0	
20 M	7	"	7.0	2.7	13.9	–	8.9	<1.0	17.4
36 M	7	"	10.0	<2.5	**2.5**	**59**	**1.3**	**15**	
61 F	8	"	**12.4**	**6.4**	4.1	–	4.6	2.8	
75 F	12	"	–	–	22.7	–	**23**	**5.6**	45.5
18 F	21	Carcinoma	**0.4**	**110**	17.8	–	10.9	<1.0	25.3
40 M	29	Adenomas	7.2	<2.5	7.8	–	7.8	2.6	
64 F	30	Carcinoma	**7.6**	**172**	10.6	–	9.0	1.2	
Ailingnae exposed									
8 F	1	Adenomas	7.8	2.0	12.1	–	14.0	<1.0	
53 F	8	Nodule 0.5 cm '73; neg. '74	–	–	7.4	–	9.4	<1.0	
51 F	25	Adenomas	7.1	2.6	6.3	–	6.9	3.9	
45 F	32	"	–	–	4.6	4.9	4.6	1.7	
41 M	44	Nodule left lobe	5.6	2.7	4.5	<5.0	6.0	4.4	13.3
In utero exposed									
83 M	2nd tri	Adenomas	–	–	6.7	–	**6.2**	**7.0**	12.9
Utirik exposed									
2208 F	35	Adenomas	–	–	5.1	<5.0	7.0	<1.0	16.3
2212 F	35	"	10.1	3.1	5.0	<2.5	**4.4**	**9.8**	20.1
2229 F	20	Carcinoma (follicular)	2.5	11	5.9	–	5.5	1.4	35.7
2221 F	54	Adenoma	–	–	4.9	<5.0	**3.6**	**54**	15.6

with marked growth retardation (Nos. 3 and 5). T_4 levels before surgery were lower in subjects exposed in childhood than in unexposed controls. Subjects exposed as adults who developed either benign or malignant thyroid lesions did not show notable impairment of thyroid function. The serum iodine levels of exposed and unexposed people for the past 15 years are given in Appendix 8.

In 1965, the rates of urine and thyroid accumulation of ^{132}I were measured over a 4-hr period in 21 Rongelap subjects, of whom 9 were unexposed and 12 were exposed as adults.[18] The data are summarized in Table 26. Compared with North American normal values obtained in 1965[18] (urine, $\lambda_{61} \cong 2$; thyroid, $\lambda_{21} \cong 1$; thyroid fraction $\cong 0.33$), both the exposed and unexposed Marshallese showed slow urine and thyroid transfer rates of extrathyroid iodide but a high thyroid fraction. Similar studies done in 1972 on 12 Rongelapese without detectable thyroid abnormalities,

Table 28

Thyroid Status of Exposed Rongelap Subjects Without Recognized Thyroid Lesions, 1974

Subject No. and sex	Age (1974)	Plasma T_4, μg/dl			Plasma TSH,** μU/ml	TBG-binding capacity, μg T_4/dl
		Pre-TSH	24 hr post-TSH	Increment*		
4 M	58	6.9	7.8	0.9	7.0	
6 M	21	6.8	8.3	1.5	3.9	
7 M	56	4.7	5.3	0.6	2.1	18.5
9 M	42	4.5	5.3	0.8	4.5	9.1
10 M	44	9.2	12.8	3.6	<1.0	
12 F	38	6.5	8.7	2.2	4.4	
16 M	59	6.4	8.3	1.9	5.3	14.4
24 F	33	8.2	12.1	3.9	4.4	
34 F	65	7.1	10.6	3.5	6.3	
35 M	33	5.9	7.8	1.9	3.1	16.7
37 M	40	4.6	6.6	2.0	2.6	21.7
39 F	35	8.3	11.5	3.2	5.1	
41 M	64	6.0	9.4	3.4	4.4	18.4
48 F	26	7.2	12.2	5.0	3.2	
51 F	45	6.9	9.1	2.2	3.9	
66 F	52	7.0	9.4	2.4	3.1	
68 M	64	8.9	10.4	1.6	6.0	
70 F	35	7.9	11.8	3.9	1.5	
73 M	38	7.8	12.0	4.2	2.3	
76 M	31	5.4	7.0	1.6	4.7	18.6
78 F	57	6.3	7.1	0.8	8.8	25.5
80 M	66	8.1	9.1	1.0	3.0	
81 F	28	5.8	7.8	2.0	3.4	21.2
82 M	70	5.8	6.7	0.9	3.8	
83 M	20	6.2	8.6	2.4	7.0	
85 M	20	7.8	11.4	3.6	2.3	
1 F	74	5.1			1.4	
11 M	70	6.8			3.9	
14 F	45	6.7			3.0	
22 F	37	3.5			2.7	
27 M	36	7.5			2.7	
44 M	24	5.8			4.2	
58 F	79	8.2			5.3	
63 F	56	8.2			6.3	
67 F	34	7.4			2.7	
71 F	48	4.2			10.3	
32 M	24	7.5			<1.0	
47 M	28	8.4			7.0	
Mean ± S.D.		6.6±1.7		2.35±1.2		

*Normal in 13 Pittsburgh subjects: 4.7±1.0 μg/dl.[88]

**Measured in pre-TSH samples. Normal is <5 μU/ml

who had been exposed at age 10 to 20 years, gave results similar to those obtained in 1965. Thus the Rongelapese do not show a trend toward decreasing thyroid iodine uptake as do North Americans (attributed to increased dietary iodine intake).[90] The main purpose of the later study, however, was to see whether the exposed individuals in the intermediate age group might be developing thyroid failure despite the absence of thyroid nodules; its results indicated no evidence for this. (However, as discussed below, some exposed Rongelap people without thyroid abnormalities are now showing evidence of reduced function on the basis of response to TSH stimulation.)

Radioimmunoassays (RIA) for T_4 and TSH have been carried out since 1972 on the exposed Rongelap group and on other people who have had thyroid surgery. The results on subjects with known thyroid lesions are presented in Table 27. Of the 32 subjects tested, 17 or 53% had at least one TSH level above the upper limits of normal (these are in boldface in Table 27). These findings indicate that the residual thyroid tissue is inadequate to sustain euthyroidism in these cases and also reflect inadequate adherence to the prescribed T_4 replacement regimen. Inadequate T_4 replacement is apparently a chronic problem for certain patients (Nos. 5, 23, 33, 65, and 72). Elevations of plasma TSH have been observed also in a number of exposed people without known thyroid lesions (the TSH concentration was >10 μU/ml only in subjects No. 71 and 74 – see Table 28 and Appendix 8). Presumably these subjects, as well as several others with plasma TSH concentration >5 but <10 μU/ml (Nos. 4, 16, 34, 47, 68, and 78), are not receiving the T_4 therapy as regularly as had been hoped. In the Rongelap control group plasma T_4 concentrations were determined in 109 subjects. In those with $T_4 < 5$ μg/dl (6% of those tested) TSH was determined; no elevated levels were found (data not shown). Only 1 of 99 Utirik subjects tested has had an elevated serum TSH level (No. 2232).

These normal findings in the unexposed and Utirik groups suggest that in the irradiated Rongelap group there is impaired thyroid function without palpable lesions which could become symptomatic in the future. The test results probably lead to underestimation of the true incidence of impaired thyroid function, since presumably many of the patients are taking the medication as directed. It should be noted that it is the personal experience of many of the thyroidologists involved in this study that it is extremely difficult to make a clinical diagnosis of hypothyroidism in this population. This difficulty emphasizes the importance of the plasma TSH measurement, which is now recognized as the most sensitive indicator of primary thyroid dysfunction. The status of thyroid function in exposed people without apparent thyroid lesions was further tested with exogenous TSH in 1974, as described below.

3. Thyroid Status of Exposed Rongelap People Without Apparent Thyroid Lesions

In preparation for TSH testing of reserve thyroid function, prophylactic T_4 medication was discontinued for 2 months before the 1974 survey in all exposed subjects without recognized lesions. During the survey, plasma samples were obtained before and 24 hr after intramuscular injection of 10 units of bovine TSH (Thyrotropar, Armour). Both sets were analyzed for T_4 and the first set also for TSH. The results, and thyroxine-binding globulin-binding capacities (TBG-binding capacities) in some cases, are given in Table 28. The mean increment in plasma T_4 following TSH was 2.35 ± 1.2 μg/dl (mean $\pm$ S.D.). The mean T_4 prior to TSH injection was 6.6 ± 1.7 μg/dl. Similar tests[88] on 13 subjects at the University of Pittsburgh showed a mean increment in plasma T_4 of 4.7 ± 1.0 μg/dl, and a baseline plasma T_4 of 7.3 μg/dl which is not significantly different from that of the exposed Rongelap group being tested. Thus, the T_4 response to TSH is significantly less ($p < 0.001$) in this group of 26 exposed Rongelap subjects than in the group of 13 subjects from the United States. Because of the possibility that the smaller increment in plasma T_4 24 hr after TSH in the exposed subjects was due to factors other than decreased thyroidal reserve, TSH stimulation tests were done on 10 euthyroid unexposed Rongelap and Utirik people during a subsequent survey. The mean initial plasma T_4 in this group was 6.0 ± 1.7 μg/dl; and the mean increment 24 hr after TSH injection was 4.2 ± 1.3 μg/dl, significantly greater ($p < 0.001$) than in the exposed subjects.

These results and the finding of elevated plasma TSH levels suggest that there is underlying, clinically inapparent, thyroid damage in the exposed Rongelap population. While it is conceivable that the T_4 replacement program may have led to decreased thyroid reserve, the test results indicate a need for continued close follow-up of the exposed

population. The findings also support the program of thyroxine replacement in the exposed Rongelap population to prevent both clinical hypothyroidism and the associated rise in plasma TSH, which may be tumorigenic.

4. Thyroid Function in the Utirik and Unexposed Rongelap Groups

During the 1973 survey, T_4 and TSH concentrations were measured in plasma from 109 unexposed Rongelap and 97 exposed Utirik subjects (see Table 29). The mean T_4 in the unexposed Rongelap group, 7.7 μg/dl, was the same as in the United States, and, as in the United States, the plasma T_4 concentrations were skewed to the upper range. In the Utirik group, the mean T_4 was significantly lower (6.0±1.8 μg/dl). The frequency distribution calculations (see Table 29 and Figure 39) indicate that a substantial portion of the Utirik population has plasma T_4 concentration <5 μg/dl. Measurements of plasma triiodothyronine (not shown) showed similar reductions in those patients with low T_4 values. Plasma

Table 29

Plasma Thyroxine Concentrations (Mean ± S.D.) in Utirik and Unexposed Rongelap Populations, March 1973

Group	Plasma T_4, μg/dl	Subjects with value <5.0	Subjects with value >11.3
109 Rongelap unexposed	7.7±2.0	6%	6%
97 Utirik exposed	6.0±1.8*	32%**	6%

*Significantly different, $p<0.001$.
**6% had values <3.8.

Table 30

Thyroxine-Binding Globulin (TBG) Levels (Mean ± S.D.) in Serum, 1973

Group	TBG by RIA		TBG-binding capacity	
	n	mg/dl	*n*	μg T_4/dl
Rongelap				
Exposed	12	1.45±0.54	14	21.5± 6.9
Unexposed	13	1.82±1.22	11	25.3±15.7
All sera	25	1.64±0.95	25	23.1±11.5
All sera*	22	1.34±0.45	21	19.1± 6.2
Utirik				
All sera	22	1.40±0.42	31	18.9± 7.8
All sera*	22	1.40±0.42	27	16.4± 4.3

*Omitting sera with TBG>3 or TBG-binding capacity >30.

Statistical comparisons revealed no significant difference ($p>0.05$) between any of the groups.

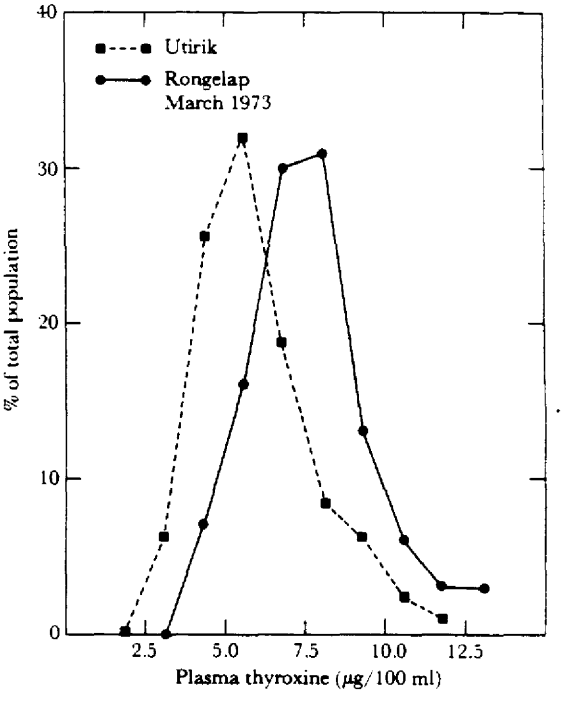


Figure 39. Percentages of people in Utirik and in Rongelap unexposed groups having given thyroxine levels, 1973.

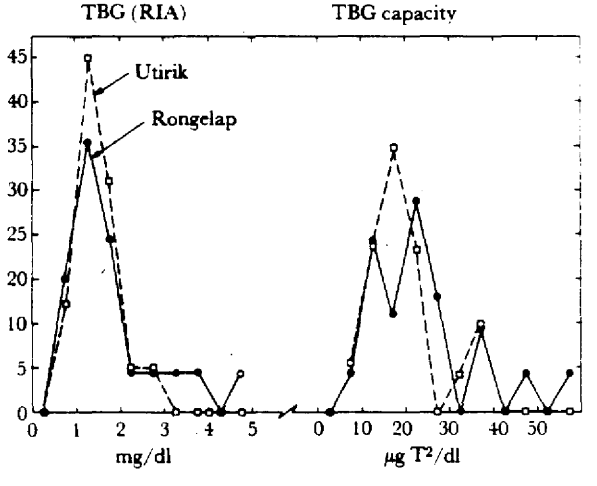


Figure 40. Thyroxine-binding globulin levels in serum and TBG-binding capacity, 1973.

TSH concentrations were normal. For this reason, TBG-binding capacities were determined in a number of subjects with abnormal T_4 values.

TBG analyses were done on sera from 56 subjects (25 Rongelap and 31 Utirik) selected as representative of three groups: people with low, normal, or high plasma T_4 concentrations. Two methods were used: TBG was measured by radioimmunoassay (M.C. Gershengorn and J. Robbins, unpublished observations, 1974) and TBG-binding capacity by reverse-flow paper electrophoresis. The results were comparable since the mean ratio of TBG-binding capacity (μg T_4/dl) to TBG (mg/dl) was 13.9. On the basis of one mole T_4

Table 31

Total and Free Thyroxine, Thyroxine-Binding Globulin, and Thyrotropin Levels in Marshallese

Subject No.	TBG, μg/dl	T_4, μg/dl	$DFT_4 \times 10^4$	Free T_4, ng/dl	TSH, μU/ml
Subjects with TBG-bind capacity <14 μg T_4/dl					
827	7.6 (1.06)*	5.1	3.37	1.72	–
917	7.0 (1.20)	4.4	3.53	1.55	3.1
981	8.0 (0.69)	4.4	3.24	1.43	2.3
2115	7.7	4.4	3.21	1.41	<1.0
2126	9.1	4.9	3.20	1.57	<5.0
2144	7.0	4.0	3.76	1.50	–
2174	7.1	4.1	4.23	1.73	<2.5
2193	11.1 (1.03)	4.8	3.23	1.55	<1.0
2220	13.7	3.2	3.12	0.998	4.2
2248	11.9 (0.94)	3.8	2.94	1.12	3.6
2185	–	4.6	2.80	1.29	<2.5
Mean	9.02	4.34	3.33	1.44	
S.D.	±2.39	±0.543	±0.40	±0.230	
Subjects with TBG-binding capacity 14 to 26 μg T_4/dl					
850	14.0 (1.04)	4.6	3.08	1.42	2.6
2135	15.5	6.1	2.94	1.79	2.1
2137	19.9 (1.37)	9.2	2.90	2.67	2.6
2212	20.1	5.0	1.84	0.92	<2.5
2206	17.7	7.4	2.77	2.05	2.8
2182	14.4	5.0	2.92	1.46	<2.5
2218	14.3	3.0	3.04	0.912	3.6
2221	15.6	4.8	2.68	1.29	<5.0
2232	16.2 (1.54)	4.8	2.42	1.16	6.0
2257	17.3	8.8	2.94	2.57	2.0
2168	14.1	4.2	2.72	1.14	<2.5
2136	15.5 (1.06)	7.7	2.15	1.66	2.1
2230	21.9 (1.28)	6.1	1.45	0.885	4.9
Mean	16.65	5.9	2.60	1.53	
S.D.	±2.58	±1.87	±0.503	±0.597	
Subjects with TBG-binding capacity >26 μg T_4/dl					
2138	37.0 (1.52)	9.0	2.01	1.81	2.4
2229	35.7	5.9	1.53	0.903	10.8
891	56.1 (4.56)	12.9	1.22	1.54	2.1
911	47.6 (3.92)	13.6	0.872	1.19	–
2217	32.8	7.0	1.88	1.32	2.7
2256	51.8	18.2	0.991	1.80	4.4
Mean	43.5	11.05	1.42	1.43	
S.D.	±9.62	±4.63	±0.468	±0.358	

*TBG by RIA, mg/ml.

bound per mole TBG, the expected ratio is 777000 μg/57000 mg = 13.6. The results of these analyses are summarized in Table 30 and Figure 40. There was no significant difference in TBG between the Rongelap and Utirik groups or between the exposed and unexposed Rongelap groups. Eight subjects had high TBG levels. These were all females, and five were known to be pregnant, a condition that can elevate the serum TBG.

The results of these analyses provide no explanation for the difference in serum T_4 concentrations between the larger groups of Rongelap and Utirik subjects; however, these results are based on highly selected sampling and therefore are not representative of the island groups as a whole. Examination of individual Utirik and Rongelap plasmas suggested that low T_4 concentrations were associated with relatively low TBG values. To study the interrelationship of T_4 binding and TBG levels in greater detail, the dialyzable fractions of T_4 (DFT_4) were determined in a number of plasma samples with low, normal, or elevated TBG-binding capacities. The results are presented in Table 31. In subjects with low TBG, the mean DFT_4 was greater than in subjects with normal or elevated TBG. The absolute free T_4 was not different in the three groups, which indicated that the abnormal T_4 levels among these subjects reflect alterations in plasma hormone binding rather than in thyroxine production rates. Since virtually all the subjects with low plasma T_4 concentrations in the larger Utirik and Rongelap groups had normal plasma TSH, we would expect a more systematic study of TBG levels by RIA to show a higher frequency of low TBG levels in the Utirik group. Such a study is currently in progress.

F. IODOPROTEIN STUDIES

Analyses for protein-bound iodine in sera of Marshallese people revealed a level significantly higher than that seen in North America.[18,20] Subsequent studies showed that this elevation could be attributed to an increase in the iodoprotein fraction and indicated that this was responsible for incorrect evaluation of thyroid function during the early years of the surveys. This unusual finding prompted us to determine serum iodoprotein levels in several other Pacific Islands groups (see Table 32). Except for a group of Americans living on Kwajalein Atoll, these groups also exhibited high serum levels of iodoprotein. The Maui group was comprised of persons of almost pure Hawaiian ancestry, some of whom ate seaweed in large quantity, but the iodoprotein level did not correlate with this dietary intake. In the Rongelap population, it is of interest that the iodoprotein level remained high in two athyreotic boys; after subtotal thyroidectomy; and during thyroid suppression by thyroxine administration.[20] This strongly implies an extrathyroidal (endogenous) or a dietary (exogenous) source for the iodoprotein. The cause of the elevation, however, has not been ascertained. One possibility is that it is produced in polymorphonuclear leukocytes. These cells are known to organify iodine during phagocytosis.[91] Although the Marshallese do not have unusually high leukocyte counts, they do have a mild eosinophilia

Table 32

Serum Iodoprotein Levels (μg %) in Pacific Island Populations and in Americans

Group	No. in group	Total I	T_4 I	Iodoprotein
Rongelap exposed				
No thyroid nodules	10	7.2±2.4	4.0±1.0	2.5±1.1
Thyroid atrophy	2	2.8±0.4	1.2±0.5	1.3±0.0
Thyroid nodules, pre-op	7	8.3±3.6	3.6±1.1	4.2±3.9
After partial thyroidectomy	9	5.5±1.1	4.4±1.8	2.6±2.0
After total thyroidectomy	3	6.0±1.1	2.6±0.1	2.9±1.2
Ailingnae	4	7.8±1.2	3.7±0.8	3.3±1.7
Utirik	5	16.1±8.9	3.8±0.8	3.3±1.7
Rongelap unexposed	24	8.7±2.8	4.0±1.0	4.6±3.5
Kapingamarangi	23	11.5±3.0	6.2±2.6	3.0±0.6
Pingalap	27	8.4±2.0	3.6±0.6	3.3±1.9
Hana (Maui)	13	6.7±1.3	4.1±1.1	2.2±0.8
Americans: Kwajalein	12	6.6±1.3	3.9±1.1	1.9±0.5
U.S.	9	6.4±1.3	4.1±0.5	1.1±1.0

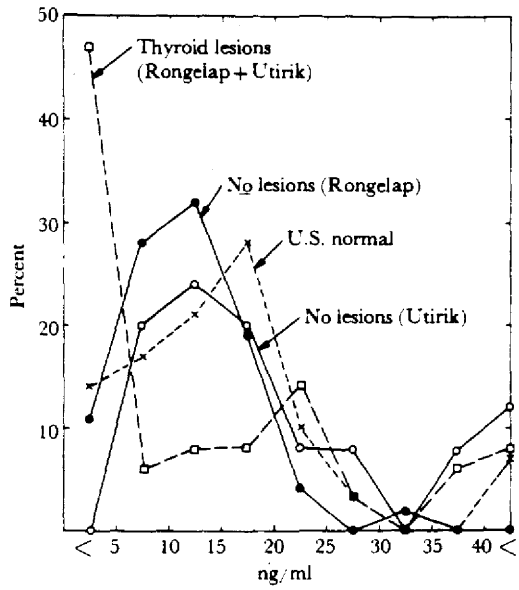


Figure 41. Percentages of people in various groups having given levels of thyroglobulin (by RIA), 1973.

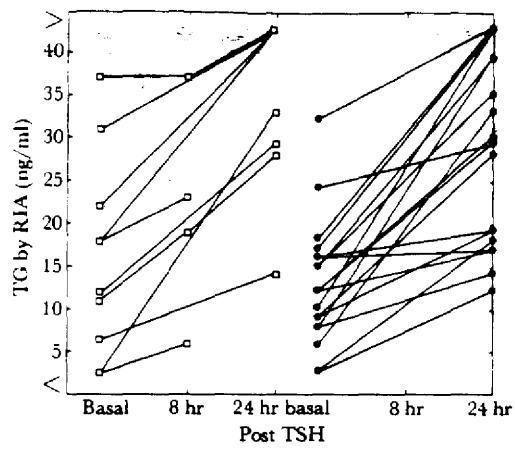


Figure 42. Effect of TSH administration on thyroglobulin levels, 1973. □, Persons with thyroid lesions (Rongelap plus Utirik); ●, without lesions (Rongelap).

and a high incidence of parasitic infections. A preliminary examination of North Americans with abnormal leukocyte counts, however, did not show a correlation between leukocytosis and iodoprotein level. Further studies on this are indicated. The chemical nature of this iodoprotein is also unknown. By analogy with findings in various thyroid diseases,[92] the iodoprotein is likely to be comprised mainly of iodoalbumin arising from the iodination of serum proteins.

It is now recognized, however, that thyroglobulin (TG) is a minor component of normal plasma. At a reported concentration of 5.1 ± 0.49 (S.E.M.) ng/ml (range <1.6 to 20.7) in normal North Americans,[93] and assuming an iodine content of 0.5%, this would be equivalent to an iodoprotein iodine level of 2.6 ± 0.25 ng/dl. Although it seems unlikely, *a priori*, that circulating thyroglobulin in the Marshallese could be elevated enough to give an iodoprotein level of 3 to 4 μg/dl (i.e., >500 μg TG/dl), the possibility was investigated by radioimmunoassay measurements (M. Izumi, J. Baulieu, and J. Robbins, unpublished observations, 1974; see Figure 41). The assay could detect TG levels >5 ng/ml; levels >40 ng/ml were not quantitated.

In the Rongelap and Utirik groups without thyroid lesions (47 and 25 subjects respectively), $\geqslant 80\%$ of the values were within the U.S. range, and no correlation was seen between elevated serum iodoprotein and abnormal TG levels. A few members of each group, including the U.S. normal group, had TG values >30 ng/ml, but the significance of this is uncertain. Of 24 subjects with elevated serum iodoprotein, only 1 had serum TG >30 ng/dl.

A striking finding (Figure 41) was that in the Rongelap plus Utirik group with thyroid lesions (36 people) almost 50% of the levels were <5 ng/ml, a much higher percentage than in the other groups. Most of these people had had prior thyroid surgery or were athyreotic, and it is presumed that they had insufficient thyroid tissue for normal TG production. Furthermore, T_4 suppression therapy may have contributed to the low TG levels in the Rongelap people.

TG was also measured before and after TSH injection in 10 Rongelap plus Utirik subjects with thyroid lesions and in 20 Rongelapese with none (Figure 42). In every case, TSH resulted in a rise in TG level, and there was no apparent difference between the two groups.

The unusually high level of iodoprotein in the Marshallese people is intriguing, and further studies are in progress, with ^{129}I used as the tracer, in an attempt to identify the protein.*

G. CORRELATION OF THYROID ABNORMALITIES WITH RADIATION EXPOSURE

Statistics on the incidence of thyroid abnormalities in people living on the Marshall Islands are

*Miss R.F. Straub, BNL, is doing the chemical analyses.

not very reliable. From our surveys of the unexposed people of Rongelap and Likiep (a nearby atoll not exposed to fallout), the incidence appears to be about 5%, mostly present in older age groups (see Figure 29 and Table 25). We have seen a few cases of hyperthyroidism and myxedema at the Majuro Hospital, but no statistics are available. Two cases of thyroid cancer are reported from hospital admissions for a 10-year period (1952-1962) for the Marshall Islands, the population varying between 15,000 and 20,000 during that period. This has been estimated by Trust Territory medical personnel to be about half the actual number of cases (therefore 8 cases per 20,000 people per 20 years was used for statistical comparisons).

Data on iodine intake and excretion in the Marshallese (tabulated below and presented in detail in Appendix 9) indicate that iodine-deficiency goiter would not be expected in this population.

Sample	Iodine, av. μg/day (range)
28 Urines (1965)	105 (19–279)
19 Urines (1974)*	127 (25–266)
7 Diets (1974)*	70 (48–152)

The urinary iodine excretion is somewhat lower than the U.S. mean of 190 μg/day (18–483) in 1941.[94] On the basis of the few diets analyzed, the daily iodine intake seems to be within the recommended range of 50 to 75 μg.[95] These iodine levels are somewhat lower than would be expected in an oceanic population but are much higher than seen in areas of endemic goiter.[96] The Marshallese diet contains no know goitrogens, and diffuse goiters (typical of endemic goiter) have not been observed.

The high incidence of both benign and malignant thyroid nodules in the exposed Rongelap people appears to be clearly related to radiation exposure with a large component due to radioiodine in the fallout. Numerous studies on animals have shown that thyroid neoplasia follows exposure both to x-irradiation and to radioiodines.[97-99] Such tumors may be benign or malignant and appear to be dose-dependent to some degree. The incidence of thyroid tumors is increased in Japanese atom bomb survivors[100-103] (Figure 43). There is a considerable amount of data showing that children who were given radiation to the head and neck region for treatment of thymic hypertrophy, acne, and fungus of the scalp have an increased incidence of both benign and malignant thyroid lesions in later years.[105-112] Reports of tumorigenic effects of radioiodine in man are more limited. Sheline et al.,[113] in their follow-up study of 250 patients treated for hyperthyroidism, reported 8 having nodular goiter, of whom 6 had been irradiated at age <20 and 4 at age <10. More recently a number of cancers of the thyroid have been reported in patients previously treated with radioiodine for hyperthyroidism.[114-116] The number of such cases reported is, however, lower than might be expected on the basis of the widespread use of ^{131}I, perhaps because the cells are more likely to undergo lethal damage.[117-122]

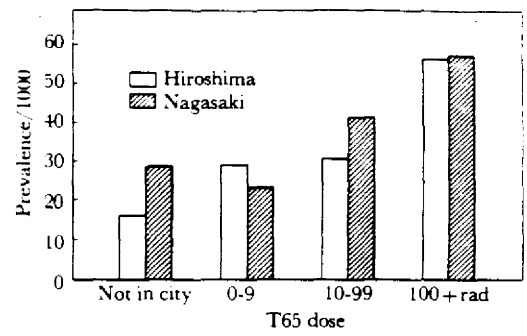


Figure 43. Prevalence of diseases of the thyroid, fifth examination cycle, by radiation dose and city for females age 0 to 19 at time of bomb. (From Belsky et al.[104])

In the more heavily exposed Rongelap group the adult thyroids received a dose (335 rads) about twice that to the whole body and those of small children (700 to 1400 rads) about 8 times that to the whole body. On the basis of the incidence of benign nodules in the unexposed Marshall Islands populations examined, about 3 to 4 cases would be expected during the 20 years in the Rongelap exposed group, whereas 24 occurred. In the Utirik group about 6 would be expected, and 6 occurred. Regarding cancer of the thyroid, on the basis of Marshall Islands statistics, about 0.033 cases would be expected in the Rongelap group over the 20-year period, whereas 3 occurred. In the Utirik population about 0.06 cases would be expected, and 1 occurred; in view of the low dose of radiation it is unlikely that this case is radiation induced.

Tables 33 and 34 show the incidence and the risk per rad in the Marshallese compared with that in other populations for both benign and malignant thyroid neoplasms. Data on benign thyroid nodularity are scarce, but the incidence in

*The iodine analyses were done by M.T. Kinsley and D.F. Leahy at BNL.

Marshallese children appears similar, on a risk per rad basis, to that in the x-rayed children studied by Hempelmann.[107,109] Risk for thyroid cancer in the Rongelap people is also similar to that reported for other populations.

The data in Table 25 indicate that at the dose levels involved there is a correlation between incidence of thyroid lesions and the estimated dose to the gland. On the basis of the incidence per rad in the high exposure Rongelap children, about 2 children with adenomas would be expected in the Ailingnae group, where 2 were noted, and 4 in the Utirik group, where none was found. The lower effectiveness per rad in the Utirik children may be

Table 33

Percent Incidence of Thyroid Lesions (20 Years Observation)

	Age at exposure			
	⩽10 yr		>10 yr	
Group	Benign	Cancer	Benign	Cancer
Rongelap	84.2	5.3	11.1	4.4
Ailingnae	33.3	0.0	33.3	0.0
Rongelap & Ailingnae	76.0	4.0	15.8	3.5
Utirik	0.0	0.0	5.1	1.0
X-rayed children (17 yr)[107]	28.0	4.3		
X-rayed children, low dose (17 yr)[107]	3.0	0.13		
Marshallese controls	1.0	1.8×10^{-5} (all ages)	5.4	1.8×10^{-5} (all ages)
Worldwide av.[108]		2.5×10^{-5} (all ages)		2.5×10^{-5} (all ages)
United States[107]	0.36–1.7		0.5–1.6	

Table 34

Risk* of Radiation-Induced Thyroid Lesions (Cases per 10^6 man-years per rad)

	Age at exposure					
	⩽ 10 yr			> 10 yr		
Group (years follow-up)	Dose range, rads (type)	Benign	Cancer	Dose range, rads	Benign	Cancer
Rongelap (20)	710–1150 ($I\beta,\gamma$)	41.9	2.6	379	10.5	7.0
Ailingnae (20)	280– 450 ($I\beta$, γ)	43.6	0	135–190	151.1	0
Rongelap & Ailingnae (20)	280–1150 ($I\beta$, γ)	42.1	2.3	327	22.5	6.4
Utirik (20)	60– 95 ($I\beta$, γ)	0	0	31	89.2	17.8
Rochester (17)[107]	335 (av.) (x rays)	64.0	5.5			
Ann Arbor (17)[107]	20 (av.) (x rays)	24.0	2.2			
Beach & Dolphin (20)[123]	(x rays)		1.7			
UNSCEAR (17)[124]	100– 300 (x rays)		0.5–1.5			
ABCC (20)[101]	20–1000 (γ, n)		1.3 (all ages)			
ABCC (20)[101]	<20 (γ, n)		0.2 (all ages)			

*Risk is calculated from the equation

$$\text{Risk} = \frac{\text{No. of cases} \times 10^6}{\text{dose} \times \text{years at risk}},$$

or, alternatively,

$$\text{Risk} = \frac{\text{No. of cases} \times 10^6}{\text{No. of subjects} \times \text{mean dose} \times \text{mean No. of years at risk}}.$$

Both equations give almost identical results for the Marshallese because of the uniformity of the data. No correction has been made for expected number of cases because the expectation among unexposed subjects is too low to affect the results (see Table 33).

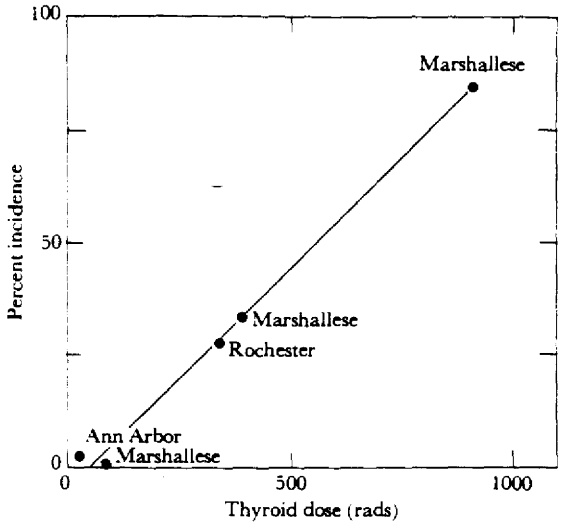


Figure 44. Incidence of benign thyroid nodules.

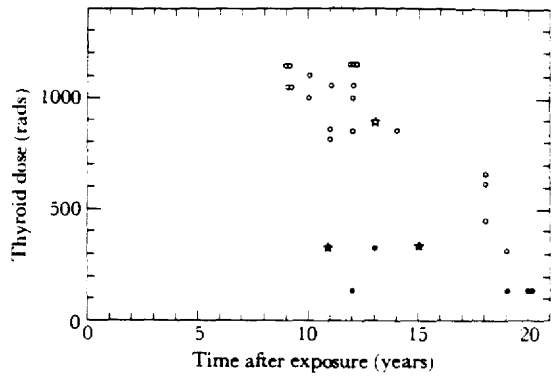


Figure 45. Time of appearance of thyroid lesions in Marshallese. Exposed at age <10: ○, benign; ☆, malignant. Exposed at age >10: ●, benign; ★, malignant.

partly related to the smaller contribution to the dose from short-lived isotopes of iodine. Nevertheless the Ann Arbor children in Hempelmann's study[107] had an increased incidence of thyroid tumors following a mean dose of only 30 rads to the gland (Figure 44), and more recently Modan et al.[110] and Harley et al.[111] have reported an increased incidence of thyroid tumors in children who had received about 6.5 rads to the thyroid gland during x-ray treatment of the scalp for fungus infection. Modan et al. state that "one would strongly suspect a mini-epidemic of thyroid adenoma" in the group studied. In considering the risk data of Hempelmann's and Modan's groups, the large element of Jewish people (who have increased susceptibility to thyroid tumors) should be kept in mind. Also, Hempelmann's were irradiated as infants and therefore represent only a narrow age range.

1. Latent Period

In Figure 45 the development of thyroid abnormalities is plotted according to radiation dose and time after exposure; the latent period appears to be longer with lower doses. Figures 46 and 47 show the relationship between dose and age at development of thyroid lesions. These data indicate that the radiation-induced lesions occur at earlier ages in the exposed Rongelap people than in the Utirik or unexposed groups. In the latter groups almost all the thyroid nodularities develop in the older people. Recent data of Hempelmann et al.[109] indicate that the latent period may be >30 years for development of radiation-induced thyroid tumors. Thyroid cancer has been reported as long as 40 years after radiation exposure.[125]

Correlation with sex showed that slightly more exposed Rongelap females (18 of 45) developed thyroid lesions than did males (11 of 41). All three cases of cancer of the thyroid were in females.

The thyroid gland is generally thought to be more sensitive to radiation during childhood.[120,121] Pochin[118] estimates a percent incidence per 100 rads of 0.5 to 1.0 for adults and 1.3 for children. Certainly the largest number of benign lesions occurred in the Marshallese children. This is probably due mainly to the smaller size of the thyroid glands resulting in larger doses per gram of gland. Doniach[120] theorizes that thyroid tumors are produced by an initiating factor (radiation) plus a promoting factor (TSH) which increases mitosis and enhances the expression of a possible malignant clone. In addition, in children the growth factor may be important, since at maturity the thyroid weighs about 20 times as much as at birth. These factors would increase the chances for malignant transformation. The lower incidence of cancer of the thyroid in children than in adults may be related to the lack of dose dependence of the carcinogenic effect of radiation at high doses. Several investigators have shown that high doses of radiation (both x rays and radioiodine) are associated with a lower incidence of thyroid malignancy than lower doses, possibly because the high doses produce enough cell destruction to preclude malignant transformation.[85,120,121,126,127] This may be the case with Marshallese children. It has been noted that following treatment of hyperthyroidism with large doses of radioiodine, although

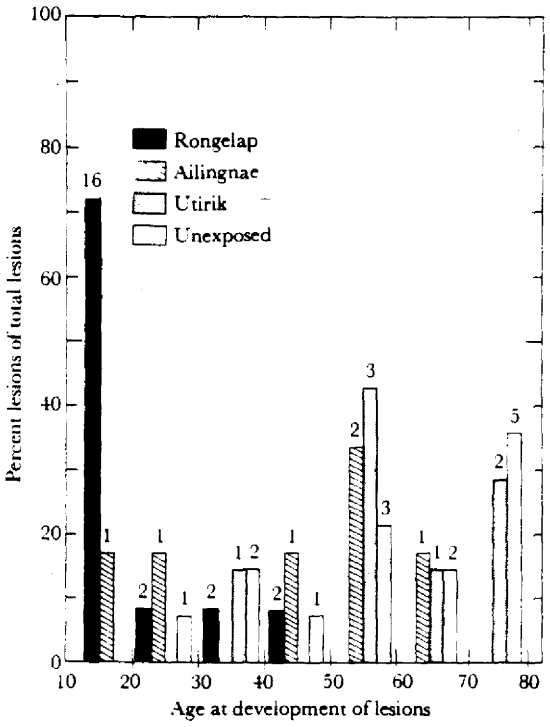


Figure 46.

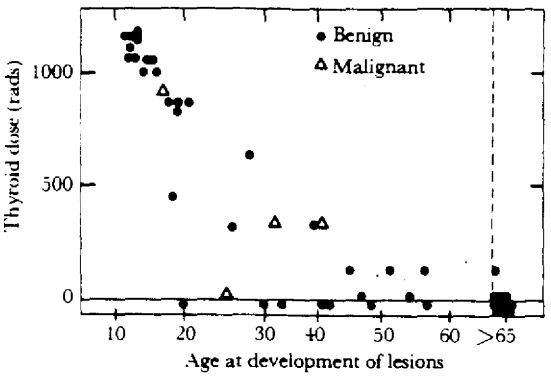


Figure 47. Relationship of thyroid dose to age when lesions develop in Marshallese. ●, Benign; △, malignant.

hypothyroidism develops, the cases of thyroid cancer are fewer than expected.[120,121,128,129] The development, in the two stunted Marshallese boys, of thyroid atrophy with hypothyroidism but without the development of tumors is in line with this reasoning. Offsetting, somewhat, the greater incidence of thyroid cancer in children is the finding that children survive longer than older people, even with the well differentiated types.[130]

The possible effects of the stress of puberty in the development of thyroid lesions have been previously noted.[23] The stress of frequent pregnancies, which had occurred before the development of malignant lesions in the three Rongelap women, may have been a factor in development of neoplasia. Both these correlations, however, may be fortuitous.

2. Comparison of Thyroid Neoplasias From X-Ray Radiation and Radioiodine Irradiation

The data in Tables 33 and 34 show that the risk per rad for the development of thyroid neoplasms in the Marshallese was quite similar to that in populations exposed to x-irradiation. The data in Figure 44 indicate a linear relationship between nodularity in the Marshallese children, who received their dose largely from radioiodines, and the groups receiving x-irradiation. These data are not extensive enough to show possible threshold effects. Since in animal experiments ^{131}I is only about $\frac{1}{10}$ to $\frac{1}{15}$ as effective as x-irradiation in producing thyroid tumors,[85,120,129,130] why do the Marshallese data indicate near equality of effect? It is estimated that thousands of children have received diagnostic ^{131}I (20 to 50 μCi) in the past resulting in thyroid doses up to hundreds of rads, yet only 1 case with thyroid tumors has been reported.[131,132] U.S. Public Health Service workers recently reviewed a large number of case histories of people who had received radioiodines for treatment of hyperthyroidism.[122] They were unable to show any clear-cut increase in incidence of thyroid tumors in this group compared with a group treated by surgical thyroidectomy. Increasing numbers of the patients treated with ^{131}I developed varying degrees of hypothyroidism in later years. The low incidence of tumors following such treatment may be related to the high doses of radiation given to the thyroid, sufficient to destroy its regenerative capacity.

It should be noted, however, that in the past few years a number of thyroid malignancies have been reported following radioiodine therapy for hyperthyroidism.[114-116] The number of cases reported is lower than expected on the basis of dosage to the thyroid. The increased tumorigenesis in the Marshallese may be related to the nature of the radiation, more than half the dose being due to short-lived isotopes of iodine (particularly ^{132}I, ^{133}I, and ^{135}I), which are more energetic (see Appendix 9C). Vasilenko and Klassovskii[133] have demonstrated that when these shorter-lived isotopes of iodine are combined with ^{131}I the tumori-

genic effect in rats and dogs is equal on a rad basis to that of x rays. Walinder et al. have reported similar findings.[134,135] Dunning,[136] in estimating thyroid dose from absorption of radioiodines in fallout, considers the shorter-lived isotopes to be 4 times as energetic as ^{131}I. The more energetic beta radiation with a higher dose rate and longer range gives a more uniform tissue distribution of radiation than is produced by ^{131}I. This might explain why the incidence of thyroid lesions in the children receiving 1000 rads falls on a line corresponding to 1000 rads of x-radiation (Figure 44), or it might indicate that the radioiodine exposure was about 10 times as effective as ^{131}I alone would have been (see Cole[137]). Thus the two boys with atrophy of the thyroid may have had doses to the gland comparable with 4000 to 6000 rads or more of x-irradiation.

3. The Development of Thyroid Abnormalities From *In Utero* Exposure

The development of thyroid adenomas in one of the Marshallese boys exposed *in utero* is of interest. Four children were exposed *in utero:* in the higher radiation group, one at the end of the second trimester and two at the end of the first trimester; in the lower dose Rongelap group, one at the end of the second trimester. Only the boy in the higher dose group and exposed in the second trimester has developed thyroid lesions. Several cases have been reported of children who had myxedema following *in utero* exposure during treatment of the mother with large doses of radioiodine.[138-141] However, this Marshallese boy appears to be the first reported case of thyroid adenomas developing presumably from *in utero* exposure to radioiodines.

In utero thyroid dose calculations are not possible, since insufficient data are available on fetal thyroid uptake of radioiodines from the mother at various stages of gestation.[142] Figure 48 shows thyroid function of the human fetus. The gland is not thought to begin to function and to accumulate iodine until about the 12th week of gestation.[144,145] At the time of exposure of the Marshallese boy (about 22 to 24 weeks) the thyroid should have been actively functioning. He received 175 rads of gamma radiation but the dose from radioiodines is uncertain. In view of the high uptake of radioiodines per gram by the fetal thyroid at 22 weeks, the thyroid dose might be expected to be high. However, since it appears from our data that

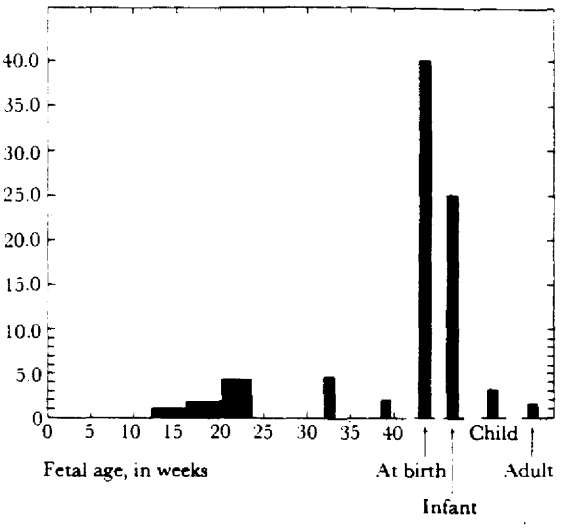


Figure 48. Thyroidal uptake of ^{131}I (% per gram) versus age. (From Evans et al.[143])

longer latent periods are associated with lower doses of radiation to the thyroid, it is likely that the thyroid in this boy exposed *in utero* received a lower dose than that in Marshallese children exposed at a young age. Also, this boy and the others exposed *in utero* have shown no impairment in growth and development. It is of interest that the mother of this boy has not developed any thyroid abnormalities and appears to be euthyroid. The two children exposed at the end of the first trimester probably had nonfunctioning primordial thyroid glands at that time so that the glands received only the gamma exposure and were therefore less likely to develop thyroid abnormalities. None of the four children exposed *in utero* has shown microcephaly or mental retardation, which have been noted in some of the children exposed *in utero* in Japan.

V. Neoplasia

Among the late effects of radiation, the enhancement of the development of neoplasia has been well documented. In irradiated animals, malignancies not only may be induced at an earlier age but appear in increased numbers. In the Japanese atom bomb survivors, leukemia and thyroid neoplasia have shown the most clear-cut association with radiation exposure, though more recently

Table 35

Cancer Cases Among Marshallese
up to 20 Years Post Exposure

Case No.	Age and Sex	Type	Year of death
Exposed Rongelap (82 people)			
62	60 F	Ovarian	1959
30	60 F	Cervix*	1962
13	71 F	Uterus*	1956
68	64 M	Stomach	1974
54	18 M	Leukemia	1972
18	35 F	Thyroid	
64	41 F	Thyroid	
72	22 F	Thyroid	
Unexposed Rongelap (~190 people)			
861	68 F	Cervix*	1960
Exposed Utirik (157 people)			
2122	87 M	Rectum*	1959
2229	37 F	Thyroid	

*Diagnosis not confirmed by autopsy.

other forms of malignancy are beginning to show correlations.[177]

The cases of malignancy recorded among the various Marshallese populations under study during the past 20 years are listed in Table 35. In these people, thyroid malignancies (discussed above) show a correlation with radiation exposure; other types cannot be ascribed definitely to radiation exposure.

A. A CASE OF ACUTE MYELOGENOUS LEUKEMIA

In 1972 an exposed Rongelap male (subject No. 54) died of leukemia at age 19.[146] He had been exposed to 175 rads of gamma radiation on Rongelap at age 1 year, and had experienced early transient symptoms, nausea and vomiting and itching and burning of the skin. He showed moderate depression of lymphocytes, platelets, and neutrophils, his WBC dropping to 3900 by 6 weeks and his platelets to 140,000 by 4 weeks. He developed beta burns of the skin, particularly over the neck, arms, and legs, and some epilation of the scalp (Figure 49). These lesions healed uneventfully. His blood elements showed slow recovery toward normal levels by 1 year. He remained generally healthy, with usual childhood infections, until age 13, when nodules developed in the thyroid and he was taken to the U.S. for study (Figure 50). The nodules removed at surgery (including a Hürthle cell adenoma) were benign. He was placed on continuous thyroid hormone treatment and remained euthyroid, with normal growth and development. (His mother, father, and two brothers also had thyroid lesions surgically removed.)

Figure 49. Subject No. 54 at age 1 year, a month after exposure to fallout, when he had spotty epilation and scattered beta burn lesions on the scalp, neck, arms, legs, and anal region.

Figure 50. Subject No. 54 being examined at age 13.

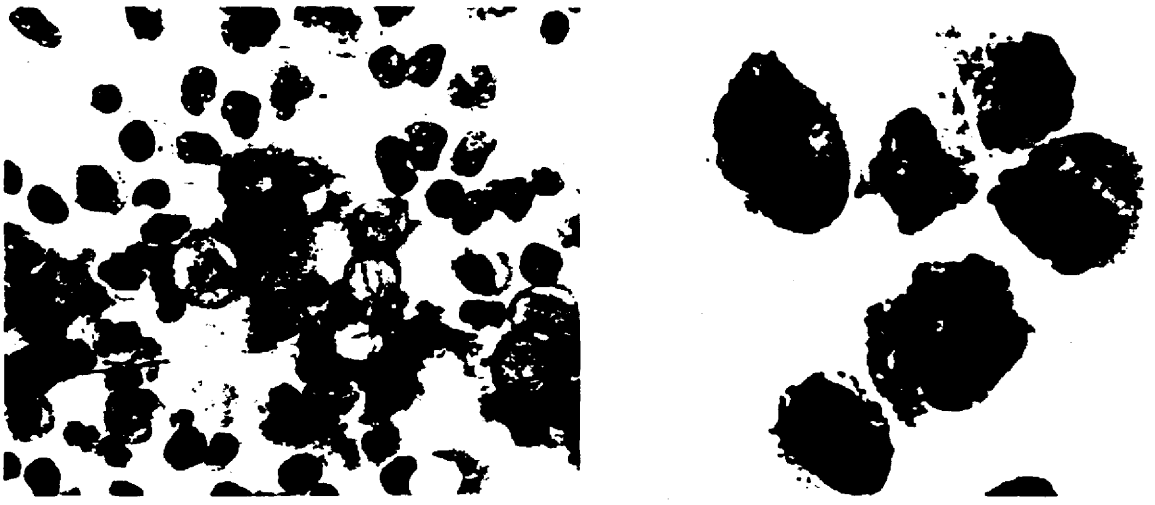


Figure 51. *Left*: Immature myelocytic cells (many promyelocytes) in marrow of subject No. 54, 6 weeks before death. ×1650. *Right*: Promyelocytes with Auer rods were numerous. ×1650.

During the 1972 annual examination the subject was found to have a WBC of 2000 and a platelet count of 120,000. He showed no other significant findings and appeared to be a healthy and husky 19-year-old. He was brought to BNL, where a bone marrow examination revealed typical acute myelogenous leukemia, predominately of the promyelocytic type with numerous Auer rods (see Figure 51). He was transferred to the National Cancer Institute at Bethesda, where examination resulted in few physical findings besides the bone marrow changes noted.* He had a slight inflammation of the pharynx, periodontitis around the right third molar, and a left chronic otitis media. Antileukemic medication included cytosine arabinoside, 6-thioguanine, heparin, oxacillin or cephalothin with gentamicin, and later ampicillin. He received blood and platelet transfusions, some from an older brother flown in from the Marshall Islands, but compatible platelets were not available. Platelet levels continued low, and within 2 weeks he developed signs of a hemorrhagic diathesis with marked hemithorax. He was placed in a respirator, but his condition worsened, and he died 6 weeks after admission. (See Appendix 11 for hospital summary.)

The principal autopsy findings included acute myeloblastic (promyelocytic?) leukemia in spleen, lymph nodes, arachnoid, and bone marrow. There had been massive pulmonary hemorrhage bilaterally and inflammation of the parotid gland. It was

*Drs. Edward S. Henderson, James Mabry, Joan Bull, Maria Tomaszewski, and Jacqueline Peng of the National Cancer Institute and Dr. Gundabhaktha Chikkapa of Brookhaven National Laboratory assisted with this study.

Table 36

Difference in Mean Peripheral Blood Levels Between Subject With Leukemia (No. 54) and Exposed Peers

	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Plate. ($\times 10^{-3}$)	Hct., %
1954					
Exposed peers	7.7	4.1	3.0	246	37.3
Leukemia case	5.7 (−26%)	2.5 (−39%)	2.8 (−6%)	187 (−24%)	36.4 (−2.5%)
1955-72					
Exposed peers	8.3	3.8	3.5	293	39.2
Leukemia case	6.5 (−21%)	2.7 (−30%)	3.1 (−11%)	271 (−7.5%)	40.5 (+3%)

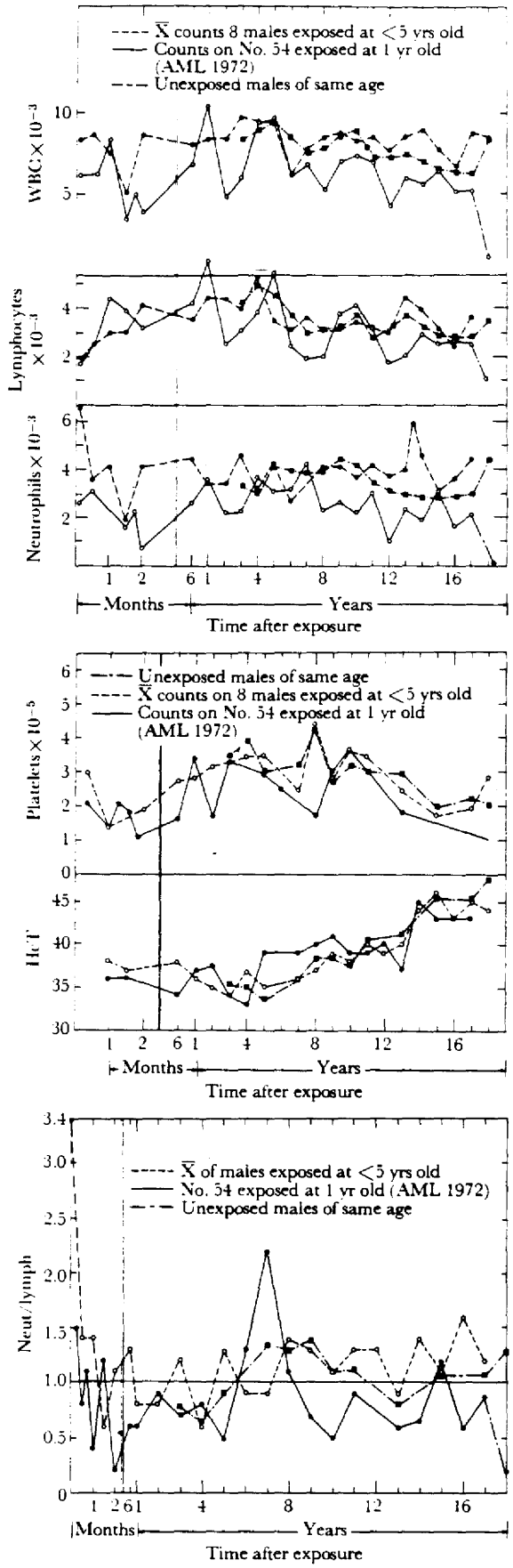


Figure 52. Mean levels of WBC, lymphocytes, neutrophils, platelets, hematocrit, and neutrophil/lymphocyte ratio for subject with leukemia (No. 54, solid line in each case) compared with those of exposed (– – –) and unexposed (— — —) boys in same age range, over 18 years since exposure.

interesting that radiochemical analysis of bone samples showed ^{90}Sr levels about the same as in people living in New York.

Retrospective study of hematological data. The hematological data gathered over the 18-year period on the subject with leukemia (No. 54) were compared with the mean levels of 8 other Rongelap boys exposed at <5 years of age and of 8 unexposed boys in the same age range (Figure 52). The percentage differences in mean peripheral blood levels between subject No. 54 and his exposed peers are given in Table 36 for the first year and for the subsequent years. The subject with leukemia showed greater depression of neutrophils both during the immediate post-exposure period and in subsequent years. The least difference appeared to be in the erythropoietic system. Morphological studies of peripheral blood smears revealed no significant abnormalities until the development of leukemia; unfortunately no bone marrow examinations had been made before.

Since the Marshallese children are prone to frequent infections associated with granulocytosis, it was of interest to compare this response in the subject with leukemia and in the other exposed boys. The records showed that the white cell count exceeded 10,000 a total of 36 times in 131 counts (27%) in the other 8 exposed boys and only once in 18 counts (5%) in subject No. 54. However, if this indicated a reduction in marrow reserve in this boy, the reduction apparently was insufficient to result in any illness serious enough to require hospitalization.

Other data related to leukemia were generally negative in subject No. 54, including alkaline phosphatase levels of neutrophils, basophil counts, Australia antigen, and blood proteins (except for a slightly elevated gamma globulin level, which is common in the Marshallese). Chromosome studies of the peripheral blood made 12 years after exposure and of the bone marrow during his terminal illness did not show aneuploidy or structural changes of the chromosomes. However, increased chromosome breakage (11% of the cells) was noted terminally, which may have been related to radiation exposure to chemotherapy, or possibly to his disease.

Discussion. It is possible that radiation exposure was involved in the etiology of leukemia in subject No. 54. Radiation exposure has long been associated with the development of this disease (in physicians, particularly radiologists,[147,148] in pa-

tients after radiation treatment for ankylosing spondylitis,[149] etc.), and its etiological role was established without question by the high incidence of leukemia in the Japanese exposed to the atom bomb.[150,151]

Some findings in the Japanese tend to support radiation etiology in this Marshallese boy. In his case the latent period was quite long, but the incidence of leukemia in Japan is still (after >25 years) higher among the exposed group than among the general population. Also, the incidence has been higher among those exposed in childhood, particularly males, in whom the granulocytic form of leukemia has been prevalent; and the same form has been more prevalent in cases with longer latent periods and lower exposures.[152] On the basis of 2 cases per year per million people per rad among the exposed Japanese, the expectancy among the exposed Marshallese would be a total of about 0.33 cases. However, the normal incidence of leukemia in the Marshallese, from the sketchy statistics available, appears to be low, about 20 cases per year per million compared with 60 to 70 in the U.S.; thus the number of spontaneous cases expected among the exposed Marshallese would be a total of 0.02 to 0.067. The ratio of radiation-induced to spontaneous expectancy (0.33:0.02 or 0.33:0.067) indicates that chances are about 5 to 15 times as great that this Marshallese case was radiation-induced as that it was spontaneous.

Review of the hematological data on subject No. 54 over the 18-year period before the development of leukemia is of interest. The greater degree of hematologic depression in him than in the other exposed boys indicates either a greater radiation dose to the hematopoietic tissues or greater sensitivity or proclivity to marrow depression. (None of the peripheral granulocyte levels in the other boys, exposed or unexposed, plotted individually, were as low as his.) Perhaps his radiation dose was actually greater, since he was the youngest Rongelap child exposed and may therefore have been in closer contact with the fallout source on the ground. Being younger might also imply greater radiosensitivity.

In view of the later development of the granulocytic form of leukemia, it is of interest that the early bone marrow injury was characterized by having the greatest effect on the granulocytes, with the thrombocytic and lymphocytic cells less affected and the erythrocytic cells least. The erythrocytic system did not show evidence of failure until near death. In spite of the long-term findings, morbid evidence of the disease was not apparent until the last few months. One year before the diagnosis of leukemia the peripheral blood cells appeared normal except for a possible increase in atypical monocytes.

A preleukemic syndrome has been described for some cases of nonlymphocytic leukemias.[153,154] Linman and Saarni[154] state that the frequency of the syndrome may be as high as two cases in five and that preleukemic findings "reflect the early stage of myeloproliferative disorders which will eventually fulfill the criteria to be classified as myelomonocytic leukemias." The syndrome is characterized by various findings, not all necessarily present, such as anemia with erythrocyte abnormalities, thrombocytopenia, atypical platelets in the peripheral blood, and neutropenia.[153,154] Chromosome abnormalities are reported in about half the cases.[153] Isolated neutropenia is an occasional preleukemic finding; in the case reported here, the relative neutropenia is the only finding that might be considered as preleukemic. If the preleukemic phase is considered to date back to near the time of radiation exposure in this case, then it had a longer course than is usually noted.[154]

The possibility that the granulocytic reserve was reduced in subject No. 54 as a result of radiation exposure was indicated by his granulocyte response being lower than that of his peers in response to challenge by childhood infections. One might speculate that the frequent infections to which the Marshallese are exposed may have played a role in accelerating the development of a radiation-induced mutant leukemic clone. This would be similar in some ways to the role thought to be played by the thyroid-stimulating hormone in enhancing the development of tumors in radiation-injured thyroid glands, as has been seen in the Marshallese years after exposure of their thyroid glands to radioiodine from the fallout. The above findings do not rule out the possibility of a viral etiology for this leukemia.

Because of the development of this case of leukemia, complete hematological examinations on the exposed Marshallese population are now being carried out semiannually instead of annually.

B. OTHER MALIGNANCIES

Malignancies other than thyroid cancer and leukemia (see Table 35) appeared to have a higher

incidence in the exposed Rongelap people than in the unexposed group and the low-level exposed Utirik group. Whether or not this is correlated with radiation exposure cannot be ascertained. The data must be interpreted with considerable caution since (a) the populations are small, (b) the unexposed population was not examined before 1957 and has undergone changes due to both attrition and addition, (c) the diagnosis of malignancy is not certain in all cases because of the difficulty of obtaining autopsies for verification, and (d) the types of malignancy were not those that have been correlated with radiation exposure in the Japanese exposed at Hiroshima and Nagasaki.[177]

VI. Radiological Monitoring of Personnel and Environment

A. GENERAL

During their 3-year sojourn on Majuro (1954-1957), the Rongelap people's body burdens of radionuclides decreased rapidly, as shown by radiochemical analyses of urine. By 6 months radionuclides in the urine were barely detectable.[2] The Utirik people were moved back to their home island after the initial examinations and were exposed to very low levels of residual radioactivity there. In 1957 (3 years after the accident) gamma spectrographic analyses were carried out on 4 Rongelap and 2 Utirik people at Argonne National Laboratory in Chicago.[155] The finding of detectable levels of ^{137}Cs and ^{65}Zn (higher in the Utiriks) indicated the feasibility of using this technique in the islands. When the Rongelap people returned to their home island in 1957, the low levels of environmental contamination were soon reflected in increased body burdens of some radionuclides.[7] A number of radiological surveys[156-164] at Rongelap and Utirik have been carried out in conjunction with personnel monitoring, largely by University of Washington staff and more recently also by a group from the BNL Health Physics and Safety Division. These studies have provided important information on the movements of radionuclides from the soil through the marine and plant food chain to man and should prove useful in predicting future body-burden patterns of people returning to Bikini and Eniwetok. The principal residual radioactive elements on Rongelap and Utirik were ^{137}Cs, ^{90}Sr, ^{65}Zn, and ^{55}Fe, with small but measurable amounts of other fission products and neutron-

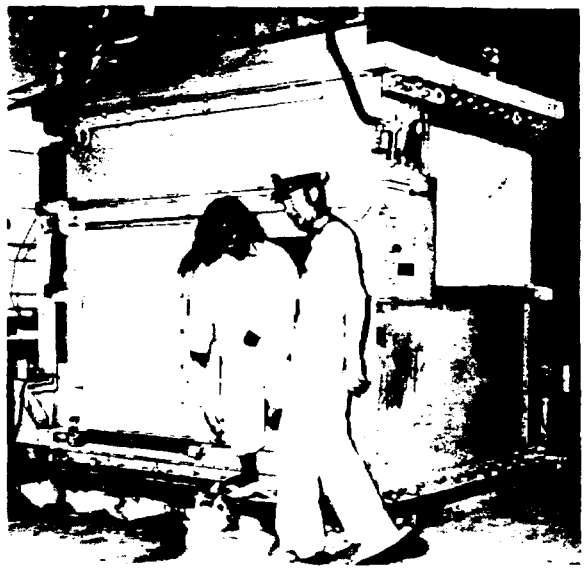


Figure 53. Steel room used for whole-body gamma spectroscopy.[7]

Figure 54. Arrangement of lead bricks used for whole-body counting.

Table 37

Radiochemical Analyses of Urine (Data in Average pCi/liter)

Year	No. in group	Av. vol., ml	Av. Ca, mg/liter	^{90}Sr	^{137}Cs	^{238}Pu	^{239}Pu	$^{239,240}Pu$
Rongelap								
1970	20	895	152.4	3.5	2700			
1971	15	534	336.1	3.7	2400			
1972	18	461	120.3	2.4	2600			
1973	11	249	247.2	6.5	4600			0.21
1974	14	558	706.8	2.8	4500			
Utirik								
1974	11	542	734.9	1.3	1300			
Bikini								
1970	Pooled		120.0	1.2	115	0.003	0.003	
	Urine G	1100		2.2		0.013	0.020	
	Urine M	930		1.9		0.015	0.024	
	HASL* control	3000	160.0	1.0	12	0.003	0.003	
	HASL control	1000		1.6		0.014	0.022	
1971	Pooled	2670	84.5	1.7	183			0.004
1972	Pooled	2700	204.0	4.2	910			
1973	14	294	173.5	6.7	1500			
1974 (Spring)	11	141	310.0	2.0	1100			0.02

*US AEC Health and Safety Laboratory, New York, N.Y.

Table 38

Radiochemical Analyses of Well Water From Bikini (Data in pCi/liter)

Year	Sample	Vol., ml	^{90}Sr	^{137}Cs	^{3}H	$^{239,240}Pu$
1971	"good well"	1830	6.0 ±17%	600 ±1%	770±40%	0.04 ± 25%
	"bad well"	1830	25 ± 3%	850 ±1%	1040±30%	0.05 ± 20%
	"good well (closed)	1810	103 ± 2%	1044 ±1%		0.058± 15%
	"good well" (opened)	1980	125 ± 3%	818 ±1%		5.76 ± 6%
	drinking water (camp area)	3580	0.46± 4%	1.53±8%		0.004±100%
1972	well water	1000	15.4 ± 9%	800 ±1%		
	drinking water	1960	0.61± 6%	1.8 ±8%		
1973	new well	60	52	600		0.38 ± 40%
	B-1 well	225	11	724		0.08 ± 50%

Table 39

Radiochemical Analyses of Coconut Crabs From Bikini (Data in pCi wet weight)

Year	Wet wt., g	% Ash	g Ca per kg wet wt.	^{90}Sr	^{137}Cs	^{238}Pu	^{239}Pu
1970	1164	23.3	81	23,300	11,800	0.06 ± 50%	1.5 ±10%
	1930	18.5	61	24,800	14,800	0.001±100%	0.07±37%
1971	1812	17.8	60	132,000	11,400		
	1827	21.5	72	412,000	8,600		
1973	1190		63.5	45,700	9,290		

induced activities (^{59}Fe, ^{57,58,60}Co, ^{54}Mn, ^{144}Cs-^{144}Pr, ^{95}Zr-^{95}Nb, and ^{106}Ru-^{106}Rh). Radiological assessment has included surveys for gamma radiation levels on the islands; radiochemical studies of soil profiles, lagoon bottoms, marine life, plants, animal life (e.g., rats, coconut crabs), food items, and urine samples; and gamma spectrographic analysis of internally deposited gamma emitters in personnel. The medical team has been responsible since 1969 for monitoring the body burdens of the people returning to Bikini, as well as of the Marshallese exposed to fallout and their controls, and more recently also of the people returning to Eniwetok. The 1974 studies of environmental radiation on Rongelap, Utirik, and Bikini are being published separately.[165]

Until 1965 gamma spectroscopy was done in a 21-ton steel chamber built at BNL for shielding, with additional housing for the counting equipment (Figure 53). The subject to be counted took a soap and water shower, put on paper pajamas, and then stayed in the chamber for about 15 min, during which soft music was piped in. Only a few small children were apprehensive during the procedure. More recently whole-body counting has been done with a smaller "shadow-shield" arrangement of lead bricks, a modification of the Hanford model[166] (Figure 54). In 1974 spectrographs showed a characteristic peak for ^{137}Cs and ^{65}Zn, but ^{60}Co was barely discernible, if at all, at the low levels present in the people (see Figure 55).[167,168] Annual collections of 24-hr urine samples from a number of people have been made for radiochemical analysis.

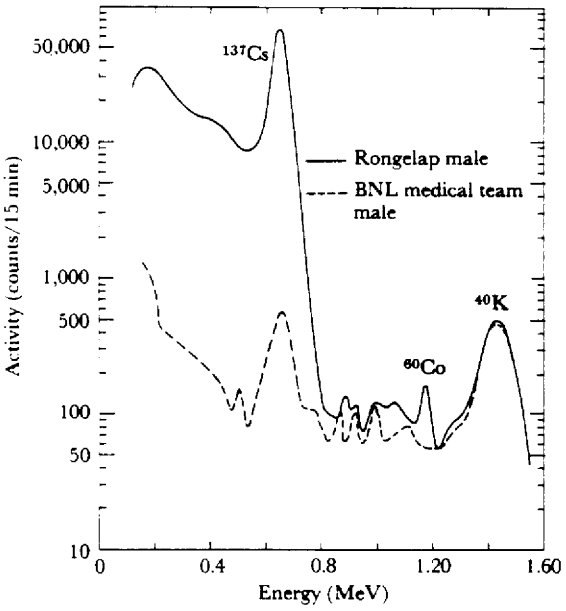


Figure 55. Gamma spectra obtained by whole-body counting, males, 1974 (——, Rongelap; – – –, BNL medical team).

The results of radiochemical analyses of urine, water, and crabs over the past 5 years are summarized in Tables 37 to 40; the results of individual urinalyses are given in Appendix 12.* By 1974 the only detectable gamma emitter was ^{137}Cs; its levels are given for Rongelap, Utirik, and Bikini individuals in Appendix 12 and for groups in Table 41. Since the return to Rongelap, no differences have been seen between exposed and unexposed groups.

Figures 56 and 57 show the estimated body burdens of gamma emitters and ^{90}Sr for the Rongelap people at various times after the initial exposure in 1954. After their return to Rongelap the body burdens increased. ^{65}Zn was present for a few years, apparently from fish in the diet; the reason for its rapid decrease was not immediately apparent but may be related to movements of marine life in and out of the lagoon. Small amounts of ^{60}Co were identified in personnel during the early years after the return (the highest concentration was in clams). ^{90}Sr body burdens reached their highest level during 1962 to 1965 at about 12 nCi

*We are indebted to Mr. Edward P. Hardy, Jr., Director, Environmental Studies Division, AEC Health and Safety Laboratory, New York, N.Y., for carrying out the radiochemical analyses.

Table 40

Radionuclides in Coconut Crabs From Rongelap

Year	^{137}Cs, pCi/kg	^{90}Sr, pCi/g Ca
1961		1,140
1962		1,224 (1,317; 1,086; 1,113; 1,378)
1964	50,281 (39,292; 45,318; 66,234)	758 (865; 628; 780)
1965	12,700	724
1969	7,775 (8,540; 7,010)	705 (910; 500)
1972	5,900 (5,600; 6,100; 6,800; 5,100)	353 (376; 419; 359; 259)
	12,900*	933*
1973	37,065 (64,700; 9,430)*	3,950 (6,639; 1,267)*
1974	6,600 (5,000; 8,200)	448 (290; 606)
	4,800*	290*

*From northern islands of Rongelap Atoll, where radiation dose was much higher.

Table 41

Mean Cesium-137 Levels Obtained by Whole-Body Counting, 1974

	Male			Female		
	No.	nCi	nCi/kg body wt.	No.	nCi	nCi/kg body wt.
Bikini	8	128	1.84 (0.43–5.11)	13	73	1.15 (0.22–3.26)
Utirik	9	262	4.05 (2.64–6.84)	13	133	2.13 (0.96–3.85)
Rongelap	22	475	7.76 (4.37–16.3)	24	304	5.13 (2.71–13.46)
BNL med. team	4	2.93	0.0352 (0.0134–0.0791)			

Table 42

Dose to Bone Marrow (Data in mrad/year)

	1958	1974		
	Rongelap	Rongelap	Utirik	Bikini
Internal				
^{90}Sr	11	8	6	5
^{137}Cs	120*	60	31	16
Natural (^{40}K, etc.)	44	44	44	44
Total	175	112	81	65
External				
Residual gamma (in village areas)	250	15	7	65
Natural (cosmic etc.)	134**	39	39	39
Total	384	54	46	104
Combined total	559	166	127	169

*$^{137}Cs + ^{65}Zn$.

**High value due to increased cosmic-ray dose from above-average sunspot activity. The year 1958 was the time of peak radioactivity at Rongelap.

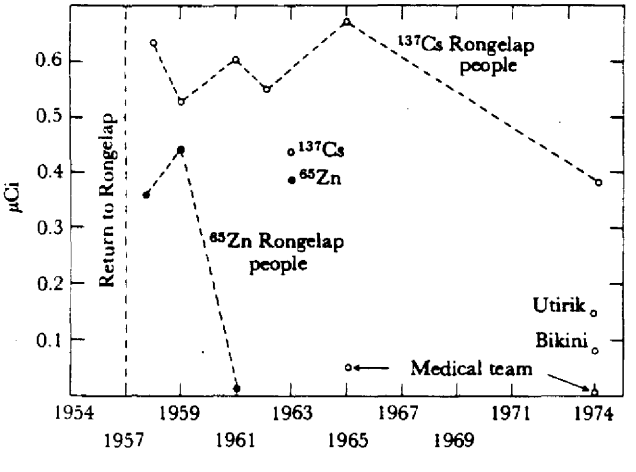


Figure 56. Body burdens of gamma emitters, obtained by whole-body gamma spectroscopy (○, ^{137}Cs; ●, ^{65}Zn).

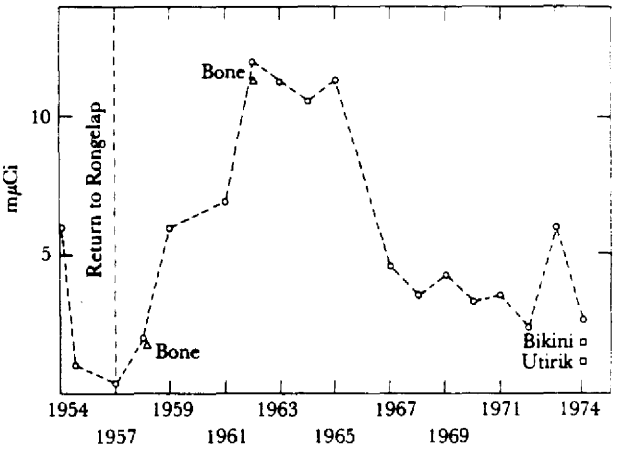


Figure 57. Body burdens of strontium-90, obtained by radiochemical analysis of urine.

in adults and 22 nCi in children, about 6 and 11% respectively of the maximum permissible lifetime body-burden levels for the population at large, i.e., 1/10 the ICRP value.[169] Analyses of bone samples from several autopsies during the past 20 years gave estimated ^{90}Sr body burdens similar to those obtained from urinalysis (see Figure 57). ^{137}Cs body burdens also reached their peak in about 1965, at nearly 0.7 μCi (23% of the permissible level for the general population). The body burdens of the Utirik people were considerably below those of the Rongelapese. Table 42 shows that the estimated bone marrow doses of Rongelap people from internal and external sources (both natural and man-made radioactivity) were considerably reduced by 1974, and presents data also for Utirik and Bikini.*

The coconut crab was banned from the diet on Rongelap because of its high level of radioactivity, but about 2 years ago the ban was lifted on the southern islands of the atoll because the radioactivity had decreased sufficiently (see Table 40).

B. ^{55}Fe BODY BURDENS IN RONGELAPESE

In 1971 blood samples from 62 Rongelap residents were analyzed** for ^{55}Fe, and their body burdens were estimated by the method of Beasley et al.[170] ^{55}Fe was known to be present in the Marshall Islands in fairly high levels as a result of the local nuclear testing program and also from worldwide fallout, and to be concentrated by marine life, particularly fish. The estimated body burdens for the Marshallese tested (Table 43) are higher than those found for Japanese bomb survivors and for others tested, but they are not more than 1/100 of the maximum permissible levels for non-occupationally exposed individuals estimated with the total body as the critical organ. The values are slightly higher in females than in males, and significantly higher in older persons. ^{55}Fe emits photons of very low energy, and, since it is incorporated into the hemoglobin of the red cells, it results in relatively little irradiation of nucleated cells. Some blood samples from the 1974 survey are also being analyzed for ^{55}Fe.

*Mr. R. Fairchild at BNL calculated the bone marrow dose for the 1974 data.

**The ^{55}Fe analyses were carried out by T.M. Beasley and E.E. Held at the University of Washington School of Fisheries, Seattle.

Table 43

Average Body Burdens of ^{55}Fe in Rongelapese, 1971[170]

	Age	No. of samples	Body burden, μCi
Males	16–20	8	0.31
	21–31	4	0.33
	32–42	5	0.52
	43–53	2	0.58
	54–64	6	0.53
	>64	3	0.48
Females	16–20	6	0.23
	21–31	12	0.34
	32–42	5	0.33
	43–53	7	0.66
	54–64	2	0.57
	>64	2	0.66

C. PERSONNEL MONITORING AT BIKINI

In 1946, before Operation Crossroads, the residents were evacuated from Bikini. After stays at Rongerik and at Kwajalein which proved unsatisfactory, they were relocated on Kili Island in the southern Marshalls, which also proved unsatisfactory. The Eniwetok people were relocated at Ujelang Atoll, to the west, after their evacuation.

After the 1958 moratorium on atmospheric nuclear testing, numerous radiological surveys were done on Bikini and later on Eniwetok Atoll.[171-174] In 1967 the principal isotopes contributing to the gamma radiation field on Bikini and Eneu Islands were ^{137}Cs, ^{60}Co, ^{125}Sb, and ^{155}Eu; slight amounts of Pu were also found. Considerable variation was seen in the contamination of individual islands comprising the atolls of Bikini and Eniwetok since different tests had been conducted on various ones. The contamination of Rongelap and Utirik was more uniform, being due largely to fallout from a single detonation, Bravo.

In 1968 an *ad hoc* committee reviewed the survey results for Bikini and decided that Eneu and Bikini Islands were safe for habitation, with certain measures recommended to reduce exposure (see Appendix 13). In 1969 about 30 people started work on Bikini Atoll (living on Eneu), and in 1971 several Bikini families moved back to Bikini Island itself, which now has about 50 Bikini people plus a few administrators and construction workers. Annual monitoring of personnel has been carried out since 1969. In 1974 radiochemical analyses were done on urine (see Table 37 and Appendix 12) and other samples, whole-body gamma spectroscopy was carried out on personnel living on

Bikini (see Table 41 and Appendix 12), and a gamma survey of the island was made by Greenhouse et al.[156] (see Appendix 14).

On Bikini Island almost all the gamma radiation is now due to ^{137}Cs. The gamma level due to natural radioactivity is considerably less on these islands than in the U.S.; this is why the average gamma dose to an inhabitant is lower in Bikini Village than in certain parts of the U.S. The people living on Bikini eat a diet consisting almost entirely of imported food, but they ingest slight amounts of radioactivity from local seafood. The newly planted coconut, pandanus, and breadfruit trees will not bear fruit in any significant quantity for some years yet. The urinary radionuclide level of the Bikini people is now several times as high as when they were living on Kili Island, but still considerably below the permissible range. The total estimated internal bone marrow dose in 1974 to the people living on Bikini was about half that to the people living on Rongelap, but with the external dose included, the dose to the bone marrow was about the same (see Table 42). It is reassuring that analyses of urine for plutonium show levels near background in the Bikini people (see Table 37). Well water on Bikini has had low levels of activity (see Table 38). The dietary levels projected for Bikini when the newly planted trees bear fruit should be considerably lower than the levels based on analyses of fruit from old trees because of the soil removal procedures used at planting, but their actual evaluation must await maturity of the trees and further analyses. It is planned to add supplementary calcium to the diet of the Bikini people to help reduce absorption of ^{90}Sr. Experiments are under way to investigate the tolerance of Marshallese people for powdered milk as a source of calcium. Races other than the Caucasian are reported to have a lower tolerance for milk because of a genetic inability to digest lactose which gives rise to abdominal discomfort, diarrhea, and other symptoms.[175]

VII. Comments and Conclusions

A. THE EXAMINATIONS

The primary responsibility assigned the medical team by the AEC (now ERDA) was the diagnosis and treatment of possible effects of radiation exposure in the Marshallese, but inevitably the examining groups have been concerned also with general health care at the time of their visits. The responsibilities have been filled by carrying out extensive annual examinations, supplemented in recent years by semiannual hematology checks and quarterly visits by a resident physician. The examinations, which have been carried out with the assistance of the Trust Territory Health Services, have resulted in extensive medical histories and records for each individual and have made it possible to diagnose and treat many diseases and illnesses at an early stage.

An important aspect of the medical surveys is to maintain rapport with the people, to keep them informed of the medical findings, to explain the need for examinations, and at times to correct unfounded rumors about fallout effects. Unfortunately this has not always been successfully kept up because of differences in language and culture.

Some studies have been of benefit to the Marshall Islands as a whole, such as the surveys of the incidence of diabetes and of parasitic infestation. In addition, a great deal of data has accumulated from studies of genetically inherited characteristics which may prove valuable not only in determining possible genetic effects of radiation but also in anthropology.

B. COMPARISON WITH OTHER HUMAN EXPOSURES

In contrast to other groups exposed to radiation, the Marshallese are unique in that they comprise the only human population ever exposed to acute radiation from fallout. The accident focused attention for the first time on the hazards of fallout from nuclear detonations. The atomic blasts above Hiroshima and Nagasaki resulted in casualties due to penetrating gamma and neutron radition directly from the bombs with little or no fallout involved and therefore caused no effects due to internal absorption of radioactive materials. In contrast, the Marshallese were not exposed to direct effects of the detonation but only to radioactive fallout resulting in whole-body, skin, and internal exposure. Trauma and extreme psychological disturbances did not contribute to the effects in the Marshallese as they did in the Japanese. The importance of the hazard from internal absorption of radioactive iodine has been clearly demonstrated by the Marshallese experience. The 23 Japanese fishermen on the *Lucky Dragon* received an exposure similar to that of the Rongelap group but

probably with less internal absorption of radionuclides, since their stored water and food were covered, and they have had no thyroid effects (see Appendix 2).

Evaluation of the effects of radiation exposure in human beings (patients, physicians using radiation, accident cases, etc.) is always difficult because of uncertainties regarding exact dosage, fractionation and dose-rate effects, partial-body exposure, complicating diseases, etc. The doses received by the Marshallese, like most human exposures, could be only roughly estimated, although the hematological data were compatible with the calculated whole-body doses. Even greater uncertainties were encountered in estimating the doses due to internal absorption of radionuclides.

The data on the effects of fallout radiation in the Marshallese have provided important information that will apply in a general way to any population exposed acutely to fallout. However, the effects may be modified in other situations if nuclear explosions occur in regions with different terrain, soil types, climate, and availability of protective measures.

C. ACUTE EFFECTS

The most serious acute effects of the exposure in the Marshallese were due to penetrating gamma radiation. These included transient anorexia, nausea, and vomiting and significant depression of the peripheral blood elements in many members of the higher exposure Rongelap group. The hematological depression was not sufficient to produce definite clinical signs and required no specific therapy.

Contamination of the skin in the Rongelap group resulted in wide-spread beta burns on parts of the body not covered by clothing and in spotty epilation of the scalp. These effects were probably aggravated by delay in decontamination and by perspiration due to the warm climate causing the fallout to stick to the skin. The superficial nature of the lesions, rapid healing with minimal residual skin changes, and regrowth of hair were no doubt due to the low average energy of the beta radiation in the fallout.

The lack of recognizable acute effects from the internal absorption of radionuclides is noteworthy in view of the serious thyroid abnormalities that later developed.

Because of residual contamination on the islands, radiological monitoring of personnel and environment has been an important part of the surveys in evaluating body burdens of radionuclides in the Rongelap and Utirik people. Recently the areas undergoing such monitoring have included Bikini Atoll and the people who have returned to live there, and they will also include Eniwetok when its people return home.

D. LATE EFFECTS

The possible emergence of late effects of exposure in the Marshallese has received considerable attention in follow-up examinations. Except for the thyroid lesions and the one case of leukemia, only a few findings possibly related to radiation exposure have been seen; otherwise the general incidence of illnesses and the overall physical condition have been similar in the exposed and in the unexposed comparison groups. The increase in miscarriages and stillbirths among the exposed Rongelap women during the first 5 years after exposure may or may not have been related to radiation effects. No genetic effects have been noted in the children born of exposed parents; this is not surprising in view of the generally negative findings in the much larger Japanese study. The findings of persistent chromosome aberrations in cultured peripheral blood lymphocytes at 10 years post exposure and a possible somatic mutation in hemoglobin in several of the exposed group suggest that genetic mutations may also be present. The possibility of genetic effects in the offspring is of serious concern to the exposed people and deserves further study.

Effects of radiation on life shortening or mortality are difficult to evaluate because of the small number of people and the differences in age distribution between the exposed and comparison groups. The only death that may be related to exposure is that from leukemia. The occurrence of a few additional cases of cancer (other than thyroid) cannot be ascribed definitely to radiation exposure. The lack of skin cancer from beta burns may be related to the minimal nature of the residual skin changes, probably due to insufficient radiation injury to the dermis, but the possibility of skin cancer developing must be kept in mind because the latent period may be very long.

The development of a case of acute leukemia in the Rongelap boy may or may not be related to radiation exposure. However, this disease appears to be even rarer in the Marshall Islands than in the U.S. It is noteworthy that his disease was the

myelogenous form, since his past hemograms showed a fairly consistent depression of neutrophil counts compared with those of other exposed boys of the same age.

The inability to demonstrate clear-cut aging effects in the exposed group in spite of repeated attempts with a variety of tests is in accord with the generally negative results of similar attempts in the much larger exposed Japanese populations.

The absence of radiation-induced cataracts is not unexpected since the dose to the lens was probably below threshold and neutrons (known to have a higher RBE for cataract induction than gamma radiation) were not involved in the Marshallese exposure.

E. THYROID EFFECTS

The high incidence of thyroid neoplasms (in 27 of 86 exposed Rongelap people, including 3 with carcinomas) and the development in some children of hypothyroidism and growth retardation provide clear evidence for the seriousness of thyroid injury due to radioiodine absorbed from fallout associated with atomic detonations. Had not careful medical studies in the population been instituted soon after the exposure and continued to the present date, the extent of thyroid effects might not have been discovered.

Thyroid exposure is likely to be greater when individuals are exposed within 100 to 150 miles of the bomb if detonated near the ground. In such situations, if lethal exposure to penetrating radiation does not occur, the principal hazard appears to be the late development of thyroid tumors and leukemia. Since the latter is ultimately fatal, it is more serious than thyroid malignancies, most of which are well differentiated and have an excellent prognosis. The relative incidence of thyroid malignancies from radiation appears to be about the same as that of leukemia on a "per rad" basis.[177] The incidence of thyroid cancer is considerably higher than the mortality from it. Of 40 cases among the exposed Japanese, 34 were living in 1973, and only one death had been attributed to this cause. No evidence of thyroid dysfunction had been noted at the time of diagnosis. In the Marshallese reduced function was not found in two of the cancer cases exposed as adults, but was in the case exposed as a child.

The high incidence of thyroid effects in children exposed at <10 years of age was no doubt related to the higher dose to the child's thyroid because of its smaller size. The rapid growth of the glands during childhood probably increased the chances for neoplastic changes. The growth retardation in some of these children was thought to be related to reduced thyroid function resulting in lower hormone levels. This deficiency was not recognized during the early years because of falsely high PBI levels resulting from unusually high iodoprotein levels, which turned out to occur generally in the Marshallese and are now being further studied.

Most of the thyroid glands of the exposed people undergoing surgery contained multiple nodules or areas of adenomatous change. Many microscopic areas, although considered benign, were composed of discrete areas of atypical cells, suggestive in some cases of malignant potential.

The risk of developing benign and malignant neoplasms in the Rongelap people appeared to be about the same as that noted in people exposed to x-radiation. Clinical experience with ^{131}I suggested that the risk would have been less, but the higher energy of the short-lived isotopes of iodine (particularly ^{132}I, ^{133}I, and ^{135}I), resulting in higher dose rate and more uniform exposure of the thyroid, is thought to have been the important factor in increasing the number of thyroid abnormalities above that expected from similar doses from ^{131}I alone. It is not unreasonable to speculate that tumor incidence in the Marshallese would have been considerably smaller if only ^{131}I had been involved in the exposure. The lesser amount of short-lived iodine isotopes in the Utirik exposures (because of the later arrival of the fallout) may have been an important factor in reducing the dose effect to their thyroids, but the number of people involved is too small for any firm conclusion to be drawn.

Treatment of the exposed Rongelap people with thyroid hormone has been of benefit in enhancing growth and development in the growth-retarded children and in maintaining a normal metabolic state in the operated cases. It is not certain whether it has prevented the development of thyroid nodules.

The documentation of these thyroid effects has importance not only for the people involved but also for the advancement of medical knowledge and for planning with regard to Civil Defense and remotely possible future accidents involving release of radioiodines (e.g., from a nuclear power plant). The Marshallese accident represents ex-

posure under extreme conditions, with no corrective measures taken to reduce internal absorption of radioisotopes prior to evacuation of the exposed people from the fallout area. It did not involve the contaminated pasture-cattle-milk cycle, which might be an important pathway of radioiodine to man in other types of accidents (such as the Windscale accident).[176]

Civil Defense planning can provide for several measures that will reduce the hazard of thyroid exposure due to radioiodine absorption and thus largely preclude the degree of thyroid injury sustained by the Marshallese. Since the hazard from radioiodine is acute for a period only of days, early protective measures are extremely important. These include avoiding inhalation of radioiodines by shelter protection; consuming food and water only from closed containers; feeding cows in contaminated areas protected fodder; and temporarily withholding contaminated milk supplies and diverting them into processed products with a shelf life longer than the life of the isotope. The addition of stable iodine to food or water during the first week woulld provide a relatively inexpensive method of reducing thyroid uptake of radioiodines by isotope dilution and saturation with non-radioactive iodine; this should rarely produce any serious side effects and would be of particular value in children and pregnant women. When exposure of the thyroid has already occurred, prophylactic treatment with thyroid hormone, now being used in the Marshallese, may help prevent development of thyroid tumors, and even after tumor development, surgical excision may reduce mortality due to malignancy.

The thyroid effects in the Marshallese were not anticipated at the time of the accident or during the early years afterwards. In retrospect this is not surprising, for several reasons. At that time the thyroid was thought to be relatively radioresistant, particularly with regard to radioiodine exposure (on the basis of animal studies and diagnostic and therapeutic use of ^{131}I in people), and the calculated thyroid doses in the Marshallese were considered to be below the levels likely to produce tumors. In addition, neither the importance of the exposure to short-lived iodine isotopes in fallout nor the thyroid dose differential in children due to the smaller size of their gland was fully appreciated.

It is quite likely that the final results of thyroid lesions in the Marshallese are incomplete at this time since new lesions are still occurring. The mean latent period for radiation-induced thyroid tumors may be as long as 30 years. Cases have been seen as late as 40 years after exposure. Furthermore, on the basis of the present data the risk of developing radiation-induced thyroid neoplasia is probably underestimated, since surgical removal of potentially malignant tissues may have occurred and the hormone treatment may have inhibited the development of some tumors, although the latter is questionable. As has been pointed out, also, the true carcinogenic potential of the exposure, particularly in the children receiving the higher dose, may have been masked by excessive cellular destruction. The recent finding that subclinical thyroid deficiency is present in some of the exposed people who have not shown any thyroid abnormalities indicates that the thyroid effects in the Marshallese may not yet be completely manifest, and continued careful surveillance of this population is necessary.

Acknowledgments

Because of the broad scope of the surveys and the complexity of operations it would be impossible to acknowledge all the individuals and organizations that have contributed to the success of the surveys over the past 20 years. We would like, however, to express thanks to some who have contributed vitally in more recent years.

Our foremost appreciation is to the people of Rongelap and Utirik who have participated in the examinations, including the unexposed people comprising the control population. We treasure the friendship and trust of these people. We are grateful to many Rongelap and Utirik people for assisting the examining teams over the years, including the Magistrates of both islands – at Rongelap: John Anjain, Billiet Edmond, Jabwe Jorju, and Niktimos Antak; at Utirik: Kabto Campus, Maja Lena, Harold, the school principal, and Joata, the health aide.

The senior author is most grateful to the many fine medical and other specialists (listed in Appendix 2) who have so willingly given of their time and talents, without remuneration, to participate in the surveys. Without their dedicated efforts the surveys could never have succeeded. He is also greatly indebted to the many technical assistants who have contributed much to the success of the surveys, particularly to Mr. W. Scott and Mr. D.

Clareus of BNL for long years of faithful service and also to Mr. P. Heotis and Mr. M. Makar. In the Trust Territory, Messrs. S. Shoniber, N. Zetkeia, K. Mitzutani, J. Saul, and P. Bien, Mrs. R. Harris, and many others have provided invaluable assistance over the years.

At Brookhaven National Laboratory contributions have been made by many individuals and by many departments and divisions, including Medical, Instrumentation, Health Physics and Safety, Photography and Graphic Arts, Purchasing, Supply and Materiel, Staff Services, and others. We are indebted to the following personnel at BNL for their advice and support: in the Director's Office, Drs. L.J. Haworth, M. Goldhaber, G.H. Vineyard, and V.P. Bond, and Mr. P. Rathvon; in the Medical Department, Drs. E.P. Cronkite, R.B. Aronson, H.L. Atkins, L.K. Dahl, A.D. Chanana, G. Chikkappa, J. Iwai, J.S. Robertson, L.V. Hankes, C.H. Wu, R.D. Stoner, and D. Slatkin, Messrs. W.A. Finn, C. Goldman, W.J. Walsh, and W. Hanson, Mrs. I. Montanez for secretarial help, and the nursing staff and orderlies who gave the Marshallese patients excellent care; in the Health Physics and Safety Division, Mr. C.B. Meinhold and Mr. N.A. Greenhouse, Jr.; in the Instrumentation Division, Dr. M.V. Radeka. We are also grateful to Mr. K.H. Thompson for statistical computations and Mrs. M. Dienes for editorial assistance. We are most appreciative to the people of BNL for generous donations of clothing, books, and toys for the Marshallese.

We are indebted to the Atomic Energy Commission (now ERDA) for their staunch support, particularly to Drs. J. Totter, J.L. Liverman, C.E. Carter, W.W. Burr, C.W. Edington, S. Marks, and N.F. Barr and also to Dr. M.B. Biles and Messrs. J. Deal and T.F. McCraw; and at the Nevada Operations Office to Mr. M.E. Gates and Mr. R. Ray. We wish to acknowledge the outstanding contributions of the Pacific Area Support Office in Honolulu, by Messrs. W. Bonnett, W.B. Hills, J. Miller, W. Morrison, J. Abreu and particularly W.S. Streenan, whose devoted service was essential to the successful completion of the surveys. At the AEC Health and Safety Laboratory we appreciate the assistance of Drs. E.P. Hardy, Jr. and J. Harley in carrying out radiochemical analyses.

In the Deparment of the Interior, Messrs. S.S. Carpenter, J. DeYoung, and H.U. Brown have been most cooperative. In the Trust Territory many individuals have been of tremendous assistance in carrying out this joint mission, in particular Mr. E.E. Johnston, High Commissioner, Mr. P. Coleman, Mr. D. Heine, Dr. M. Kumangai, and Dr. W. Peck at Saipan, and at Majuro Mr. O. DeBrum, Dr. J. Tobin, Mr. J. Pualoa, and Drs. J. Iaman, E. Riklon, J. Anjain, M. Korean, and others.

At Kwajalein (Kwajalein Missile Range) we are grateful for the splendid support of the U.S. Army Group: Col. J.L. Fishback, Col. R.L. Russell, Col. R.A. Ehlert, Col. R.F. Sullivan, Mr. J.S. Beavers, and Mr. R. Haley; and to personnel from Global Associates: Mr. R. Bryant, Mr. D.D. McAfee, Dr. J. Schaefer, and particularly Mr. F. Grannich and Mr. H. Hauck; to Trust Territory representative Mr. W. Ownbey; and our special thanks go to the crew of the LCU (*Liktanur*).

In Japan we are indebted to the staff of the Atomic Bomb Casualty Commission (now Radiation Effects Research Foundation) particularly Drs. G. Darling, L. Allen, H. Maki, and I. Nagai for generous support and assistance in maintaining liaison between the two studies; also to Dr. T. Kumatori and others of the staff of the National Institute of Radiological Sciences at Shiba.

In England it is a pleasure to acknowledge the advice and help of Dr. E.E. Pochin of the University College Hospital Medical School, London.

Among medical specialists from other institutions we are most grateful to Drs. Shields Warren, W. Meissner, J. Reid, A. Upton, and D.A. Wood for their personal interest in the examinations and for their advice on pathological diagnosis; also to the following pathologists who have contributed: Drs. L.V. Ackerman, B. Castleman, H. Cottier, H. Johnson, G.H. Klinck, M.A. Legg, S. Lindsay, S. Lippincott, S.J. Robbins, R.V. Rosvoll, A.L. Vickery, T. Winship (deceased), and L.B. Woolner.

We appreciate very much the assistance of Col. E.J. Kamin and his staff at Tripler Army Medical Center in caring for Marshallese patients brought to Honolulu.

At the University of Washington Drs. A.H. Seymour, E. Held, and V. Nelson have advised and cooperated in studies of mutual interest.

We are most grateful to the charitable organization New Eyes for the Needy, Inc. (Short Hills, N.J.) for donating hundreds of pairs of eyeglasses for the Marshallese. They were fitted and distributed at the time of the surveys and probably no other gift has been more gratefully received by the people.

We have been greatly saddened by the recent deaths of several individuals in the Marshall Islands who have contributed much to the surveys: Mr. E. Libby, Drs. A. Hicking and T. Ishoda, and Mr. M. Lacjohn. We express our heartfelt appreciation for all they did for us.

References

1. HATZEL, A.M., Health survey of the Trust Territory of the Pacific Islands, *U.S. Armed Forces Med. J.* **10**: 1199-222 (1959).
2. CRONKITE, E.P. ET AL., *Some Effects of Ionizing Radiation on Human Beings: A Report on the Marshallese and Americans Accidentally Exposed to Radiation Fallout and a Discussion of Radiation Injury in the Human Being*, AEC-TID 5385, U.S. Government Printing Office, Washington, D.C., 1956.
3. BOND, V.P., CONARD, R.A., ROBERTSON, J.S., AND WEDEN, E.A. JR., *Medical Examination of Rongelap People Six Months After Exposure to Fallout*, WT-937, Operation Castle Addendum Report 4.1A, April 1955.
4. CRONKITE, E.P. ET AL., *Twelve-Month Postexposure Survey on Marshallese Exposed to Fallout Radiation*, BNL 384 (T-71), Aug. 1955.
5. CONARD, R.A. ET AL., Medical survey of Marshallese two years after exposure to fallout radiation, *J.A.M.A.* **164**: 1192 (1957); BNL 412 (T-80), March 1956.
6. CONARD, R.A. ET AL., *March 1957 Medical Survey of Rongelap and Utirik People Three Years After Exposure to Radioactive Fallout*, BNL 501 (T-119), June 1958.
7. CONARD, R.A. ET AL., *Medical Survey of Rongelap People, March 1958, Four Years After Exposure to Fallout*, BNL 534 (T-135), May 1959.
8. CONARD, R.A. ET AL., *Medical Survey of Rongelap People Five and Six Years After Exposure to Fallout*, BNL 609 (T-179), Sept. 1960.
9. CONARD, R.A. ET AL., *Medical Survey of Rongelap People Seven Years After Exposure to Fallout*, BNL 727 (T-260), May 1962.
10. CONARD, R.A. ET AL., *Medical Survey of Rongelap People Eight Years After Exposure to Fallout*, BNL 780 (T-296), Jan. 1963.
11. CONARD, R.A. ET AL., *Medical Survey of the People of Rongelap and Utirik Islands Nine and Ten Years After Exposure to Fallout Radiation (March 1963 and March 1964)*, BNL 908 (T-371), May 1965.
12. CONARD, R.A. ET AL., *Medical Survey of the People of Rongelap and Utirik Islands Eleven and Twelve Years After Exposure to Fallout Radiation (March 1965 and March 1966)*, BNL 50029 (T-446), April 1967.
13. CONARD, R.A. ET AL., *Medical Survey of the People of Rongelap and Utirik Islands Thirteen, Fourteen, and Fifteen Years After Exposure to Fallout Radiation (March 1967, March 1968, and March 1969)*, BNL 50220 (T-562), June 1970.
14. CONARD, R.A., Medical survey of Marshallese people five years after exposure to fallout radiation, *Int. J. Radiat. Biol. Suppl. 1:* 269-81 (1960).
15. CONARD, R.A., The biological hazards of a fallout field, in *Radioactivity in Man*, pp. 249-65, G.R. Meneely, Editor, Thomas, Springfield, Ill., 1961.
16. SUTOW, W.W., CONARD, R.A., AND GRIFFITH, K.M., Growth status of children exposed to fallout radiation on Marshall Islands, *Pediatrics* **36**: 721-31 (1965).
17. CONARD, R.A. AND HICKING, A., Medical findings in Marshallese people exposed to fallout radiation: Results from a ten-year study, *J.A.M.A.* **192**: 457-9 (1965).
18. RALL, J.E. AND CONARD, R.A., Elevation of the serum protein-bound iodine level in inhabitants of the Marshall Islands, *Amer. J. Med.* **40**: 883-6 (1966).
19. CONARD, R.A., RALL, J.E., AND SUTOW, W.W., Thyroid nodules as a late sequela of radioactive fallout in a Marshall Islands population exposed in 1954, *N. Engl. J. Med.* **274**: 1392-9 (1966).
20. ROBBINS, J., RALL, J.E., AND CONARD, R.A., Late effects of radioactive iodine in fallout, *Ann. Intern. Med.* **66**: 1214-42 (1967).
21. CONARD, R.A., SUTOW, W.W., COLCOCK, B.P., DOBYNS, B.M., AND PAGLIA, D.E., Thyroid nodules as a late effect of exposure to fallout, in *Radiation-Induced Cancer (Proc. IAEA Symp., Athens, April 1969)*, pp. 325-36, IAEA, Vienna, 1969.
22. SUTOW, W.W. AND CONARD, R.A., The effects of fallout radiation on Marshallese children, in *Radiation Biology of the Fetal and Juvenile Mammal (Proc. 9th Annu. Hanford Biology Symp., Richland, Wash., May 1969)*, pp. 661-73, M.R. Sikov and D.D. Mahlum, Editors, CONF-690501, 1969.
23. CONARD, R.A., DOBYNS, B.M., AND SUTOW, W.W., Thyroid neoplasia as a late effect of acute exposure to radioactive iodines in fallout, *J.A.M.A.* **214**: 316-24 (1970).
24. A report on the people of Rongelap and Utirik relative to medical aspects of the March 1, 1954 incident: Injury, examinations and treatment, Presented by the Special Joint Committee Concerning Rongelap and Utirik Atolls (Public Law No. 4C-33) at Fifth Congress of Micronesia, First Regular Session, Feb. 1973.
25. HINES, N.O., *Proving Ground: An Account of the Radiological Studies in the Pacific, 1946-1961*, University of Washington Press, Seattle, 1962.
26. JAMES, R.A., *Estimate of Radiation Dose to Thyroids of the Rongelap Children Following the Bravo Event*, UCRL 12273, Dec. 1964.
27. HARRIS, P., Unpublished data, 1954.
28. MOCHIZUKI, Y., MOWAFY, R., AND PASTERNACH, B., Weights of human thyroids in New York City, *Health Phys.* **9**: 1299-301 (1963).
29. SIRI, W.E., Personal communication, 1965.
30. TRAENKLE, H.L., X-ray-induced skin cancer in man, *Nat. Cancer Inst. Monogr.* **10**: 423-32 (1963).
31. TELOH, H.A., MASON, M.L., AND WHEELOCK, M.C., A histological study of radiation injuries of the skin, *Surg. Gynecol. Obstet.* **90**: 335-48 (1950).
32. CONARD, R.A., An attempt to quantify some clinical criteria of aging, *J. Gerontol.* **15**: 358-65 (1960).
33. BYRNES, V.A., BROWN, D.V.L., ROSE, H.W., AND CIBIS, P.A., Chorioretinal lesions due to thermal

radiation from the atomic bomb, *AMA Arch. Ophthalmol.* **54**: 909-13 (1955).
34. Byrnes, V.A., Eye injuries in nuclear warfare, *Military Medicine*, April 1956, pp. 286-92.
35. Cogan, D.G., Donaldson, D.D., and Reese, A.B., Clinical and pathological characteristics of radiation cataracts, *AMA Arch. Ophthalmol.* **47**: 55 (1955).
36. Duke-Elder, S., *Textbook of Ophthalmology*, Vol. 6, pp. 6569-71, Mosby, St. Louis, 1954.
37. Hirose, K. and Fugino, T., Cataracts due to atomic bomb, *Acta Ophthalmol. Japon.* **54**: 449 (1950).
38. Sinskey, R.M., The status of lenticular opacities caused by atomic radiation, *Amer. J. Ophthalmol.* **39**: 285 (1955).
39. Fujinaga, Y., One hundred-fifty cases of A-bomb cataracts, *Jap. Rev. Clin. Ophthalmol. (Ganka Rinsho Iho)* **65**: 101-3 (1971).
40. Merriam, G.R. Jr. and Focht, E.F., A clinical study of radiation cataracts and the relationship to dose, *Amer. J. Roentgenol. Radium Ther. Nucl. Med.* **77**: 759-85 (1957).
41. Greulich, W.W. and Pyle, S.I., *Radiographic Atlas of Skeletal Development of the Hand and Wrist*, 2nd ed., Stanford University Press, Stanford, Calif., 1959.
42. Greulich, W.W., Dorfman, R.I., Catchpole, H.R., Solomon, C.I., and Culotta, C.S., Somatic and endocrine studies of puberal and adolescent boys, in *Monographs of the Society for Research in Child Development*, Vol. 7, No. 3, National Research Council, Washington, D.C., 1942.
43. Shuttleworth, F.K., The adolescent period: a pictorial atlas, in *Monographs of the Society for Research in Child Development*, Vol. 14, No. 50, Child Development Publications, Evanston, Ill., 1951.
44. Belsky, J.L. and Blot, W.J., Adult stature in relation to childhood exposure to the atomic bombs of Hiroshima and Nagasaki, *Amer. J. Pub. Health Nat. Health* **65**: 489-94 (1975).
45. Sutow, W.W. and Conard, R.A., Effects of ionizing radiation in children, *J. Pediat.* **67**: 658-73 (1965).
46. Dahl, L.K. and Love, R.A., Etiological role of sodium chloride intake in essential hypertension, *J.A.M.A.* **164**: 397-400 (1957).
47. Upton, A.C., Ionizing radiation and the aging process, *J. Gerontol.* **12**: 306 (1957).
48. Casarett, G.W., Similarities and contrasts between radiation and time pathology, *Advan. Gerontol. Res.* **1**: 109 (1964).
49. Upton, A.C., Kimball, A.W., Furth, J., Christenberry, K.W., and Bennett, W.H., Some delayed effects of atomic bomb radiation in mice, *Cancer Res.* **20**: 1 (1960).
50. Seltser, R. and Santwall, P.E., The influence of occupational exposure to radiation on the mortality of American radiologists and other medical specialists, *Amer. J. Epidemiol.* **81**: 2 (1965).
51. Warren, S. and Lombard, O.M., New data on the effects of ionizing radiation on radiologists, *Arch. Environ. Health* **13**: 415 (1966).
52. Anderson, R.E., Key, C.R., Yamamoto, T., and Thorslund, T., Aging in Hiroshima and Nagasaki atomic bomb survivors, *Amer. J. Pathol.* **75**: 1-11 (1974).
53. Hollingsworth, J.W., Ishii, G., and Conard, R.A., *Skin aging and hair graying, Hiroshima*, Atomic Bomb Casualty Commission Technical Report T-60, 1960.
54. Hollingsworth, J.W., Hashizume, A., and Jablon, D., Correlations between tests of aging in Hiroshima subjects, an attempt to define "physiological age," *Yale J. Biol. Med.* **38**: 11 (1965).
55. Beebe, G.W., Kato, H. and Land, C.E., Mortality and radiation dose, atomic bomb survivors, 1950-1966, in *4th Int. Radiat. Res. Conf., Evian, 1970*, TR 11-70, ABCC, NAS-NRC, 1970.
56. Anderson, R.E., Longevity in radiated human populations with particular reference to the atomic bomb survivors, *Amer. J. Med.* **55**: 643-56 (1973).
57. Conard, R.A., An attempt to quantify some clinical criteria of aging, *J. Gerontol.* **15**: 358-65 (1960).
58. Conard, R.A., Lowrey, A., Eicher, M., Thompson, K., and Scott, W.A., Aging studies in a Marshallese population exposed to radioactive fallout in 1954, in *Radiation and Aging*, pp. 345-60, P.J. Lindop and G.A. Sacher, Editors, Taylor and Francis, London, 1966.
59. Conard, R.A., Demoise, C.F., Scott, W.A., and Makar, M., Immunohematological studies of Marshall Islanders sixteen years after fallout radiation exposure, *J. Gerontol.* **26**: 28-36 (1971).
60. Conard, R.A., Effects of ionizing radiations on aging and life shortening in human populations, *Front. Radiat. Ther. Oncol.* **6**: 486-98 (1972).
61. Yang, H. and Conard, R.A., Effect of aging on acetate incorporation in nuclei of lymphocytes stimulated with phytohemagglutinin, *Life Sci. Pt. 2*, **11**: 677-84 (1972).
62. Walford, R.L., *The Immunologic Theory of Aging*, p. 31, Williams & Wilkins, Baltimore, 1969.
63. Demoise, C.F. and Conard, R.A., Effects of age and radiation exposure on chromosomes in a Marshall Islands population, *J. Gerontol.* **27**: 197-201 (1972).
64. West, K.M., Diabetes in American Indians and other native populations of the New World, *Diabetes* **23**: 841 (1974).
65. Sloan, N.R., Ethnic distribution of diabetes mellitus in Hawaii, *J.A.M.A.* **183**: 123 (1963).
66. Prior, I.A.M., A health survey in a rural Maori community with particular emphasis on cardiovascular, nutritional, and metabolic findings, *New Zealand Med. J.* **61**: 333 (1962).
67. Prior, I.A.M., The epidemiology of diabetes in Polynesians and Europeans in New Zealand and the Pacific, *New Zealand Med. J.* **65**: 375 (1966).
68. Prior, I.A.M., Ross, S.S., Harvey, H.P.S., and Davidson, F., Hyperuremia, gout, and diabetic abnormality in Polynesian people, *Lancet* **1**: 336 (1966).
69. Prior, I.A.M., The price of civilization, *Nutrition Today* **6**: 2 (1971).
70. Tulloch, J.A., *Diabetes Mellitus in the Tropics*, pp. 26-31, E. and S. Livingstone, London, 1962.
71. Lisco, H. and Conard, R.A., Chromosome studies on Marshall Islanders exposed to fallout radiation, *Science* **157**: 445-7 (1967).
72. Ishihara, T. and Kumatori, T., Chromosome aberrations in human leukocytes irradiated *in vivo* and *in vitro*, *Acta Haematol. Japon.* **28**: 291 (1965).

73. Bender, M.A. and Gooch, P.C., Somatic chromosome aberrations induced by human whole-body irradiation: the "Recuplex" criticality accident, *Radiat. Res.* **29**: 568 (1966).
74. Bloom, A.D., Neriishi, S., Kamada, N., Iseki, T., and Keehn, R.J., Cytogenetic investigations of survivors of the atomic bombings of Hiroshima and Nagasaki, *Lancet* **2**: 672 (1966).
75. Popp, R.A., Bailiff, E.G., Hirsch, G.P., and Conard, R.A., Errors in human hemoglobin as a function of age, *Gerontologia*, in press.
76. Dayhoff, M.O., Hunt, L.T., McLaughlin, P.J., and Barker, W.C., Globins, in *Atlas of Protein Sequence and Structure*, pp. 17-25, M.O. Dayhoff, Editor, Nat. Biomed. Res. Found., Silver Spring, Md., 1972.
77. Loftfield, R.B., The frequency of errors in protein biosynthesis, *Biochem. J.* **89**: 82-92 (1963).
78. Crick, F.H.C., The origin of the genetic code, *J. Mol. Biol.* **38**: 367-79 (1968).
79. Steward, A. and Kneale, G.W., Radiation dose effects in relation to obstetric x rays and childhood cancers, *Lancet* **1**: 1185-8 (1970).
80. Heyssel, R., Brill, A.B., Woodbury, L.A., Nishimura, E.T., Ghese, T., Hoshino, T., and Yamasaki, M., Leukemia in Hiroshima atomic bomb survivors, *Blood* **15**: 313-31 (1960).
81. Neel, J.V. and Schull, W.J., Editors, *The Effect of the Atomic Bombs on Pregnancy Termination in Hiroshima and Nagasaki*, Publ. No. 461, Nat. Acad. Sci.–Nat. Res. Council, Washington, D.C., 1956.
82. Neel, J.V., Ferrell, R.E., and Conard, R.A., The frequency of "rare" protein variants in Marshall Islanders and other Micronesians, *Amer. J. Hum. Genet.*, in press.
83. Dobyns, B.M. and Bertozzi, G., The identification of cold thyroid lesions at operation and its place in the surgical management of carcinoma of the thyroid, *Ann. Surg.* **17**: 703-10 (1970).
84. Dobyns, B.M., Vickery, A.L., Maloof, F., and Chapman, E.M., Functional and histological effects of therapeutic doses of radioactive iodine on the thyroid of man, *J. Clin. Endocrinol. Metab.* **13**: 548-67 (1953).
85. Klassovskii, Iu.A., Dependency of irradiation effect on determination of dose in thyroid histological structures, in *Sb. Mater. Radiatsionnaia Endokrinologiia*, A.A. Voitkevich, Editor, Akad. Med. Nauk SSSR, 1967, pp. 40-2 (Trans.), NIH-71-199.
86. Lawrence, A.M., Wilber, J.F., and Hogan, T.C., The pituitary and primary hypothyroidism, *Arch. Intern. Med.* **132**: 327 (1973).
87. Bernstein, R. and Robbins, J., Intermittent therapy with L-thyroxine, *N. Engl. J. Med.* **281**: 144-8 (1969).
88. Larsen, P.R., Direct immunoassay of triiodothyronine in human serum, *J. Clin. Invest.* **51**: 1939 (1972).
89. Larsen, P.R., Dockalova, J., Sipula, D., and Wu, F.M., Immunoassay of thyroxine in unextracted human serum, *J. Clin. Endocrinol. Metab.* **37**: 177-82 (1973).
90. Pittman, J.A., Dailey, G.E., and Beshi, R.J., Changing normal values for thyroidal radioiodine uptake, *N. Engl. J. Med.* **280**: 1431-4 (1969).
91. Stolc, V., Stimulation of iodoprotein and thyroxine formation on human leukocytes by phagocytosis, *Biochem. Biophys. Res. Commun.* **45**: 159-66 (1971).
92. Robbins, J. and Rall, J.E., Proteins associated with the thyroidal hormones, *Physiol. Rev.* **40**: 415-89 (1960).
93. van Herle, A.J., Uller, R.P., Matthews, N.L., and Brown, J., Radioimmunoassay for measurement of thyroglobulin in human serum, *J. Clin. Invest.* **52**: 1320-7 (1973).
94. Bruger, M., Hinton, J.W., and Lough, W.G., The iodine content of blood, urine, and saliva of normal persons in the New York City area, *J. Lab. Clin. Med.* **26**: 1942-4 (1941).
95. *Textbook of Medicine (Cecil-Loeb)*, 13th ed., p. 1424, P.B. Beeson and W. McDermott, Editors, Saunders, Philadelpha, 1971.
96. Wayne, E.J., Kontras, D.A., and Alexander, W.D., *Clinical Aspects of Iodine Metabolism*, F.A. Davis Co., Philadelphia, 1964.
97. Maloof, F., Dobyns, B.M., and Vickery, A.L., The effects of various doses of radioactive iodine on the function and structure of the thyroid of the rat, *Endocrinology* **50**: 612-38 (1952).
98. Doniach, I., Comparison of the carcinogenic effect of x-irradiation with radioactive iodine on the rat's thyroid, *Brit. J. Cancer* **11**: 67-76 (1957).
99. Lindsay, S. and Chaikoff, I.L., The effects of irradiation on the thyroid gland with particular reference to the induction of thyroid neoplasms: A review, *Cancer Res.* **24**: 1099-1107 (1964).
100. Hollingsworth, D.R., Hamilton, H.B., Tamagaki, H., and Beebe, G.W., Thyroid disease: A study in Hiroshima, Japan, *Medicine (Baltimore)* **42**: 47 (1963).
101. Wood, J.W., Tamagaki, H., Neriishi, S., Sato, T., Sheldon, W.F., Archer, P.G., Hamilton, H.B., and Johnson, K.G., Thyroid carcinoma in atomic bomb survivors, Hiroshima and Nagasaki, *Amer. J. Epidemiol.* **89**: 4-14 (1969).
102. Sampson, R.J., Key, C.R., Buncher, C.R., and Iijima, S., Thyroid carcinoma in Hiroshima and Nagasaki, I: Prevalence of thyroid carcinoma at autopsy, *J.A.M.A.* **209**: 65-70 (1969).
103. Pochin, E.E., Frequency of induction of malignancies in man by ionizing radiation, in *Handbuch der medizinischen Radiologie*, pp. 341-55, O. Olsson et al., Editors, Springer-Verlag, Berlin, 1972.
104. Belsky, J.L., Tachikawa, K., and Jablon, S., The health of the atomic bomb survivors: A decade of examinations in a fixed population, *Yale J. Biol. Med.* **46**: 284-96 (1973).
105. Pincus, R.A., Reichlin, S., and Hempelmann, L.H., Thyroid abnormalities after radiation exposure in infancy, *Ann. Intern. Med.* **66**: 1154-64 (1967).
106. Toyooka, E.T., Pifer, J.W., and Hempelmann, L.H., Neoplasms in children treated with x-rays for thymic enlargement, III: Clinical description of cases, *J. Nat. Cancer Inst.* **31**: 1379-405 (1963).
107. Hempelmann, L.H., Risk of thyroid neoplasms after irradiation in childhood, *Science* **160**: 159-63 (1969).
108. *Cancer Incidence in Five Continents, International Union Against Cancer, 1970 Study*, Vol. 2, R. Doll, C. Muir, and T. Waterhouse, Editors, Springer-Verlag, New York, 1970.

109. HEMPELMANN, L.H., HALL, W.J., PHILLIPS, M., COOPER, R.A., AND AMES, W.R., Neoplasms in persons treated with x-rays in infancy: Fourth survey in 20 years, *J. Nat. Cancer Inst.*, in press (1975).
110. MODAN, B., BAIDATZ, D., MART, H., STEINITZ, R., AND LEVIN, S.G., Radiation-induced head and neck tumours, *Lancet* **1**: 277-8 (1974).
111. HARLEY, N.H., ALBERT, R.E., SHORE, R., AND PASTERNACK, B.S., Follow-up study of patients treated by x-ray epilation for tinea capitis, VI: Estimation of the dose to the thyroid and pituitary glands and other structures of the head and neck, *Arch. Environ. Health*, in press (1975).
112. REFETOFF, S., HARRISON, J., KARANFILSKI, B.T., KAPLAN, E.L., DEGROOT, L.J., AND BEKERMAN, C., Continuing occurrence of thyroid carcinoma after irradiation to the neck in infancy and childhood, *N. Engl. J. Med.* **292**: 171-5 (1975).
113. SHELINE, G.E., LINDSAY, S., MCCORMACK, K.R., AND GALANTE, M., Thyroid nodules occurring late after treatment of thyrotoxicosis with radioiodine, *J. Clin. Endocrinol. Metab.* **22**: 8-18 (1962).
114. MCDOUGAL, I.R., Thyroid cancer after iodine-131 therapy, *J.A.M.A.* **227**: 438 (1974).
115. KREPS, E.M., KREPS, S.M., AND KREPS, S.L., Treatment of hyperthyroidism with sodium iodide I^{131}: Carcinoma of the thyroid after 20 years, *J.A.M.A.* **226**: 774-5 (1973).
116. HAMBURGER, J.I. AND MEIER, D.A., Cancer following treatment of an autonomously functioning thyroid nodule with sodium iodide I^{131}, *Arch. Surg.* **103**: 762-4 (1971).
117. DOLPHIN, G.W., The risk of thyroid cancers following irradiation, *Health, Phys.* **15**: 219-28 (1968).
118. POCHIN, E.E., Radiation exposure from the use of radioiodine in thyroid disease, *Proc. Roy. Soc. Med.* **57**: 564-5 (1964).
119. MACGREGOR, A.G., in *The Thyroid and Its Diseases*, A.S. Mason, Editor, Pitman Medical Publishing Co., London, 1963.
120. DONIACH, I., Effects of radiation on thyroid function and structure, in *Handbook of Physiology* (Sect. 7: Endocrinology; III: Thyroid), pp. 359-75, M. Greer and D.H. Solomon, Editors, Williams and Wilkins, 1974.
121. OTWAY, H.J. AND ERDMANN, R.C., Leukemia and thyroid carcinoma: A comparison of late mortality risks from reactor accidents, *Nucl. Safety* **11**: 482-9 (1970).
122. DOBYNS, B.M., SHELINE, G.E., WORKMAN, J.B., TOMKINS, E.A., MCCONAHEY, W.M., AND BECKER, D.V., Malignant and benign neoplasms of the thyroid in patients treated for hyperthyroidism (a report of the Cooperative Thyrotoxicosis Therapy Follow-up Study), *J. Clin. Endocrinol. Metab.* **38**: 976-98 (1974).
123. BEACH, S.A. AND DOLPHIN, G.W., A study of the relationship between x-ray dose delivered to the thyroids of children and the subsequent development of malignant tumors, *Phys. Med. Biol.* **6**: 583 (1962).
124. *Report of the United Nations Scientific Committee on the Effects of Atomic Radiation, Suppl. No. 12* (A/5814), United Nations, New York, 1964.
125. GOOLDEN, A.W.G., Carcinoma of the thyroid following irradiation, *Brit. Med. J.* **2**: 954-5 (1958).
126. SHELLABARGER, C.J., BOND, V.P., AND CRONKITE, E.P., Studies on radiation-induced mammary gland neoplasia in the rat, VII: The effects of fractionation and protraction of sublethal total-body irradiation, *Radiat. Res.* **17**: 101-9 (1962).
127. MARKS, S. AND BUSTAD, L.K., Thyroid neoplasms in sheep fed radioiodine, *J. Nat. Cancer Inst.* **30**: 661-73 (1963).
128. BOOK, S.A. AND BUSTAD, L.K., Effects of radioiodine and x-ray on beagle pups, in *Annual Report, 1973, Radiobiology Laboratory*, pp. 137-9, UCD 472-120, June 1973.
129. SAENGER, E.L., SALTZER, R.A., STERLING, T.D., AND KEREIAKES, J.G., Carcinogenic effects of I^{131} compared with x-irradiation: A review, *Health Phys.* **9**: 1371-84 (1963).
130. DOLPHIN, G.W. AND BEACH, S.A., The relationship between radiation dose delivered to the thyroids of children and the subsequent development of malignant tumors, *Health Phys.* **9**: 1385-90 (1963).
131. PILCH, D.Z., KAHN, G.R., KETCHAM, A.S., AND HENSAN, D., Thyroid cancer after radiation, I: Diagnostic procedures in children, *Pediatrics* **51**: 898-902 (1973).
132. LEROY, G., Personal communication, 1973.
133. VASILENKO, I.Ia. AND KLASSOVSKII, Iu.A., Remote consequences of thyroid irradiation with radioactive iodine isotopes, in *Sb. Mater. Radiatsionnaia Endokrinologiia*, A.A. Voitkevich, Editor, Akad. Med. Nauk SSSR, 1967, pp. 17-18 (Trans.), NIH-71-198.
134. WALINDER, G. AND SJÖDÉN, A., Effect of irradiation on thyroid growth in mouse foetuses and goitrogen challenged adult mice, *Acta Radiol. Ther. Phys. Biol.* **10**: 579-92 (1971).
135. WALINDER, G., JONSSON, C.-J., AND SJÖDÉN, A., Dose rate dependence in the goitrogen stimulated mouse thyroid, *Acta Radiol. Ther. Phys. Biol.* **11**: 24-36 (1972).
136. DUNNING, G., Two ways to estimate thyroid dose from radioiodine in fallout, *Nucleonics* **14** (2): 38-41 (1956).
137. COLE, R., *Inhalation of Radioiodine From Fallout: Hazards and Countermeasures*, Defense Civil Preparedness Agency, Environmental Science Associates, Burlingame, Calif., Aug. 1972.
138. RUSSEL, K.P., ROSE, H., AND STARR, P., The effects of radioactive iodine in maternal and fetal thyroid function during pregnancy, *Surg. Gynecol. Obstet.* **104**: 560-4 (1957).
139. FISHER, W.D., VOORHESS, M.L., AND GARDNER, L.I., Congenital hypothyroidism in infant following maternal I^{131} therapy, with a review of hazards of environmental radioisotope contamination, *J. Pediat.* **62**: 132-46 (1963).
140. HAMILL, G.C., JARMAN, J.A., AND WYNNE, M.D., Fetal effects of radioactive iodine therapy in a pregnant woman with thyroid cancer, *Amer. J. Obstet. Gynecol.* **81**: 1081-23 (1961).
141. RAY, E.W., STERLING, K.S., AND GARDNER, L.I., Congenital cretinism associated with I^{131} therapy, *A.M.A. J. Dis. Child.* **98**: 112-13 (1959).
142. VAN MIDDLESWORTH, L., Factors influencing the thyroid uptake of iodine isotopes from nuclear fission: A review, *Health Phys.* **9**: 1197-1211 (1963).

143. EVANS, T.C., KRETZCHMAR, R.H., HODGES, R.E., AND SONG, C.W., Radioiodine uptake studies of the human fetal thyroid, *J. Nucl. Med.* **8**: 157-60 (1967).
144. HODGES, R.E., EVANS, T.C., BRADBURY, J.T., AND KEETTEL, W.C., The accumulation of radioactive iodine by human fetal thyroids, *J. Clin. Endocrinol. Metab.* **15**: 661-7 (1955).
145. MAYS, C.W., University of Utah, Personal communication, 1974.
146. CONARD, R.A., A case of acute myelogenous leukemia following fallout radiation exposure, *J.A.M.A.* **232**: 1356-7 (1975).
147. HENSHAW, P.E. AND HAWKINS, J.W., Incidence of leukemia in physicians, *J. Nat. Cancer Inst.* **4**: 339-46 (1944).
148. MARCH, H.C., Leukemia in radiologists, *Radiology* **43**: 275-8 (1944).
149. COURT-BROWN, W.M. AND DOLL, R., Mortality from cancer and other causes after radiotherapy for ankylosing spondylitis, *Brit. Med. J.* **2**: 1327-32 (1965).
150. LANG, R.D., MOLONEY, W.C., AND YAMAWAKI, T., Leukemia and atomic bomb survivors. I. General observations, *Blood* **9**: 574-85 (1954).
151. MOLONEY, W.C., Leukemia and survivors of atomic bombing, *N. Engl. J. Med.* **253**: 88-90 (1955).
152. ANDERSON, R.E., Symposium on the delayed consequences of exposure to ionizing radiation: pathology studies at the Atomic Bomb Casualty Commission, Hiroshima and Nagasaki, 1954-1970, *Hum. Pathol.* **2**: 469-573 (1971).
153. PIERRE, R., Preleukemic states, *Sem. Hematol.* **11**: 73-92 (1974).
154. LINMAN, J.W. AND SAARNI, M.I., The preleukemic syndrome, *Sem. Hematol.* **11**: 93-100 (1974).
155. MILLER, C.E. AND STEINGRAHER, O.J., Measurements on some residents of the Marshall Islands, *Argonne National Laboratory Semiannual Report*, ANL 5755, pp. 53-7, 1957.
156. *A Radiological Study of Rongelap Atoll, Marshall Islands, During 1954-55, Applied Fisheries Laboratory, University of Washington, Seattle*, Office of Technical Services, U.S. Department of Commerce, Washington, D.C., 1955.
157. *Radiological Resurvey of Rongelap and Ailingnae Atolls, Marshall Islands, October-November, 1955, Applied Fisheries Laboratory, University of Washington, Seattle*, Office of Technical Services, U.S. Department of Commerce, Washington, D.C., 1955.
158. LOWMAN, F.G., PALUMBO, R.F., AND SOUTH, D.J., *The Occurrence and Distribution of Radioactive Nonfission Products in Plants and Animals of the Pacific Proving Ground*, University of Washington Fisheries Laboratory Report UWFL-51, Technical Information Service Extension, Oak Ridge, pp. 1-61, June 1957.
159. WELANDER, A.D., *Radiological Studies of the Fish Collected at Rongelap and Ailingnae Atolls, July 1957*, Office of Technical Services, U.S. Department of Commerce, Washington, D.C., 1958.
160. FOSBERG, F.R., Long-term effects of radioactive fallout on plants? *Atoll Research Bulletin* (Pacific Science Board, NAS, Washington, D.C.) **No. 61**: 1-11 (May 15, 1959).
161. WALKER, R.B., HELD, E.E., AND GESSEL, S.P., Radiocesium in plants grown on Rongelap Atoll soil, in *Recent Advances in Botany*, pp. 1363-7, University of Toronto Press, 1961.
162. HELD, E.E., Qualitative distribution of radionuclides at Rongelap Atoll, in *Radioecology (Proc. Symp., Fort Collins, Colorado, 1961)*, pp. 167-9, V. Schultz and A.W. Klement, Jr., Editors, Rheinhold, New York, 1963.
163. CHAKRAVARTI, D. AND HELD, E.E., Chemical and radiochemical composition of the Rongelap diet, *J. Food Sci.* **28**: 221-8 (1963).
164. HELD, E.E., GESSEL, S.P., AND WALKER, R.B., *Atoll Soil Types in Relation to the Distribution of Fallout Radionuclides*, Laboratory of Radiation Biology, University of Washington, Seattle, UWFL-92, 1965.
165. GREENHOUSE, N.A. ET AL., *Radiological Survey of Bikini, 1974*, To be published as BNL report.
166. PALMER, H.E. AND ROESCH, W.C., A shadow shield whole-body counter, *Health Phys.* **11**: 1213 (1965).
167. COHN, S.H., CONARD, R.A., GUSMANO, E.A., AND ROBERTSON, J.S., Use of a portable whole-body counter to measure internal contamination in a fallout-exposed population, *Health Phys.* **9**: 15 (1963).
168. COHN, S.H. AND GUSMANO, E.A., The determination of body burdens of radionuclides by computer analysis of gamma-ray spectral data, *Health Phys.* **11**: 109 (1965).
169. *Recommendations of the International Commission on Radiological Protection, Report of Committee II on Permissible Dose for Internal Radiation (1959)*, ICRP Publ. 2, Pergamon, New York, 1959.
170. BEASLEY, T.M., HELD, E.E., AND CONARD, R.A., Iron-55 in Rongelap people, fish and soils, *Health Phys.* **22**: 245-50 (1972).
171. BECK, H.L., BENNETT, B.G., AND MCCRAW, T.F., *External Radiation Levels on Bikini Atoll, May 1967, December 1967*, US AEC Health and Safety Laboratory, New York, HASL-190.
172. HELD, E.E., *Radiological Resurvey of Animals, Soils and Groundwater at Bikini Atoll, 1969*, University of Washington, College of Fisheries, Laboratory of Radiation Ecology, Seattle, NVO-269-8, Nov. 1969.
173. *Eniwetok Radiological Survey*, US AEC Nevada Operations Office, Las Vegas, NVO-140, Vols. I, II, III, Oct. 1973.
174. MCCRAW, T.F. (US AEC Div. of Operational Safety, Germantown) AND LYNCH, O.D.T. JR. (US AEC Radiological Operations Div., Las Vegas), *Exposure Rate Reduction on Bikini Island Due to Concrete Dwellings*, June 1973.
175. SASAKI, Y., IIO, M., KAMADA, H., UEDA, H., AOYAGI, T., CHRISTOPHER, N.L., BAYLESS, T.M., AND WAGNER, H.N., Measurement of ^{14}C-lactose absorption in the diagnosis of lactase deficiency, *J. Lab. Clin. Med.* **76**: 824-35 (1970).
176. The nuclear reactor accident at Windscale, October 1957: environmental aspects, App. H (pp. 129-39) in *The Hazards to Man of Nuclear and Allied Radiations, A Second Report to the Medical Research Council*, Her Majesty's Stationery Office, London, Dec. 1960.
177. *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation*, Report of the Advisory Committee on Biological Effects of Ionizing Radiation, NAS-NRC, U.S. Gov't Printing Office, Washington, D.C., Nov. 1972.

Appendix 1

Chronological Listing of Events in the Marshall Islands

1946 Bikini people moved from home island to Rongerik Atoll (later evacuated to Kwajalein and finally settled on Kili Island in the southern Marshalls).

Kwajalein established as U.S. Navy Base.

Operation Crossroads at Bikini.

Trust Territory of the Pacific Islands established, administered by U.S. Navy.

1947 Administration of Trust Territory transferred to U.S. Department of the Interior.

1948 Eniwetok people moved to Ujelang.

1954 March 1: Fallout accident following detonation of Bravo, a thermonuclear device. Exposed people from Rongelap, Rongerik, and Utirik evacuated to Kwajalein Atoll for examination and treatment. Exposed people on a Japanese fishing vessel Fukuryo Maru (Lucky Dragon) return to Tokyo.

June: Utirik people returned to home atoll; Rongerik servicemen transferred to Tripler Army Hospital. Rongelap people resettled in temporary village at Ejit Island, Majuro Atoll.

September: Second medical examination of exposed Rongelap people. Decision made to examine Rongelap people annually, Utirik people every 3 years.

1957 New village constructed at Rongelap. People returned after 3 years away. Medical examinations and periodic survey of environment continued.

1963 Epidemic of poliomyelitis at Rongelap and in rest of Marshall Islands.

At Kwajalein, Pacific Missle Range under U.S. Army Command established.

1964 Ex gratia compensation (~ $11,000 per person) granted Rongelap people by U.S. Congress.

Thyroid abnormalities begin to appear in Rongelap exposed people. Thyroid surgery performed on 3 children at the Guam Naval Hospital.

1965 - 1968 Thyroid surgery on 12 Rongelap people carried out in Boston.

1969 Cleanup of Bikini for return of inhabitants.

Five Marshallese had thyroid surgery in U. S.

1970 One Marshallese had thyroid surgery in Cleveland.

1971 Marshallese Congressman (Congress of Micronesia) visits Japan and invites a Japanese team to visit Rongelap. Team arrives but because of conflict over credentials they are denied visas and returned to Japan.

Return of two Bikini families to Bikini to live. BNL medical team assumes responsibility for radiological monitoring of returning Bikini and Eniwetok people.

Documentary movie Thyroid Neoplasia as a Late Sequella of Radioactive Fallout filmed in Marshall Islands. Shows the medical team in action.

1972 January: Marshallese Congressman accuses the U.S. of knowingly and consciously allowing the Marshallese people to be exposed to radioactive fallout in order to study the effect of radiation on human beings; accuses medical team of using Rongelap people as guinea pigs and not giving them proper medical examinations and adequate treatment. In March he told the Rongelap and Utirik people not to cooperate with the medical team. Annual medical survey not completed. Also 4 patients who had been operated for thyroid cancer and were to be reexamined at Tripler Army Hospital were stopped and told to return home.

April: Congress of Micronesia establishes a Special Joint Committee concerning the medical examinations at Rongelap and Utirik atolls. Chairman: Senator Olympio T. Borja.

August: Resident physician stationed in Marshall Islands.

September: Annual medical survey resumed, with cooperation from Congress of Micronesia and participation of four appointed medical observers from several countries. Comprehensive report generally favorable to the medical examinations published.

October/November: A young exposed male, found to have acute myelogenous leukemia, dies at National Cancer Institute, Bethesda, Maryland, and is returned for burial at Rongelap.

Decision made to conduct hematological surveys at 6-month intervals. Two Rongelap people had thyroid surgery in Cleveland.

1973 Eniwetok radiological survey completed. Report NYO 140 published.

Six Marshallese (3 Rongelap and 3 Utirik) had thyroid surgery in Cleveland.

1974 BNL survey group organized for radiological surveys in Marshall Islands. To be coordinated with medical surveys and personnel monitoring of returning Bikini and Eniwetok people.

Special bills passed by Congress of Micronesia regarding further compensation to Rongelap and Utirik people, and providing special benefits and hospitalization privileges in case of illness from any cause.

Documentary movie The Bikinians filmed at Bikini by the University of Georgia.

Three Rongelap people had thyroid surgery in Cleveland; one case exposed in utero.

LCU obtained for medical and radiological surveys. Christened Liktanur.

Appendix 2

Survey Participants 1954 - 1974

Name			Year 19'
Adamik, Emil	Technician	BNL	59,61,62
Anjain, Jeton, D.O.	Dental Officer	TTPI	64,72
Argonza, W.S.	Technician	US Navy,NRDL	54
Ash, Joseph A.	Technician	BNL	74
Bach, Sven A., M.D.	Physician	US Army,AFSWP	57
Barton, Johnny M., M.D.	Physician	US Air Force,AFSWP	58,
Bateman, John L,, M.D.	Int. Medicine	BNL	69
Bender, Byron, Ph.D.	Anthropologist	TTPI	64
Bien, Peter	Medex	TTPI	70,74
Blumberg, Baruch, M.D.	Endocrinologist	NIH	59
Bond, Victor, M.D.,Ph.D.	Physician	US Navy,NRDL	54
Boon, R.	Technician	TTPI	61
Border, W.K.	Technician	US Navy,NMRI	55,56
Brown, Robert A.	Technician	BNL	73,74
Browning,L.E., M.D.	Physician	US Army,AFSWP	54
Cannon, Bradford, M.D.	Surgeon	Mass.Gen. Hosp.	56,57,63
Carter, Edwin L., M.D.	Int. Medicine	US Navy,NMRI	57
Carter, Robert E., M.D.	Pediatrician	State U. Iowa	64
Carver, Russel K.	Parasitologist	USPHS	58
Chapman, W.H.	Rad. Scientist	US Navy,NMRI	54
Clareus, Douglas	Electronic Specialist	BNL	61-70;72-74
Clutter, W.G.	Technician	US Navy,NMRI	54-57
Cohn, Stanton H., Ph.D.	Scientist	BNL	54,59,61,74
Colcock, Bentley P.,M.D.	Surgeon	Lahey Clinic	67
Cole, William, M.D.	Radiologist	USPHS	72
Cook, Lawrence	Technician	BNL	63,65
Conard, Robert A., M.D.	Physician	BNL	54,56-74
Cronkite, Eugene P., M.D.	Hematologist	US Navy,NMRI	54,55
Deisher, Joseph B., M.D.	Physician	TTPI	66
Demoise, Charles F., Ph.D.	Scientist	BNL	68,69
Dobyns, Brown M., M.D.	Surgeon	Case Western Reserve Univ.	69,70,72,73
Dunham, Charles L., M.D.	Physician	AEC,DBM	57-59,65,73
Eicher, Maynard	Electronic Specialist	NMRI	57-59
Elanjo, Laijo	Technician	TTRI	74
Emil, Menassa, D.O.	Dental Officer	TTPI	67
Ezaki, Haruo, M.D.	Surgeon	Hiroshima School of Med.	72
Farr, R.S., M.D.	Hematologist	US Navy,NMRI	54
Flanagan, J	Technician	US Navy,NMRI	54
Gays, W.	Technician	TTPI	64,65
Gibbs, W.H.	Technician	US Navy,NRDL	54
Gideon, Kalman	Technician	TTPI	64,65
Gilmartin, James T.	Technician	BNL	59
Glassford, Kenneth	Technician	US Navy,NMRI	59
Goldman, Morris, Ph.D.	Parasitologist	USPHS	58
Gomez, Wentolin	Technician	TTPI	67,69
Greenhouse, Nathaniel	Rad. Scientist	BNL	74
Greenough, James J.	Technician	BNL	57,59
Griffin, David, M.D.	Physician	US Navy,NRDL	55
Gusmano, Ernest, Ph.D.	Rad. Technician	BNL	61,65

Survey Participants 1954 - 1974

Name			Year 19'
Hamby, J. W.	Technician	US Navy,NMRI	54-57
Hammerstrom, Richard, Ph.D.	Scientist	BNL	63
Hansell, R.E.	Technician	US Navy,NMRI	54
Hartley, Marion L.	Technician	US Navy,NMRI	58
Hayakawa, C., M.D.	Physician	Tokyo, Japan	72
Hechter, Hyman	Statistician	NRDL	57,58
Helkena, Jack, D.O.	Dental Officer	TTPI	60
Hendrie, J.C.	Technician	US Navy,NRDL	54
Heotis, Peter M.	Technician	BNL	73,74
Hicking Arobati, M.O.	Medical Officer	TTPI	62-68
Hollingsworth, James W., M.D.	Int. Medicine	ABCC, Japan	59
Huggins, C.E., M.D.	Int. Medicine	US Navy,NMRI	56
Humphrey, Douglas	Photographer	BNL	70
Iaman, John, M.O.	Medical Officer	TTPI	59
Jaffe, A.A., D.D.S.	Dentist	TTPI	60,61
Jesseph, Joseph E., M.D.	Surgeon	BNL	65
Jomule, Jude	Technician	TTPI	67,72
Jones, Irving	Technician	So. Nassau Comm. Hosp.	61-63
Karnofsky, David, M.D.	Oncologist	Sloan-Kettering Inst.	61
Kenny, John	Technician	US Navy	55
Knudsen, Knud, M.D.	Int. Medicine	BNL	71-74
Kumatori, Toshiuki, M.D.	Physician	NI Radiological Sci. Japan	64,72
Lanwi, Issac, M.O.	Medical Officer	TTPI	59,60,64,65
Larsen, Reed, M.D.	Endocrinologist	Univ. of Pittsburgh	72,74
LeRoy, G.V., M.D.	Int. Medicine	AEC, DBM	54
Lewis, William H., M.D.	Cardiologist	Memorial Hospital, N.Y.	59
Libby, Ernest	Technician	TTPI	67-69,71-73
Lowrey, Austin, Jr., M.D.	Ophthalmologist	US Army(Ret)	56-59,62,64,67, 71,72
Lyon, Harvey W., D.D.S.	Dentist	US Navy,NMRI	59
MacDonald, Eugene H., M.D.	Physician	TTPI	60,61
Makar, Michael S.	Technician	BNL	69-72
McPherson, S.D., M.D.	Ophthalmologist	US Navy,NMRI	55
Meyer, Leo M., M.D.	Hematologist	V.A.	61-65,67,72,74
Mizutani, Kosang	Technician	TTPI	67,72,74
Moloney, William C., M.D.	Hematologist	Boston City Hospital	62,63
Momotaro, Francis, D.O.	Dental Officer	TTPI	72
Murray, William G.	Photographer	NRDL	57,58
Obten, Antak	Technician	TTPI	62
Oh, Yang, H., Ph.D.	Scientist	BNL	71
Otto, James S.	Technician	US Navy, Naval Med. Center	58
Paglia, Donald E., M.D.	Hematologist	UCLA	68
Peck, William, M.D.	Physician	TTPI	72
Pochin, Edward, M.D.,FRPC	Radiologist	U. Coll. Med. School, London	72
Potter, David W.	Scientist	BNL	58,59
Rai, Kanti, R., M.D.	Pediatrician	BNL	70
Rall, Joseph E., M.D.	Endocrinologist	NIH	57,65,71
Richards, J.B., M.D.	Physician	US Navy,NMRI	56
Riklon, Ezra,M.O.	Medical Officer	TTPI	59-63,69,72,73
Riklon, Kimra	Technician	TTPI	72

Survey Participants 1954 - 1974

Name			Year 19'
Robbins, Jacob, M.D.	Endocrinologist	NIH	66,72
Robertson, James S., M.D.,Ph.D.	Scientist	BNL	54,58,65
Rothmann, John C.	Technician	BNL	74
Saul, Jospeh	Technician	TTPI	71,72
Schork, Paul K.	Technician	US Navy,NMRI	54,57
Scott, William A.	Technician	BNL	58-74
Severson, C.D.	Technician	US Navy,NMRI	56
Sharp, R.	Rad. Scientist	US Navy,NMRI	54
Shoniber, Sebio	Technician	TTPI	59-74
Shulman, N.R., M.D.	Hematologist	US Navy,NMRI	54
Sipe, Clyde	Technician	BNL	54,55,58
Smith, L.J.	Rad. Scientist	US Navy,NRDL	54
Smith, Robert F.	Photographer	BNL	59,67
Snow, Lloyd D.	Technician	US Navy,NMRI	56,59
Soras, Philipo	Technician	TTPI	61
Steele, John, M.D.	Pediatrician	TTPI	74
Strome, C.P.A.	Technician	US Navy,NMRI	54,55,56
Sutow, Wataru W., M.D.	Pediatrician	MD Anderson Hosp., Texas	58,59,61-63,65, 67-69,71,72
Tenorio, Pacifico	Technician	US Navy,NMRI	58,59
Tomesch, Charles	Physician	BNL	71
Urschel, Harold C., Jr., M.D.	Physician	US Navy,NMRI	58
Waithe, William	Technician	NY University	64
Watne, Alvin C., M.D.	Surgeon	Univ. of West Virginia	64
Weden, E.A., Jr., M.D.	Physician	US Navy,NRDL	54
Weldon, Thomas	Technician	BNL	67
Wolff, Jan, M.D.	Endocrinologist	NIH	74
Wolins, William, M.D.	Int. Medicine	BNL	58
Woodward, Kent T., M.D.	Radiologist	US Army,AFSWP	55
Zetkeia, Nelson	Technician	TTPI	61,66-74

Appendix 3

A Summary of the Findings over the 20 Year Period on the Japanese Fishermen Exposed to Fallout in 1954

On March 1 in 1954, 23 Japanese fishermen aged from 18 to 39 were exposed to radioactive fallout produced by the thermo-nuclear test explosion which was performed by the U. S. Authorities at Bikini lagoon. They were crew of a tuna-fishing boat "The 5th Lucky Dragon".

The location of the boat was 166°58' E. and 11°53' N.. At about 3.50 a.m. they saw a huge red light in the west and heard detonation-like sounds 7 - 8 minutes later while they were fishing for tuna. At about 7.00 a.m. white ashes began to fall on the boat which continued for about 4 1/2 hours. After 14 days navigation they returned to their harbor, Yaizu, on March 14, 1954. After landing, all the fishermen were found to have been injured by the radioactive materials. Seven of them were hospitalized to the Tokyo University Hospital and the other sixteen to the First National Hospital of Tokyo by March 28. They were discharged from both hospitals in May 1955, except one fatal case who died on September 23, 1954. After being discharged, most of them have been examined so far as possible on an anual basis.

A. State of Irradiation and Estimated Radiation Dose

When fallout fell most intensively they could not keep their mouths and eyes open. Fallout deposited on the deck as thick as their foot prints were marked. The persons were irradiated in the following three ways : 1) From the radioactive materials adhered to the skin 2) Externally from the radioactive materials in the cabins, on the deck etc. 3) Internally from the radioactive materials entered various organs.

The estimation of radiation dose to skin as well as the dose by internal exposure were difficult. On the other hand, the estimated

external radiation dose was approximately 170 - 600 rad for 14 days, about half or more being irradiated on the first day. The dose to each person differed depending on his behavior on the boat and the position of his cabin.

The integrated dose to thyroid glands from ^{131}I was inferred as about 20 - 120 rad. Urine samples which were collected at 4 weeks after the explosion revealed significant amount of radioactivity. However, the radioactivity decreased rapidly, e.g. at about 6 months post detonation the activity was barely detectable. In the analysis after 8 1/2 years and 10 years the levels of ^{137}Cs and ^{90}Sr in urine were the same to those of normal Japanese. At the same time the results of whole body counting showed no significant difference between fishermen and controls. The radioactivity in several organs of the fatal case was higher than controls, but low.

B. Clinical and Laboratory Findings

1. General symptoms and signs

Soon after the initial exposure most of the fishermen experienced anorexia, fatigue, and lachrymation, and in some of them nausea and vomiting occurred.

2. Skin lesions

Skin lesions were caused by beta irradiation. Shortly after the exposure, they suffered from erythema which was followed by edema, vesicle, erosion, ulceration or necrosis. Epilations were observed in 20 cases, especially 2 cases who did not wear hats during ash-fall revealed complete epilation. These skin lesions were similar to ordinary radiodermatitis histologically. The skin injries recovered gradually. At present, namely 20 years after the exposure, in a few cases depigmentation, pigmentation and capillary dilatation are observed. Atrophy of epidermis with narrowed stratum granulosum were clear in histological section of these area examined 10 years after the exposure.

3. Hematology

a. Leukocytes : Total number of leukocytes decreased gradually,

showing minimum count at 4th - 8th week. 5 cases revealed a count of less than 2,000/mm^3, 13 less than 3,000 and 5 less than 4,000. In one case, the leukocyte level depressed to 800. A correlation was found between these minimum count and the externally irradiated gamma doses of each individual. At first lymphopenia was noticed and then neutropenia became marked. Since 8th week recovery was proved. In many cases remarkable eosinophilia was observed at that time. In some cases immature neutrophils appeared in peripheral blood slightly.

b. Erythrocytes : In severe cases slight anemia was observed, accompanied by the depression of reticulocytes. Color indices were higher than 1.0. The Price-Jones curves of erythrocytes diameter were displaced to the right of normal one at first, which returned to almost normal after one year.

c. Platelets : Platelets counts showed increasing depression, reaching minimum at the 4th - 7th week (15,000 - 100,000/mm^3). Slight eoagulation disturbances observed in a few cases.

d. Bone marrow : In severe cases bone marrow was highly hypoplastic at the critical stage, which changed to hypoplastic and turned into almost normoplastic. Recovery was not complete even after a year. At the recovery stage coexistence of hypoplastic area and hyperplastic area was observed in histological sections.

e. Morphological abnormalities : Several morphological abnormalities, e.g. abnormal granules in lymphocytes or neutrophils, vacuoles in various leukocytes and megakaryocytes, giant nuclei and hypersegmentation of neutrophils, binuclear lymphocytes, abnormal mitosis of erythroblasts etc. were observed for about one year, especially at the critical and recovery stage. A little increase of "mitotically connected abnormalities" was found in bone marrow smears of a few cases after 10 years.

f. Recovery : The cumulative distribution curves of numbers of leukocytes, erythrocytes and platelets displaced to the left of normal

ones remarkably at the c ritical stage. Though the curves of erythrocytes and platelets lay on normal Japanese ones after 2 years, the curve of leukocytes still displaced slightly to the left of normal one even after 6 years.

4. Cytogenetics

Follow-up of chromosome observations in blood cells has been performed since 1964. Even 20 years after exposure, cells with chromosome abnormalities (both Cu and Cs cells) exist in the peripheral lymphocytes with much higher frequencies than in general population. The frequency of Cu cells (dicentrics + rings), however, was decreasing. On the other hand, Cs cells remained fairly constant in the frequency of around 2%. The frequencies of the chromosome abnormalities were found to be corresponding to the severity of injuries indicated by minimum leukocytes count at the critical stage. In the bone marrow, cells with chromosome abnormalities (Cs cells) occurred rather constantly with frequencies of more than 2% in all the 4 times of samplings carried out 13 - 17 years after exposure.

5. Spermatopoiesis

Number of spermatozoa decreased about 2 months after exposure, and azoospermia was found. Both fall of motility and morphological abnormalities of spermatozoa were also observed. Indications of recovery were noticed about 2 years after exposure. Then most of the patients got healthy children.

The testicle of the fatal case, which died 206 days after exposure, showed extremely reduced spermatopoiesis.

6. Other findings

Slight disturbances of the liver function were found in a few cases at the time of hospitalization. Later it became more obvious. One of the fishermen, who revealed remarkable hematological disturbances, died from liver damage. During follow-up studies elevated values of

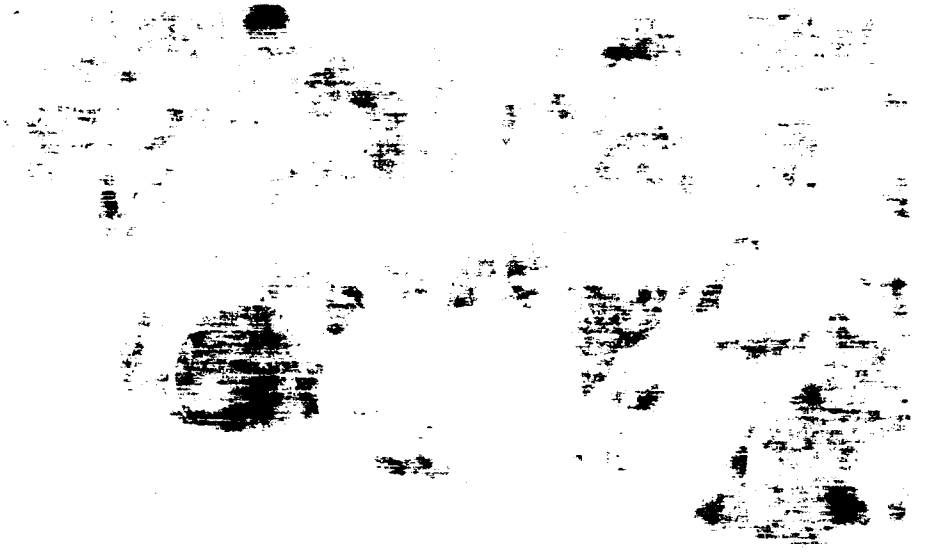
GOT and GPT were observed in several cases. In 1974, ascites were proved in 2 cases, one of which was accompanied by diabetes mellitus and sepsis. These have recovered already.

Ophthalmological examinations showed slight lenticular opacities in several cases. Its significance is not apparent yet.

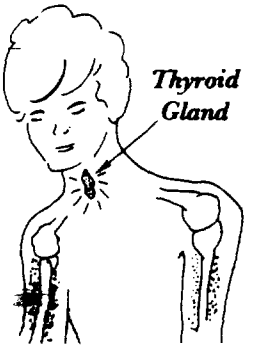
Other studies including thyroid studies are going on to detect late effects.

Toshiyuki Kumatori, M.D.

Head,
Division of Radiation Health,
National Institute of Radiological Sciences
Chiba-shi, Japan



Booklet Printed at Brookhaven National Laboratory
To Answer Questions of Rongelap and Utirik People



How does radiation cause diseases in our bodies?

Radiation in fallout caused injury to parts of the body. Later on these parts became sick and cannot do their job properly. There is very little radiation left in the body now.

Body scanner

How can we tell if we have radiation in our bodies?

It is difficult for you to know if you have radiation in your body because there is so little. Only the experts can tell by checking you every year with special machines.

How can we get rid of the radiation in our bodies, trees, ground, crabs, etc?

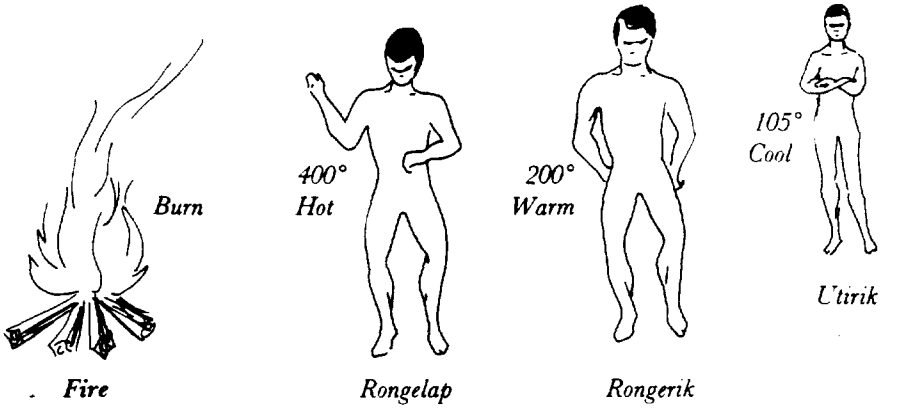
Your body will remove some of the radiation naturally when you use the benjo but this takes a long time. The only way to get rid of the radiation in the trees, soil, crabs, etc. is to collect them and dump them into the ocean. But, since the radiation is weak there is no reason to do this.

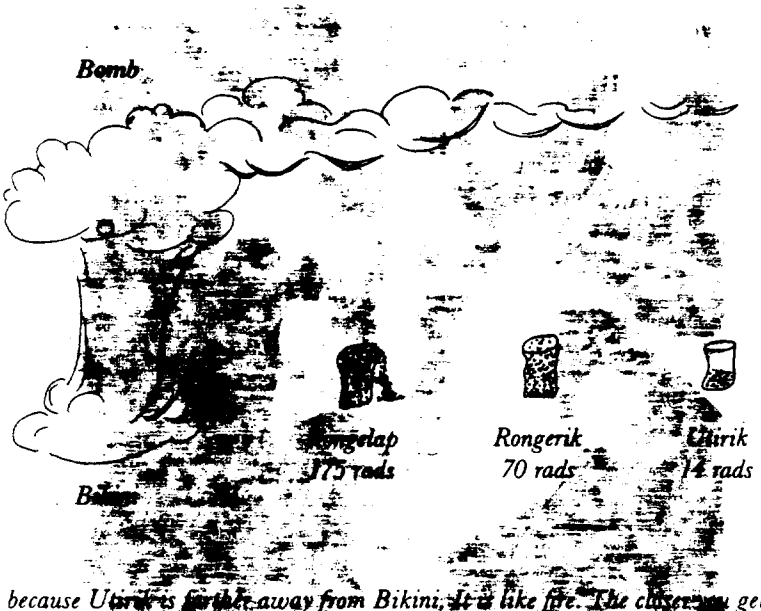
Lush growth on Rongelap

Did we people on Utirik get as much radiation as the people on Rongelap?

No, because Utirik is farther away from Bikini than Rongelap. One way to measure radiation is by counting the "rads". Below is the number of rads the people got in 1954:

People on Rongelap	175 rads
People on Ailinginae	69 rads
Americans on Rongerik	70 rads
People on Utirik	14 rads





No, because Utirik is farther away from Bikini. It is like fire. The closer you get to fire the hotter it gets. Heat is measured by degrees. Radiation is measured by rads.

Is there still radiation in the ground? How long will it last?

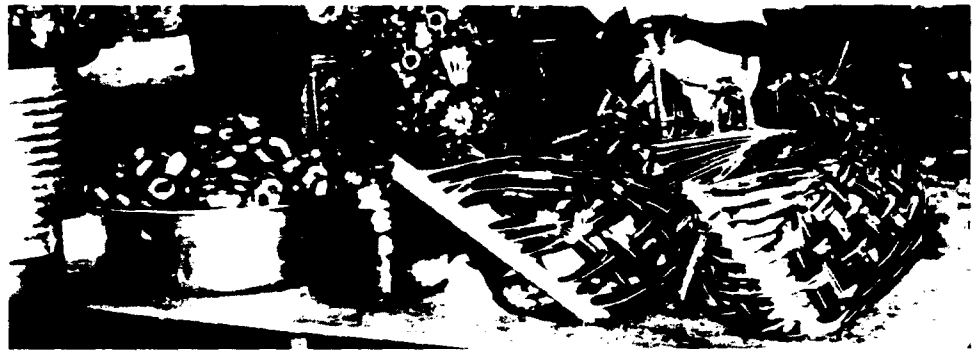
Yes, there is a little radiation left in the ground but it gets weaker and weaker as time goes by.

Woman washing clothes

It is like dirty clothes. If you wash them and lay them in the sun the dirt and poison will come away. The ground is too big to wash like clothes so the rain does it for you.

If the radiation is still in the soil of these islands, why is there no restriction of people moving into these islands?

The radiation in the ground is so weak that it is safe to live on the islands and eat the foods. There is no place in the world that does not have some radiation in the soil.



Feast on Rongelap

Why can't we eat coconut crab and arrowroot?

You can eat the coconut crabs from the southern islands of Rongelap if you do not eat more than three crabs per week. There is still some radiation in the crabs because they eat their old shells when they grow new ones. The people on Utirik may eat all the crabs they want. Arrowroot may be eaten on Rongelap and Utirik.

Is there anything else that we are not supposed to eat?

No, you may eat anything else that is good for you.

Person eating pandanus
You may eat anything else like pandanus.

If the U.S. can reach the moon, how come they did not know that the wind was going to be shifted over to the islands?

There were some mistakes made. The U.S. can reach the moon because the equipment used to get man to the moon is under his control. The direction of the wind is more difficult to predict because the wind is not controlled by man.

Are miscarriages caused by the fallout?

Some of the miscarriages in exposed Rongelap women during the first years after the fallout may have been due to radiation exposure. Since that time there were only a few miscarriages and this is normal. Women no longer have to worry about miscarriages due to radiation.

Children and women
They are healthy.

If I have trouble with my thyroid, will I be healthy again?

If you are treated properly and you follow the doctor's advice, you should be healthy and strong like anybody else. You should be examined every year so the doctor can find out if you have thyroid disease. If the doctors operate on your thyroid, they will give you medicine. If you listen to the doctor and keep taking your medicine, you will stay strong and healthy.

Since people who were not exposed to fallout have been living on the islands for some time, have the Brookhaven doctors found any signs of radiation sickness in them and if so will they be compensated?

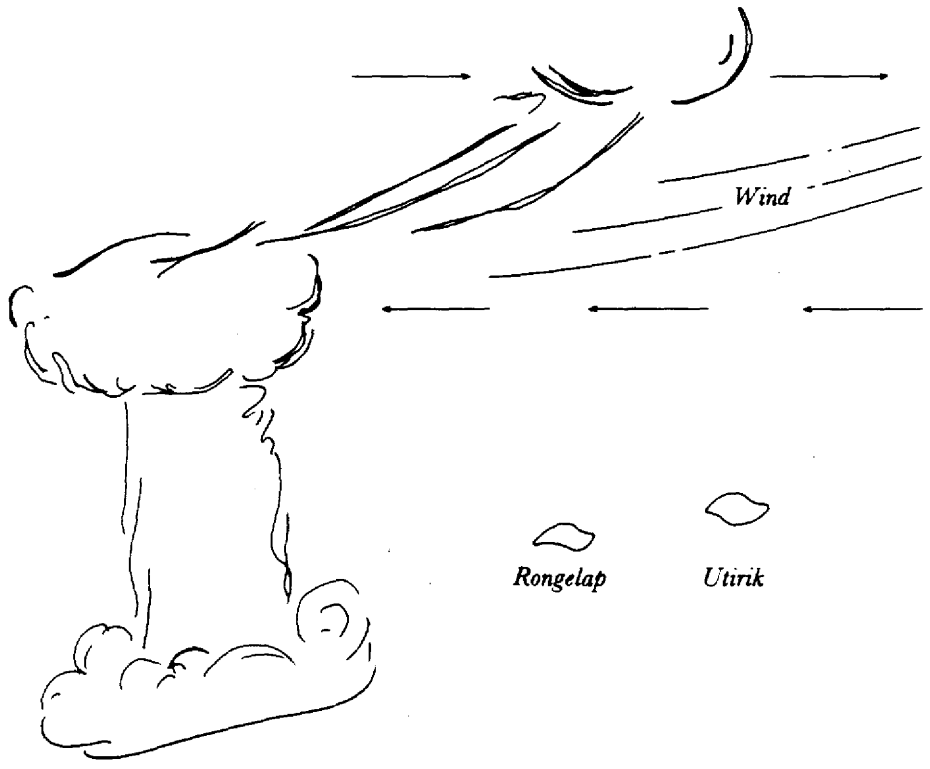
There is practically no chance that any of these people will develop any radiation sickness since the amount of radiation left on the islands is so small. If it did happen the government probably would consider compensation.

Why doesn't the AEC remove the radiation from the soil and plants?

It is impossible to do so and since the radiation is so small, there is no reason to do this.

Why didn't the U.S. explain to the people the dangers of the fallout before the test?

The people were not warned of the dangers of the fallout because the likelihood that fallout would occur outside the restricted area was considered too remote to justify warning the people. The occurrence of fallout in an unrestricted area after the Bravo Test was the first accidental event of this kind to happen after a nuclear weapon test.



Bomb explosion

Why did the U.S. not take extra precaution?

The U.S. took the precaution of trying to keep people out of the area where they expected fallout to occur.

I was not exposed to radiation so why do I have to be examined?

The doctors examine people who were not exposed because they need to know when the exposed people become sick, if their sickness is caused by radiation or something else. If you were not exposed and got sick and your brother was exposed and got the same sickness, then the doctors would suspect that the sickness was not caused by radiation. You do not have to be examined if you do not want to be. Remember, though, that you are helping your family and friends when you are examined. Also, you are receiving a free medical examination by the best doctors in the world.

Doctor examines a patient
You are examined to make sure nothing is wrong.

Why do I have to be examined every year?

Doctors still do not know everything about radiation sickness so they are checking you to make sure that you are healthy. They check you every year so that if you are sick, they can find it early and treat you. Doctor Conard's team treat many people every year, even when the sickness does not come from radiation. The Congress of Micronesia recommends that everyone take the examination.

Why are some people becoming sick now 20 years after the test?

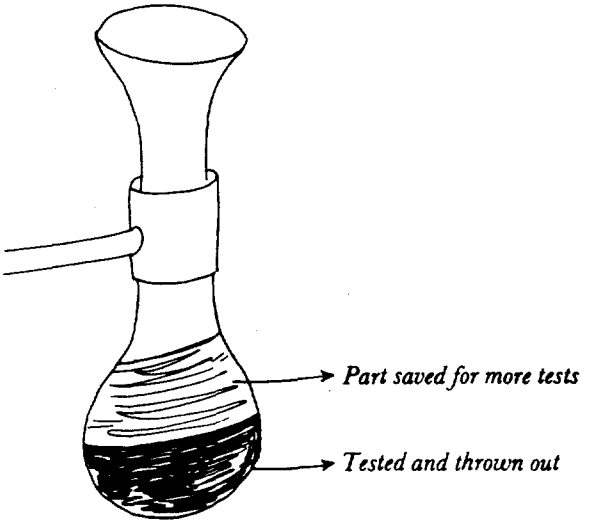
The radiation was in their bones and certain parts of their bodies such as the thyroid gland. Doctors still don't understand all about radiation disease. That is why they are checking you every year to make sure you are healthy.

Why do you have to take our blood? urine?

The doctors check your blood to find out if you have blood disease. They check your urine to find out if you have other diseases. For example, Dr. Conard found some people with diabetes (sugar sickness) which is not caused by radiation and he was able to give them medicine. Another reason is that the urine removes radiation from your body so the doctors want to find out if you still have radiation by looking in your urine.

Why do they take our blood for examination and then throw some of it away?

The doctors take your blood to study it. They need to study your blood three or four times so they want to make sure that they have enough blood. It is better to take more blood than not enough because if they need more, they don't have to stick the needle into your arm again. You have plenty of blood so it won't hurt you to lose a little.



Blood test

Part of the blood is tested and can be thrown away.
Another part is saved for other tests at Brookhaven.

Why do you have to drill into our bones?

Radiation can cause blood disease and your blood is made inside of your bones. The doctors drill your bones to see if they are healthy.

Why do people die after they have their bones drilled?

Nobody can die from having their bones drilled. Drilling into your bones is not dangerous or harmful and does not cause death.

Dentist drilling teeth

Dentists drill your teeth, too, but you do not die.

One man became blind after the fallout . . . Will I become blind too?

No one became blind from fallout and we don't expect anyone to become blind from it since there was not enough radiation to cause blindness or eye trouble.

Why do we have to be taken away to the States to be operated?

To give you the best medical care possible. The best doctors, hospitals and equipment are in the States so you are sent there for treatment. When Lekoj Anjain was sick, he was taken to the National Institute of Health Hospital in Maryland. Even the President of the United States goes to this hospital when he is sick because they have the best doctors and the best equipment in the U.S. It was sad that Lekoj died but the doctors did everything they could to treat him.

Why do I need to take medicine for my thyroid?

If you had an operation and your thyroid gland was removed, the medicine will do the job that your thyroid used to do. So, you must keep taking your medicine. Some people who did not have the operation also take the medicine so that their thyroid glands do not get sick. It is very important that you take your medicine if you want to stay healthy.

What kind of radiation caused thyroid sickness and leukemia?

Thyroid sickness in the Rongelap people exposed to fallout was caused by damage to the thyroid gland from a "thyroid radiation poison." It took a long time for the sickness to show. A few unexposed Rongelap people as well as some people in the Marshall Islands and all over the world develop thyroid sickness, but it is not due to radiation. All the "thyroid poison" on Rongelap was gone when the people moved back. Leukemia is a disease of the blood. Some cases were caused by radiation in Japan and it is possible that Lekoj's leukemia may have been due to radiation. Time is about past for any more leukemia to be expected from radiation.

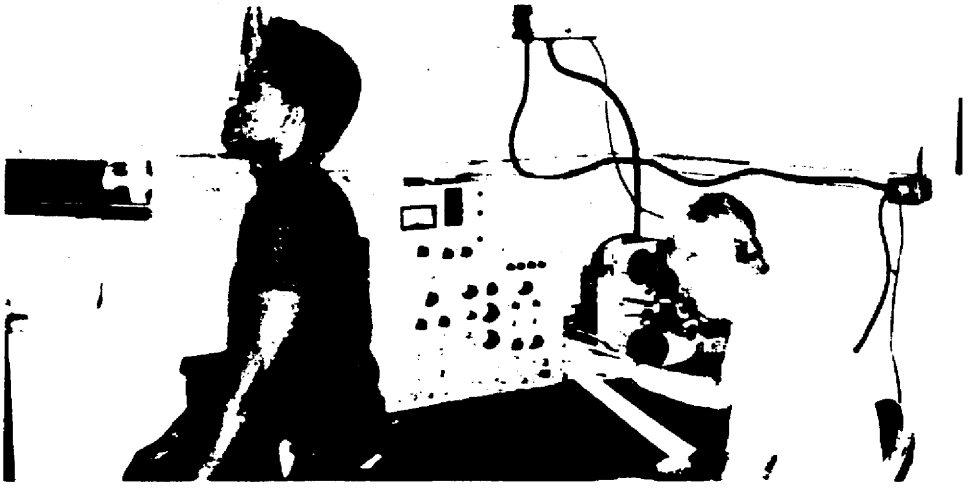
What will happen to the people who were not exposed to radiation but are now living on Rongelap and Utirik?

They don't have to worry because there is practically no chance that any of the people will get sick from radiation since there is so very little radiation left on the islands.

Feast - People
They are Rongelap people. They are healthy, too.

Why do the doctors feel our neck?

Your thyroid glands are in your neck. The doctors feel your neck to find out if you have a sickness in your thyroid gland. Another way to check your thyroid is with a special machine.



Thyroid examination
Special machine for checking thyroid gland.

What is the thyroid gland?

Your thyroid gland does a certain kind of work in your body. Your ears allow you to hear, your eyes make you see and your thyroid makes you grow normally and stay healthy. Because radiation affected their thyroid glands, some of the boys in Rongelap grew slowly after the fallout. In 1965, they were given special medicine and they grow normally. If you have a thyroid operation, it is very important that you take the medicine that is given to you. The medicine will do the same work as your thyroid and it will keep you healthy.

They were sick before. They are healthy now.

PRIVACY ACT MATERIAL REMOVED

What is cancer?

Cancer is a disease which happens when a certain part of your body becomes sick and does not do its job. The sickness can spread to other parts of your body. Cancer of the blood is called leukemia and that is the disease that had. Some kinds of cancer can be controlled by medicine and others must be stopped by operation. Some types of cancer, such as leukemia, cannot be cured. Cancer is dangerous but if the doctors examine you and find it early, they have a better chance to help you.

So far, what are the health findings? Are we healthier or sicker?

The medical examinations show that you are a healthy and strong people. The only significant differences have been the early fallout effects on the blood and skin, the higher number of miscarriages in 1957 and 1958 and the thyroid troubles.

What is our future in regards to our health?

Dr. Conard and the other doctors feel that most of you are healthy and that you do not need to worry about the future. This is also true for the unexposed people who moved onto the island after the fallout and for the children who were born after 1954. Some of you may become sick but you will be given the best medical care possible . . . even if the sickness is not caused by the radiation. If you allow the doctors to examine you and if you follow their advice, you should be able to live a normal, healthy life. Examinations of thousands of children of exposed parents in Japan have not shown any diseases from radiation. Examinations of your children also show that they are healthy.

Appendix 5

Reported Cases of Notifiable Diseases, Trust Territory Districts

Disease	Jan. '74	Jan. '75					
	Total	Total	Marshalls	Ponape	Truk	Marianas	Yap	Palau
Category "A"								
AA Cholera								
AB Plague								
AC Smallpox								
AD Yellow fever								
Category "B"								
BA Dengue fever								
BB Diphtheria								
BC Dysentery - amoebic	180	203	66	58	70	4	5	-
BD Dysentery - bacillary								
BE Encephalitis								
BF Measles								
BG Meningitis - meningococcal								
BH Meningitis - other forms	2	1	-	-	-	-	-	1
BI Pertussis	23	1	-	-	1	-	-	-
BJ Poliomyelitis								
BK Relapsing fever (louse-borne)								
BL Severe epidemic fever								
BM Typhoid fever								
BN Typhus (flea-borne)								
BO Typhus (mite-borne)								
BP Typhus (louse-borne)								
Category "C"								
CA Gonorrhea	78	48	11	16	2	7	6	6
CB Infectious hepatitis	18	4	-	-	-	2	-	2
CC Influenza	1075	1762	817	69	295	139	176	266
CD Leprosy								
CE Rheumatic fever		1	-	-	1	-	-	-
CF Syphilis								
CG Tetanus								
CH Tuberculosis - pulmonary	14	8	2	2	-	3	1	-
CI Tuberculosis - other forms								
CJ Yaws (treponematosis)		2	-	-	-	-	-	2
Category "D"								
DA Chickenpox	24	94	33	15	18	28	-	-
DB Conjunctivitis - acute infectious of newborn								
Conjunctivitis - infectious (pink eye)		150	-	-	-	150	-	-
DC Dysentery - unspecified type								
DD Filariasis	6	9	-	-	9	-	-	-
DE Fish poisoning	17	27	27	-	-	-	-	-
DF German measles								
DG Mumps	447	14	-	-	10	-	1	3
DH Septic sore throat	32	59	-	5	-	54	-	-

Appendix 6

Mean Blood Counts at Various Times After Exposure

Postexposure Day	WBC* ($\times 10^{-3}$) <5	WBC* ($\times 10^{-3}$) >5	Neutrophils ($\times 10^{-3}$) <5	Neutrophils ($\times 10^{-3}$) >5	Lymphocytes ($\times 10^{-3}$) <5	Lymphocytes ($\times 10^{-3}$) >5	Platelets** ($\times 10^{-4}$) Male <10	Platelets** ($\times 10^{-4}$) Male >10	Platelets** ($\times 10^{-4}$) Female All Ages	Platelets** ($\times 10^{-4}$) Total Group	Hematocrit, % Male <15	Hematocrit, % Male >15	Hematocrit, % Female All Ages	RBC ($\times 10^{-6}$) Male <15	RBC ($\times 10^{-6}$) Male >15	RBC ($\times 10^{-6}$) Female All Ages
(a) Exposed Rongelap (175 rads; 64 people plus 3 in utero)																
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	---	3.7	----	----	----	----	----	----	----	----	----	----
7-yr survey	----	7.4	---	3.9	---	2.9	----	24.6	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	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	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	----	----	----
13-yr survey	----	7.2	---	3.7	---	2.8	----	22.9	25.1	----	41.4	44.5	40.0	3.84	4.52	4.00
14-yr survey	----	7.0	---	3.8	---	2.6	----	----	----	----	41.5	45.7	39.1	----	----	----
15-yr survey	----	6.5	---	3.0	---	3.0	----	15.9	19.1	----	----	46.2	39.9	----	4.26	3.66
16-yr survey	----	6.4	---	3.5	---	2.3	----	----	----	----	----	43.2	38.1	----	----	----
17-yr survey	----	7.3	---	3.9	---	3.1	----	20.3	24.6	----	----	42.9	38.7	----	4.31	3.84
18-yr survey	----	6.9	---	3.9	---	2.4	----	23.2	29.1	----	----	44.3	37.0	----	4.57	4.01
19-yr survey Mar	----	7.4	---	3.9	---	2.6	----	22.0	26.0	----	----	44.7	36.9	----	4.72	4.15
19-yr survey Sep	----	7.6	---	4.2	---	2.5	----	24.8	28.7	----	----	41.1	36.0	----	4.89	4.13
20-yr survey Mar	----	7.6	---	4.3	---	2.4	----	23.5	28.0	----	----	42.0	36.6	----	5.20	4.91
20-yr survey Sep	----	8.1	---	4.3	---	2.8	----	18.4	20.2	----	----	48.7	41.9	----	5.60	4.66

*Microscopic counts first 2 yr. Coulter electronic counts yr 3 through 18. General Science (MK3) electronic counts yr 19 and 20.

**Phase microscopy through 14 yr. Coulter electronic counts 15 through 18 yr. General Science (MK4) electronic counts yr 19 and 20.

Appendix 6 (continued)

Postexposure Day	WBC* ($x10^{-3}$) <5	WBC* ($x10^{-3}$) >5	Neutrophils ($x10^{-3}$) <5	Neutrophils ($x10^{-3}$) >5	Lymphocytes ($x10^{-3}$) <5	Lymphocytes ($x10^{-3}$) >5	Platelets** ($x10^{-4}$) Male <10	Platelets** ($x10^{-4}$) Male >10	Platelets** ($x10^{-4}$) Female All Ages	Platelets** ($x10^{-4}$) Total Group	Hematocrit, % Male <15	Hematocrit, % Male >15	Hematocrit, % Female All Ages	RBC ($x10^{-6}$) Male <15	RBC ($x10^{-6}$) Male >15	RBC ($x10^{-6}$) Female All Ages
(b) Ailingnae Group (69 rads; 18 people plus one in utero)																
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	----	9.2	---	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	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	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	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	----	----	----
13-yr survey	----	6.4	---	3.0	---	2.9	----	20.7	22.8	----	36.0	43.6	36.5	3.84	4.98	4.14
14-yr survey	----	5.6	---	3.8	---	2.2	----	----	----	----	38.0	46.5	39.3	----	----	----
15-yr survey	----	5.8	---	3.2	---	2.2	----	17.2	21.4	----	----	45.0	35.6	----	4.67	3.59
16-yr survey	----	6.4	---	4.0	---	2.0	----	----	----	----	----	40.6	38.0	----	----	----
17-yr survey	----	6.7	---	3.4	---	2.6	----	17.2	28.4	----	----	43.4	35.5	----	4.33	3.81
18-yr survey	----	7.5	---	4.6	---	2.2	----	19.5	33.0	----	----	42.5	35.5	----	5.01	4.29
19-yr survey Mar	----	7.1	---	3.7	---	2.3	----	24.0	25.1	----	----	38.7	38.6	----	4.54	4.11
19-yr survey Sep	----	7.1	---	3.9	---	2.2	----	24.1	26.2	----	----	42.1	38.9	----	4.92	4.55
20-yr survey Mar	----	7.8	---	4.6	---	2.0	----	24.9	30.1	----	----	44.4	35.2	----	6.30	4.34
20-yr survey Sep	----	6.6	---	3.4	---	2.4	----	17.2	19.8	----	----	50.6	40.4	----	5.64	4.50

Appendix 6 (continued)

Postexposure Day	WBC* ($\times 10^{-3}$) <5	WBC* ($\times 10^{-3}$) >5	Neutrophils ($\times 10^{-3}$) <5	Neutrophils ($\times 10^{-3}$) >5	Lymphocytes ($\times 10^{-3}$) <5	Lymphocytes ($\times 10^{-3}$) >5	Platelets** ($\times 10^{-4}$) Male <10	Male >10	Female All Ages	Total Group	Hematocrit, % Male <15	Male >15	Female All Ages	RBC ($\times 10^{-6}$) Male <15	Male >15	Female All Ages
(c) Utirik Group (14 rads; 158 people)																
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	----	----	----
15-yr survey	----	7.5	---	3.8	---	3.2	----	----	----	----	----	45.5	39.5	----	----	----
18-19-yr survey	----	8.1	---	4.6	---	2.5	----	24.6	33.6	----	----	43.4	38.0	----	4.64	4.15
(d) Unexposed Rongelap Controls																
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	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	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	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
Rong. cont 13-yr	----	7.3	---	3.9	---	2.7	----	25.8	26.0	----	39.8	44.3	39.0	4.26	4.43	4.04
Rong. cont 15-yr	----	6.6	---	3.1	---	2.9	----	17.1	20.7	----	----	46.7	39.9	----	4.36	3.76
Rong. cont 17, 18-yr	----	7.5	---	3.9	---	3.1	----	21.6	26.6	----	----	45.1	38.2	----	4.47	3.96
Rong. cont 19-yr	----	7.9	---	4.2	---	2.7	----	22.6	27.6	----	----	42.8	37.2	----	4.63	4.22
Rong. cont 20 Sep & Mar	----	7.3	---	4.1	---	2.4	----	24.1	26.1	----	----	42.3	36.6	----	5.14	4.74

Appendix 7

Pediatric Anthropometric Data (Height and Weight), 1970-1974

Subject No. & sex	Age at exposure	1970 Ht cm	1970 Wt lb	1971 Ht cm	1971 Wt lb	1972 Ht cm	1972 Wt lb	1973 Ht cm	1973 Wt lb	1974 Ht cm	1974 Wt lb
Rongelap exposed											
83 M	*In utero*			164.5	126			164.5	128		
84 M	" "			161.2							
85 M	" "			158.8	99	162.0	110				
86 F	" "						96	150.4	94	150.4	
54 M	1 y			167.6		167.9	170				
65 F	1 y 2 m			146.7	86			147.0		147.0	
5 M	1 4	146.0		146.4	98	151.0	109	151.0	110		
3 M	1 5	154.0		157.9	151	159.0	136	159.5	122	159.5	
2 M	1 4	167.6	124			167.6	134		128	167.6	
6 M*	1 4			159.3		160.8				160.8	
33 F	1 7									160.5	
8 F*	1 8							154.0			
42 F	3			149.0	100					149.4	
21 F	3			150.8							
17 F	3 4				132			159.0			
23 M	3 5			171.0	156						
32 M	3 6			163.0				165.5	138		
69 F	3 7					158.5	150				
Rongelap unexposed	Date of birth										
805 F	2/25/54			158.5				158.5	142		
811 F	2/14/54			147.6				148.0	109		
812 F	2/ /54			157.3	114				121		
813 M	1/ 2/54			164.0							
814 M	4/ 5/52					161.0		161.0			
815 M	5/ 4/50			166.7							
817 M	10/19/50					172.0					
818 M	3/ 4/51							177.0			
879 F	4/ /54			150.5				150.5			
909 F	3/11/50			144.4							
911 F	3/ 8/53			146.5				148.0			
912 M	6/ 1/53							163.0			
925 F	5/ 4/50							149.4			
926 F	2/26/51			158.7							
955 F	5/11/52							153.0			
960 F	12/ 5/51										
978 F	10/20/50					154.0					
980 F	10/ 3/52					153.0					
981 M	8/ 8/54			161.0							
996 F	1/16/53			147.7							

*Ailingnae.

Appendix 8

Serum Iodine Levels in Marshallese, 1958-1974

Key: Plain numbers are T_4 values; arrow indicates change in T_4 with TSH;
TSH = thyroid stimulating hormone; numbers in parentheses are PBI values;
S indicates surgery; TR indicates beginning of hormone treatment.

Subject No. & sex	Age at exposure	Diagnosis	1958-61	1962	1963	1964	TR 1965	1966	1967	1968	1969	1970	1971	1972	1973	1974
Rongelap Exposed With Thyroid Lesions																
83 M	In utero (2nd tri)	Benign adenomas	(8.1)	(7.4)			(6.6)						7.5		6.7	6.2→8.6 TSH 7.0 S
3 M	1	Myxedema	(8.8)	(7.2)	(1.9)		(1.4) 1.3	(3.2) 1.6	5.9	2.0	3.3	(0.6) 3.1	5.9,4.1 12.5 TSH 81	6.7 TSH<2.5	8.9	2.7 TSH 12.5
5 M	1	Myxedema		(4.6)	(2.5)		(2.5) (1.9)	(3.1) (2.1) 2.9	1.8	1.6	1.6	(0.8) 3.3	1.3 1.6 TSH 163	2.5 TSH 79.0	0 TSH 376	3.6 TSH 1.7
33 F	1	Benign adenomas			(7.1)		(7.0)	(6.2) (5.6) S 7.3,5.01		10.3	9.3		22.3	3.7 TSH 6.8	7.4	10.1 TSH 32.0
54 M	1	Benign adenomas			(7.8)		(8.3)	(5.0) 7.0	7.8	7.5 S		(6.1)	9.6	6.5 TSH 8.2 Died Nov 72		
65 F	1	Benign adenomas	(7.1)	(6.7)	(8.8)		(7.0) 3.1→7.6	(3.1) (5.1) S 6.6	3.7		(8.7) 5.0	(7.9) 9.0	16.9	1.8 TSH 215	1.0 TSH 243	1.7 TSH 118
2 M	2	Benign adenomas	(8.0)		(10.2)		(7.9) (6.2) S 6.8	(5.2) 4.2		7.8 4.2	(10.7) 5.7	10.5	12.0	14.9 TSH<2.5	15.3	
17 F	3	Benign adenomas			(7.1)	(6.8) S	(2.3)	(1.8) <0.8	2.3	4.2	9.6	7.8	6.3	23.2 TSH 2.0	15.1	13.8 TSH<1
19 M	3	Benign adenomas	(6.4) (4.1)		(5.6)			(7.7) 3.3	5.4	S	10.1	(4.2) 6.1	9.1	13.4 TSH 3.4	3.6 TSH 32	6.9 TSH<1
21 F	3	Benign adenomas	(7.4)	(7.7)		(8.1) S	(0.7)	(1.3) <0.8	2.6	1.3	8.5	(8.2) 15.9	16.9	12.6→14.3 TSH 1.3	19.9 11.9	12.8 TSH 1.1
42 F	3	Benign adenomas		(8.8)	(9.0)			(5.0) (6.5) S 7.8,7.3		9.8	(14.1) 11.9		11.4	15.8 TSH<2.5	10.1	17.4 TSH<1
23 M	4	Benign adenomas	(9.6)	(10.3)					3.7	7.3 S	2.3	6.5	3.9 1.6 TSH 36.0	2.8 TSH 81.0	5.1 TSH 62	7.8 TSH 16
69 F	4	Benign adenomas	(12.2)	(7.8)	(8.7)	(10.2) S	(7.1)	(5.7) (2.8) 2.8				10.3 (7.3)	20.3	9.8 TSH<2.5	6.6	11.1 TSH 10
72 F	6	Carcinoma	(8.2)				(5.3)		4.9	6.0	S	(0.6) 3.3	1.3,0.3 0.2 TSH~200		0 TSH 49	<0.4 TSH 116
15 F	7	Benign adenomas	(12.8)				(6.4)		6.3	10.7	(9.5) 6.3 S	(7.9) 7.7	8.6	7.7 TSH 9.2	11.3	9.8 TSH<1
20 M	7	Benign adenomas	(7.0)		(5.5)		(7.2) (6.1) S 6.8				8.0	(2.8)	14.0	7.0 TSH 2.7	13.9	8.9 TSH<1
36 M	7	Benign adenomas	(10.6)		(2.7)		(4.1)	(4.2) (4.2) 7.0	4.2	4.2	4.2 S	4.6 (3.8)	4.9,4.7 5.0 TSH 24	10.0 TSH<2.5	2.5 TSH 59	1.3 TSH 15
61 F	8	Benign adenomas	(11.4)		(11.5)		(6.4)	(8.3) (7.5) S 7.3,8.0			(11.6) 6.2	(3.9) 6.3	16.0	12.4	4.1	4.6 TSH 2.8
75 F	12	Benign adenomas					(4.7)	(4.5)			(8.1)	(10.0)	18.9	S	22.0 23.3	23.0 TSH 5.6
67 F	14	Nodule 0.5 cm, lt. lobe					(6.6)				(6.8)	(9.1)	11.9	6.5 TSH 1.35	9.6	7.4 TSH 2.7
18 F	21	Carcinoma					(11.7)				6.3 10.2 S	15.5 (10.7)	9.9	0.4 TSH 110	17.8	10.9 TSH<1
40 M	29	Benign adenomas	(7.0)				(10.3)			9.0	13.0	8.0	12.7	7.2 TSH<2.5	7.0 9.6 S	7.8 TSH 2.6
64 F	30	Carcinoma		(10.7)	(12.0)		(10.0) (7.5) S 5.5	3.3	3.3	2.9	(10.3) 6.8	(7.0) 12.4	8.6	7.6 TSH>60; 172	10.6	9.0→7.5 TSH 1.2
56 F	66	Benign adenomas		Died 62												

Appendix 8 (continued)

Subject No. & sex	Age at exposure	Diagnosis	1958-61	1962	1963	1964	TR 1965	1966	1967	1968	1969	1970	1971	1972	1973	1974
Rongelap Exposed Without Thyroid Lesions																
85 M	In utero (1st tri)		–										7.5	6.1 TSH<2.5	5.6	7.8→11.4 TSH 2.3
86 F	In utero (1st tri)		(12.0)	(9.2)	(8.2)			(7.1) 9.8				(5.8) 8.4	10.6	12.5 TSH 3.3	9.1	
32 M	3		(10.8)		(8.7)			(4.6) 6.5	4.9	6.5	(6.2)		14.2		6.5	7.5 TSH<1
47 M	8		(8.4)	(7.9)				(7.2)				10.7	17.4	7.4 TSH<2.5	13.0	8.4 TSH 7.0
76 M	11		(7.4)				(4.4)	(5.6)			5.0	(4.7) 5.7	9.9	8.2 TSH 2.9, " 7.0	4.5 TSH 3.3	5.4→7.0 TSH 4.7
26 M	12			Died 62												
24 F	13									20.2		(9.3)	16.6	8.2 TSH 6.5 " 2.2	18.2 15.3	8.2→12.1 TSH 4.4
35 M	13		(6.0)				(4.6)				(3.0)	(5.5) 8.0	8.5	4.7 TSH 3.6	6.8	5.9→7.8 TSH 3.1
49 F	15										(6.6) 9.6	9.4	9.6	7.5 TSH 3.1	4.8 TSH 2.1	
74 F	16		(10.4)									(4.9)	9.9	10.4 TSH 1.95; 7.2;16.3	8.2	
22 F	17		(13.6)	(9.3)						14.5	(6.7)	(4.9)	11.7	10.2 1.52→6.0	24.4 18.5	3.5 TSH 2.7
39 F	17		(10.0) (9.7)				(11.9)				(6.	(10.4)	14.7	8.6 TSH 6.0	25.6 20.4	8.3→11.5 TSH 5.1
12 F	18		(6.6)		(8.8)					18.4	(10.7)	13.4	9.0	7.4 TSH 4.8		6.5→8.7 TSH 4.4
73 M	18						(8.4)				(11.6)	(11.9) 16.3	9.9	9.7 TSH<1.0	6.5	7.8→12.0 TSH 2.3
37 M	20		(11.6)				(5.3)					(4.5)	9.2	5.2 TSH<2.5	15.3	4.6→6.6 TSH 2.6
9 M	22											(6.8)	7.2	5.7 TSH 1.9	11.4 10.2	4.5→5.3 TSH 4.5
10 M	24		(11.8) (7.0)	(9.5)	(12.0)		(8.3)					(8.3)	9.3	16.6 TSH<2.5	8.9	9.2→12.8 TSH<1
14 M	25			(10.8)	(8.3)		(7.1)					12.2	6.5	6.2 TSH 2.6		6.7 TSH 3.0
27 M	26		(11.0)				(7.1) 7.9	6.7				(8.3)	11.9	18.2 TSH 1.4	15.6	7.5 TSH 2.7
77 M	26											(7.3)	10.6		13.6	5.3 TSH 2.8
71 F	28				(8.7)						6.2	(4.7)		5.2 TSH 11.6	3.3 TSH 7.0	4.2 TSH 10.3
66 F	30						(8.0)					(11.6)	14.0	21.8 TSH 3.5	10.7	7.0→9.4 TSH 3.1
7 M	36							4.9				(12.1)	10.1	8.9 TSH 2.3	7.1	4.7→5.3 TSH 2.1
63 F	36											(9.6)	10.9	10.7 TSH<2.5	18.0	8.2 TSH 6.3
78 F	37											(5.1)	10.6	12.0 TSH 4.5	15.4	5.2→6.7

Appendix 8 (continued)

Subject No. & sex	Age at exposure	Diagnosis	1958-61	1962	1963	1964	TR 1965	1966	1967	1968	1969	1970	1971	1972	1973	1974
Rongelap Exposed Without Thyroid Lesions (continued)																
4 M	38		(8.6)									(5.7)	11.1	7.3 TSH 6.0	12.0	6.9→7.8 TSH 7.0
79 M	39											(8.4)	13.8	TSH 1.7		
25 M	42		Died 56													
68 M	44						7.5	9.4	7.0			(9.3)	16.9	14.2 TSH 1.2	10.5	8.9→10.4 TSH 6.0 Died 74
34 F	45						7.5	10.6	5.9			(10.4)	21.3	15.9 TSH 1.9	13.1	7.1→10.6 TSH 6.3
80 M	46											(8.2)	8.5	12.7 TSH 1.6	9.2	8.1→9.1 TSH 3.0
82 M	50		(8.6)									(6.8)	12.7	11.2 TSH 1.4	9.4	5.8 TSH 3.8
11 M	50		(10.6)	(11.0)			(8.4)					15.7	6.1	10.8 TSH 1.7	12.1	6.8 TSH 3.9
52 F	56				Died 63											
60 F	56				(10.8)							(5.6)	9.0	TSH 6.4 Died 72		
62 F	57		Died 59													
13 F	58						5.5	6.3								
58 F	59		(8.6)				(9.2)					(9.9)	16.0	15.3 TSH<2.5	8.8	8.2 TSH 5.3
30 F	59			Died 62												
38 M	69		Died 57													
55 M	75						6.2	5.4 Died 66								
46 M	76			Died 62												
57 F	100				Died 63											
Ailinginae Exposed With Thyroid Lesions																
8 F	1	Benign adenoma (?)			(9.0)		(5.7)	(5.4) 6.5		8.8	6.7	(5.7) 10.7		7.8→15.4 TSH 2.0 S	12.2 11.9	14.0 TSH<1
53 F	8	Mass 0.5 cm, lt. lobe 73, neg. 74	(8.2)				(8.5)			5.2		(8.3) 12.4	14.0 5.4		7.4	9.4 TSH<1
51 F	25	Benign adenomas					(9.7)						6.5	7.1 TSH 2.6	6.3	3.4 6.9→9.1 S TSH 3.9
45 F	32	Benign adenomas				(9.1)	(8.4)					(4.9)	9.0		3.7 5.5 S TSH 4.9	4.6 TSH 1.7
41 M	44	Nodule lt. lobe					(6.7)					(6.2)	5.5	5.6 TSH 2.7	4.5 TSH<5.0	6.0→9.4 TSH 4.4
59 F	44	Benign adenomas		(9.8)	(11.0)		(8.6) (11.1) 8.6	(6.4) 7.3 S 8.1	9.6	11.6 Died 68						

Appendix 8 (continued)

Subject No. & sex	Age at exposure	Diagnosis	1958-61	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971	TR 1972	1973	1974
Ailingnae Exposed Without Thyroid Lesions																
84 M	In utero (2nd tri)					(9.3)		(6.2) 5.9				(4.0) 7.7	9.6			6.7
6 M	1		(10.7)	(9.7)	(7.9) (8.7)		13.8 7.2→7.9	(5.0) 5.9		3.6	6.2	7.5	6.2	8.4→14.4 TSH 2.6	7.2	6.8→8.3 TSH 3.9
44 M	4		(10.2) (6.7)	(7.9)				(4.7) 7.8								5.8 TSH 4.2
48 F	6	Hypertrophy rt. 74	(8.8)					7.8		9.6	(7.3)	(7.8) 12.4	9.6	11.9 TSH 3.0	7.6	7.2→12.2 TSH 3.2
81 F	8		(7.0)							4.4		(5.7) 6.1	7.5	2.9 5.4→12.4 TSH 2.0;5.9	3.9 TSH 7.5	5.8→7.8 TSH 1.5
70 F	15	Neurofibroma neck			(8.7)		6.7			7.0 S		(6.8)	8.0	8.3 TSH 2.8	4.7 TSH<5.0	7.9→11.8 TSH 1.5
31 M	31		Died 58													
50 M	34		(9.2)				(10.1)						10.7 Died 71			
16 M	39											(5.0)	7.7	5.7 TSH 5.0	3.7 TSH 6.0	6.4→8.3 TSH 5.3
1 F	54			(12.0)	(9.4)							(6.6)	8.3	8.1 TSH 2.3	5.9	5.1 TSH 1.4
29 M	65							Died 66								
43 F	67						Died 65									
28 F	68						Died 65									

					TR			TR
Utirik Exposed With Thyroid Lesions								
2229 F	20	Carcinoma (papillary)	S			2.5 TSH 10.8 " 13.7	5.9	5.5 TSH 1.4
2208 F	35	Benign adenoma					3.9 6.2 S TSH<5.0	
2212 F	35	Benign adenoma				9.3 TSH 3.1	3.7 6.3 S	4.4 TSH 9.8
2194 F	37	Lobular gland (normal?)					8.4	
2258 M	47	Nodule		Died 70				
2182 F	52	Nodule					4.3 5.6	6.9
2221 F	54	Sm. nodule rt. lobe					4.4;5.2 TSH 5.0 S	

Appendix 8 (continued)

Subject No. & sex	Age at exposure	Diagnosis	1958-61	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973	1974	TR
							Rongelap Unexposed With Thyroid Lesions										
938 F		Benign adenoma				(5.6)	(3.9)		5.2			S			6.0		
829 F		Benign adenoma		(8.6)	(7.1)		5.7								3.0;5.2 TSH 5.4 S	6.1 TSH 1.9	
841 F		Benign adenoma													9.6	12.2 S	
845 M		Possible nodule													9.2		
910 M		Nodule 0.5 cm, rt.													6.8		
912 M		Firm area, nodule													6.5		
1007 M		Sm. nodule, lt. lobe											5.0		6.5		
858 F		Large goiter, soft, movable													Died		
898 F		Nodule 0.5 cm, lt.													8.9 Died 73		

Appendix 9

A. Urinary Iodine Excretion

24-hr output	I, µg/ml	I, µg/24 hr	24-hr output	I, µg/ml	I, µg/24 hr
700	0.105	73	1380	0.090	124
700	0.133	93	1160	0.022	25
840	0.162	136	530	0.129	68
740	0.145	107	840	0.112	94
840	0.104	87	800	0.333	266
1190	0.121	144	580	0.275	160
1230	0.214	263	540	0.136	73
1050	0.207	217	680	0.097	66
780	0.305	238	620	0.069	43
					$\bar{X}$=126.7±74.5

B. Dietary Iodine (Recommended intake: 50 to 75 µg/day)

Sample meal No.	Total wt., g	Contents	Total I, µg/meal	Estimated I intake, µg/day
1	443	Breadfruit Clam Rice Cocoanut	60.9	152
2	430	Breadfruit Octopus	35.4	88
3	300	Clam Pandanus Cocoanut	41.8	104
4	255	Pandanus Octopus Arrowroot	20.7	52
5	294	Pandanus Octopus Arrowroot	23.5	59
6	236	Pandanus Octopus	19.3	48
7	610	Pandanus Arrowroot	36.1	90
			$\bar{X}$=34.0±14.7	84.7±36.6

C. Data on Principle Isotopes of Iodine Contributing to Thyroid Dose

(From "Inhalation of Radioiodine from Fallout: Hazards and Countermeasures" by R. Cole[137])

E_{max}, MeV	Relative abundance, %	Range in tissue, μm	50%-Dose radius,* μm
	Iodine-131 Betas		
0.246	2.2		
0.332	10.6	1,000	150
0.606	86.4	2,100	310
0.807	0.8		
	Iodine-132 Betas		
0.681	15	2,500	375
0.934	20	3,900	625
1.150	22	5,400	920
1.583	22	8,400	1,500
2.111	21	11,900	2,400
	Iodine-133 Betas		
0.49	7		
1.34	93	6,800	1,200
	Iodine-134 Betas		
0.87	5		
1.55	65	8,100	1,460
2.65	25	15,800	3,300
3.51	5		
	Iodine-135 Betas		
0.40	35	1,300	195
1.00	40	4,300	690
1.43	25	7,300	1,300

*The 50%-dose radius is defined, for a point source in tissue, as the distance within which 50% of the dose, for that energy, is distributed.
**Since this isotope with 53-min half-life is important only for about 6 hr, it was not considered in the Marshallese thyroid dose calculations.

Appendix 10

Hospital Records of Thyroid Cases, 1972-1974

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
— Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 75
(NAME)

08-42-33 R
(UNIT NO.)

ADMITTED: 7 November 1972 DISCHARGED: 13 November 1972

This 29-year-old Marshallese lady was admitted to this Hospital for evaluation of thyroid nodularity and physical status for thyroid surgery.

HISTORY OF PRESENT ILLNESS: She was brought to this Hospital from the Marshall Islands with another Rongelap girl who also had thyroid nodularity. Both had been exposed to radioactive fallout in 1954. They were accompanied by a Marshallese interpreter. Thorough thyroid studies and evaluation of her general physical status prior to surgery were carried out.

Before the detection of her thyroid abnormality in September 1972, the thyroid examinations have always been negative and she had always appeared euthyroid with normal thyroxin levels. The patient was exposed to a whole body dose of 175 rads of gamma radiation and the thyroid gland received an estimated dose of 500 rads, largely from absorption of radioiodines in the fallout.

Following her exposure to fallout, the patient experienced slight nausea and some itching and burning of the skin. She developed supericial beta burns over parts of her body which healed within a few months. She also had transitory platelet depression and mild leukocyte depression which returned to normal levels within a year. Until the development of thyroid lesions, her past history had been generally negative except for the occurrence of virus pneumonia as a child. Her growth and development had been normal and she has had six normal deliveries and one miscarriage. Her FAMILY and SOCIAL HISTORY were noncontributory.

PHYSICAL EXAMINATION: The patient is a well-developed and well-nourished, alert and cooperative individual. Except for thyroid findings, the physical examination was essentially negative. The thyroid examination revealed a 2-cm mass in the upper right lobe which was non-tender and only moderately hard. The lower part of the right lobe was enlarged but the left lobe was barely palpable. No nodes were palpated except for a small one in the posterior cervical chain.

LABORATORY & X-RAY DATA: Thyroid scan with ^{99m}Tc showed both functioning and non-functioning nodules in the right lobe of the gland. T4 level was normal (8.3 μg%) and showed good increase after TSH stimulation. Before TSH administration (10 units) the ^{123}I uptake was 39.9% and after the administration of the hormone the uptake was 55.5%. Thyroxin levels will be recorded later. BMR and cholesterol were normal. The remainder of the Laboratory and X-Ray Workup was negative, except for the presence of trichuris trichuria and ascaris lumbricoides in the stools.

-1-

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 75
(NAME)

08-42-33 R
(UNIT NO.)

HOSPITAL COURSE: Her hospital course here was uneventful and she was found to be in good condition for surgery. Prior to discharge the patient was given 25 μCi ^{131}I to ensure complete removal of thyroid tissue in case complete thyroidectomy was indicated in the eventuality of malignancy. She was discharged 13 November and transferred to Cleveland Metropolitan General Hospital.

At the above hospital on 14 November 1972 thyroid surgery was performed by Dr. Brown Dobyns. Grossly both lobes of the gland were found to be multinodular. The right lobe exhibited a 1.5 - 2 cm. nodule in the upper part of the lobe and a smaller nodule in the lower part. The left lobe was filled with smaller nodules. Both lobes were removed and a small nodule was excised from the isthmus and the remainder of the isthmus was left intact. Microscopic examination revealed numerous benign, adenomatous nodules with prominent microfollicular areas in both lobes. Recovery from surgery was uneventful and the wound was healing nicely when she was discharged for return to the Marshall Islands on 19 November 1972. Details of her hospitalization in Cleveland, including surgical and pathological reports, are attached.

FINAL DIAGNOSIS: Benign adenomatous nodules of the thyroid gland.

DISCHARGE MEDICATION: The patient was advised to continue taking her thyroxin medication (Synthroid) rigorously. A copy of the Hospital Summary and advice about post-surgical treatment as well as treatment of her intestinal parasites is being forwarded to the practitioner at the Majuro Hospital in the Marshall Islands. The patient will also be carefully followed up on subsequent medical surveys.

Robert A. Conard, M.D.

cfh
Received: 12/21/72
Typed: 12/21/72

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 8
(NAME)

08-42-34 R
(UNIT NO.)

ADMITTED: 7 November 1972 DISCHARGED: 13 November 1972

This 19-year-old Marshallese girl was admitted to this Hospital for evaluation of thyroid,nodularity and physical condition prior to thyroid surgery.

MEDICAL HISTORY: Examination in September revealed nodularity in the right upper lobe of the thyroid gland. One nodule was fairly firm and about 1 cm. in diameter and the other was smaller and soft in consistency. No lymph nodes were palpated, and the patient appeared to be euthyroid. Previous exams over the past 18 years have not revealed any thyroid abnormalities and she has always appeared euthyroid with thyroxin levels in the normal range. During the September 1972 examination she was noted to be anemic, cause unknown.

She was 17 months of age at the time of exposure to fallout and received an estimated whole-body dose of 69 rads of gamma radiation and a thyroid dose of about 500 rads, largely from absorption of radioiodines in the food and water consumed. This is the first case of thyroid nodularity in children of this lower Rongelap exposure group (Ailingnae group).

Immediate effects of fallout were few: development of mild superficial beta burns of the skin and slight epilation which healed within a few weeks, with normal regrowth of hair; development of mild platelet depression and leukopenia which returned to normal levels by one year. Since 1954 she has been in good health with no significant illnesses or injuries and her growth and development have been normal. Another sister, who was also exposed to fallout, developed benign thyroid adenomatous nodules which were removed surgically several years ago.

PHYSICAL EXAMINATION: This slender girl is well-developed and well-nourished. The thyroid examination revealed the upper part of the right lobe to be irregular with a soft, 1.5-cm. nodule in the upper part. The left lobe was barely palpable. No cervical nodes were palpated and she appeared to be euthyroid. The remainder of the Physical Examination was essentially negative except for a small papilloma in the perineum.

LABORATORY & X-RAY DATA: Thyroid scans using ^{99m}Tc showed nodularity with reduced function in the upper right lobe. T4 level was normal (9.3 µg%) and showed good increase after TSH stimulation. The ^{123}I uptake was slightly elevated and the response to TSH was fair. The Laboratory and X-ray data were otherwise normal except for the presence of Trichuris Trichuria in the stools.

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 8
(NAME)

08-42-34 R
(UNIT NO.)

HOSPITAL COURSE: Her Hospital course was uneventful and it was gratifying that in view of the anemia noted in September, her blood picture was now within normal limits and no contraindications to surgery were noted. The patient was given 25 μCi ^{131}I prior to discharge to ensure complete removal of thyroid tissue at surgery in the eventuality of malignancy. She was discharged on 13 November 1972 and transferred to Cleveland Metropolitan General Hospital.

At Cleveland Metropolitan General on 14 November 1972, surgery was performed by Dr. Brown Dobyns. At surgery the right lobe of the thyroid was quite nodular with one firm black nodule about 2 cm in diameter in the superior pole, and smaller ones in the remainder of the lobe. The left lobe appeared relatively normal. Complete right lobectomy was performed and a thorough search revealed no lymph nodes in the vicinity of the thyroid gland on either side. Microscopic examination of the excised tissues revealed small area of papillary carcinoma*. The remainder of the lobe exhibited multiple benign adenomatous lesions. Recovery from surgery was uneventful with no complications. By 19 November 1972, the wound was healing nicely and the patient was discharged to return to the Marshall Islands with the interpreter and the other thyroid patient. A summary of her Hospitalization, including surgical and pathological reports, at the Cleveland Metropolitan General Hospital is attached.

FINAL DIAGNOSIS: ?Papillary carcinoma of the thyroid.

DISCHARGE MEDICATION: The patient was placed on thyroxin medication (Synthroid) 2.1 mg weekly.

Follow-up advice and treatment instructions are being forwarded to the practitioner in charge of her case at the Majuro Hospital in the Marshall Islands. Careful follow-up exams will be carried out on this girl at the time of our annual medical exams and at six month intervals. She will also have periodic exams at a hospital for thorough thyroid studies to rule out extension of her disease.

*Further pathological review of the sections is in progress.

Robert A. Conard, M.D.

Received: 12/20/72
Typed: 12/21/72
cfh

CC- DR CONARD ON 1/4/73

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 40
(NAME)

08-45-42R
(UNIT NO.)

ADMITTED: June 2, 1973 DISCHARGED: June 10, 1973

This 48-year old Marshallese man who had been exposed to radioactive fallout in 1954 was admitted for evaluation of thyroid nodularity and physical status for possible thyroid surgery.

HISTORY: A small nodule in the right lobe of the thyroid was first detected in 1965. He, along with other people of Rongelap who had been exposed to radiation had been placed on L-thyroxin treatment and the nodule disappeared on this treatment. However, during the recent examinations in March, he was again discovered to have a hardened area in the right lobe with indistinct boundaries. No lymphadenopathy was noted. He has always appeared euthyroid and his thyroxin levels have generally been in the normal range, with only one reading slightly slow.

He was exposed to 175 rad of gamma radiation from accidential fallout of 1954 and his thyroid gland probably received about 330 rads partly from radioactive iodines absorbed. He had early acute effects with hemological depression and mild "beta" burns of the skin which he recovered from within a year. Examinations over the past 19 years have revealed only a few findings: The history of yaws in childhood, measles, occasionally bronchitis, one possible attack of pneumonia, a fistula-in-ano which was surgically corrected. He has remained in very good health and is a hard worker and leader of his people. He was magistrate of the village at the time of the fallout. His wife was operated for cancer of the thyroid, three sons for benign thyroid lesions, and 1 son died at 19 years of age with acute leukemia (possible from radiation exposure).

PHYSICAL EXAMINATION: This well developed, muscular, alert man appeared quite healthy and euthyroid. The thyroid findings noted were the same as were reported above for the March examination. The only other findings were bilateral [illegible], missing teeth and gingivitis.

LABORATORY AND X-RAY DATA: Thyroid scans showed no distinct nodularity but slightly increased size of the right lobe. Radioactive iodine uptake was normal and response to TSH stimulation was fair. T-4 levels are not reported yet. Serum was nonreactive for antithyroid globulin antibodies. Chest X-Ray was normal. Hemogram was normal. Syphilis serology was reactive (titer of 4) but this low level was not considered significant in view of his history of having had the yaws. Tests of kidney function and liver function were normal. Electrolytes normal, EKG normal, proteins normal, electrolytes and lipids normal. Stools were positive for whipworm.

HOSPITAL COURSE: During the 8 days of his hospital stay, the patient was a symptomatic until the 5th day when following TSH injection (given the previous day) he developed an acute thyroiditis with low grade fever, nausea, anorexia and slight neutrophilia. Scan showed some enlargement of the gland.. The thyroiditis reduced rapidly and at the

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 40
(NAME)

08-45-42R
(UNIT NO.)

HOSPITAL COURSE CONTINUED:

time of discharge on June 10th, he was symptom free and his thyroid of normal size. The cause of this episode is unclear.

AT the Metropolitan General Hospital thyroid surgery was performed by Dr. Brown Dobyns. 25 microcuries of 131 iodine were given orally the day before for autoradiographic studies of removed thyroid tissues. In surgery a firm area was noted at the junction of the right lobe and isthmus which contained several small nodules and also several areas of nodularity were noted in isthmus. The lower part of the isthmus and part of the right lobe were removed. The pathologist reported the lesions benign with varying degrees of hyperthrophy, hyperplasia and fibrosis. The patient withstood the surgery well and his convalescence was uneventful. He was discharged on June 18th to return to the Marshall Islands. Copies of the Hospital Summary and Pathological Reports from Cleveland incorporated with the BNL records.

FINAL DIAGNOSIS: Thyroid lobular hyperplasia and hypertrophy with slight fibrosis.

DISCHARGE MEDICATION: Continued treatment with L-thyroxin (3 MG-day) is recommended since he is part of the more heavily exposed Rongelap group, all of whom have been placed on such treatment since 1965.

Robert A. Conard, M.D.

Received: 6-28-73
Typed: 6-29-73
er

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 45
(NAME)

8-45-41R
(UNIT NO.)

ADMITTED: June 2, 1973 DISCHARGED: June 10, 1973

This 51-year-old Marshallese woman was brought to this Hospital for study of possible thyroid nodularity and evaluation of physical status for thyroid surgery.

MEDICAL HISTORY: The examination in March revealed a suspicious area of firmness about 2.5 centimeters in diameter in the region of the lower right thyroid pole. There was uncertainty as to whether it was in the thyroid gland, but because of her radiation history it was thought surgical exploration was indicated. There was no lymphadenopathy and she appeared euthyroid.

She was exposed to fallout radiation in 1954, receiving about 69 rads whole-body gamma radiation and about 134 rads to her thyroid gland from absorption of radioiodines from the fallout. She had mild radiation effects with slight transient hematological depression and superficial beta burns to the skin. She recovered from these effects within the year and in subsequent years only minor medical findings were noted. Among these were pleural thickening of the right hemidiaphragm, chronic endocervicitis, gonorrhea, pingueculae (left) and partial prolapse of the vaginal wall. She has always appeared euthyroid and several thyroxin levels were in the normal range, though the March level was somewhat low.

PHYSICAL EXAMINATION: This lady was well-nourished and somewhat overweight. The thyroid examination revealed an area of firmness as described above, in the lower right thyroid region near the clavicle. Again, it was uncertain as to whether this was actually thyroid tissue being palpated. The patient appeared euthyroid and no lymphadenopathy was noted. The physical examination was otherwise generally negative except for slight cardiac enlargement and eye findings noted above.

LABORATORY AND X-RAY DATA: The thyroid scans showed no evidence of thyroid nodularity and the thyroid gland appeared normal. The RAI uptake was normal and the response to TSH stimulation was good. Her chest x-ray showed slight cardiac enlargement and some tenting of the diaphragm on the right from scarring, probably due to an old inflammatory disease. The hemogram was normal except for a high sedimentation rate (which is frequently seen in the Marshallese, particularly the women). The blood chemistry findings were generally normal.

HOSPITAL COURSE: During her 8-day stay here, she remained generally asymptomatic except for slight pain in the right shoulder at night relieved by aspirin, and nasal congestion on several occasions which was relieved by nose drops. She was discharged

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 45
(NAME)

8-45-41R
(UNIT NO.)

on June 10th, 1973 and transferred to the Cleveland Metropolitan General Hospital.

At the Cleveland Metropolitan General Hospital, on June 12th, surgical exploration of the thyroid and upper mediastinum was carried out. No mass was found in the right lower thyroid region and it was thought that a somewhat thickened lower edge of the sternocleidomastoid muscle may have been deceiving. However, a small cyst was discovered in the lower left pole of the gland which proved to be a degenerating cystic adenoma. Convalescence was quite uneventful. A summary of the hospitalization including surgical and pathological reports at Cleveland is included in her BNL record.

FINAL DIAGNOSIS: Adenoma with cystic degeneration.

DISCHARGE MEDICATION: On the basis of her thyroid status, thyroxin treatment is not indicated. However, a decision is pending as to whether to give thyroxin supplemental treatment in this group of islanders who received a smaller dose of radiation. She will have periodic follow-up examinations including determination of serum thyroxin levels at six-month intervals.

Robert A. Conard, M.D.

RA:bwa
Dict: 6/27/73
Typed:6/28/73

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Utirik 2208
(NAME)

08-45-40R
(UNIT NO.)

ADMITTED: June 2, 1973

DISCHARGED: June 10, 1973

This 54 year old Marshallese woman who had been exposed to a slight amount of fallout in 1954 was brought to the United States and admitted to this hospital for evaluation of thyroid nodularity and physical status for possible thyroid surgery.

HISTORY:

Examination, this past March revealed 2 rather hard masses in the right lobe of the thyroid. These had not been noted on previous examinations. No lymph nodes were palpated and she has always appeared euthyroid. Her T-4 level in March was 3.7 µgm%.

She was accidentally exposed in 1954 to about 14 rad of gamma radiation and an estimated dose of 22 rads to the thyroid from fallout while she was living on Utirik Island. She showed no affects to this slight exposure. Following her initial examination she was not again examined until 1969 at which time she was found to have blood pressure readings of 200-220/100-110 and a systolic murmer was noted. Also reported was an umbilical hernia and a possible fibroid of the uterus. Her only complaints have been headaches, chronic joint pains, particularly of the right arm.

PHYSICAL EXAMINATION:

This intelligent, alert, somewhat obese Marshallese lady appeared to be healthy and euthyroid. The thyroid findings were as described above for the March examination. Other findings included inversion of the nipple of the right breast; enlargement of the heart with a harsh systolic murmur, heard best over the aorta and PMI areas; blood pressure 180/86 with no evidence of decompensation; EKG was within normal limits; tenderness around the umbilicus though the hernia was not actually palpated; Pelvic examination revealed 3 small areas of hyperplasia of the cervix.

LABORATORY AND X-RAY DATA:

The thyroid scan showed a poorly functioning nodule in the lower half of the right lobe. The radioactive iodine uptake was normal and the response to TSH stimulation was good. T-4 report is not yet in. Serum was non-reactive for antithyroid globulin antibodies. X-Ray of the chest showed moderate cardiomegaly and arteriosclerosis. The lungs were clear. The hemogram was normal except for an ESR of 40 (high values are noted in the Marshallese). Serology was negative. Serum proteins negative, and electrolytes generally normal. Urine was negative and liver and kidney function tests normal.

HOSPITAL COURSE:

During her 8-day hospital stay, she remained generally asymptomatic except for some pain and stiffness in her right arm which was relieved with aspirin. Her appetite was good and she was completely ambulatory. Her blood pressure remained normal after the slight elevation noted on admission. She was discharged on June 10th for travel to Cleveland.

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Utirik 2208
(NAME)

08-45-40R
(UNIT NO.)

HOSPITAL COURSE CONTINUED: At the Cleveland Metropolitan General Hospital, surgery was performed by Dr. Brown Dobyns. One day, prior to surgery 25 μ Ci ^{131}I was given orally in order to do autoradiographic studies of removed thyroid tissues. Surgical exploration revealed a single mass in the mid-right lobe which was removed along with a part of the right lobe. The pathological diagnosis was thyroid adenoma with degeneration. The tumor was "cool" based on radiation counting. The patient withstood the surgery well and convalescence was uneventful. She was discharged to travel back to the Marshall Islands on June 18th. The Hospital Summary and Pathological Reports from the Cleveland Hospital are incorporated in these records.

FINAL DIAGNOSIS: Degenerative cystic thyroid adenoma.

DISCHARGE MEDICATIONS AND RECOMMENDATIONS: In view of the fact that the patient had adequate thyroid function and so little of the thyroid was removed at surgery, no supplementary thyroxin treatment was prescribed. The patient was advised to have the lesons of her cervix checked at intervals. We will carry out regular followup examinations on this case.

Robert A. Conard, M.D.

Received: June 29, 1973
Typed: July 2, 1973
er

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Utirik 2212
(NAME)
8-45-38R
(UNIT NO.)

ADMITTED: June 2, 1973 DISCHARGED: June 10, 1973

This 54-year-old Marshallese woman, who had a slight exposure to fallout radiation in the Marshall Islands in 1954, was admitted here for studies of thyroid nodularity and evaluation of physical status for thyroid surgery.

MEDICAL HISTORY: In 1966, a small 0.5 cm nodule was found in the right lobe of the thyroid. Subsequent examinations showed an increase in the number of nodules of the gland, and in March of this year three distinct nodules were palpated, two in the left lobe and 1 near the isthmus, the largest being about 2 cm in the lower part of the left lobe. The nodules were slightly tender to palpation. No lymphadenopathy was noted. She noticed the "lumps" on swallowing. She appeared euthyroid though her T-4 level in March was slightly low (3.7 µg%).

She was exposed to 14 rads of gamma radiation and about 22 rads to her thyroid gland in 1954 from fallout exposure. No effects from this exposure have been discernible. Examinations over the 19 years since the exposure have revealed the following: occasional cough, frequent worms in stools, joint pains with arthritic changes, tonsillar hypertrophy, multiple lipomata.

FAMILY AND SOCIAL HISTORY: Non-contributory.

PHYSICAL EXAMINATION: Positive findings on physical examinations included: slight obesity, multiple lipomata (asymptomatic), reduced hearing left ear and BP generally normal, but slightly elevated at times; slight cardiomegaly with no evidence of decompensation. Her thyroid findings have not changed since those outlined above for the March examination. She appears euthyroid.

LABORATORY AND X-RAY DATA: Thyroid scan shows a large non-functioning nodule in the lower left lobe. Radioactive iodine uptake and response to TSH stimulation were adequate. T-4 level is not available yet. Her serum was non-reactive for antithyroid globulin antibodies. Chest x-ray showed cardiomegaly but the lungs were clear. EKG showed incomplete bundle branch block which was not considered significant. The hemogram showed slight lymphocytosis (46%) and eosinophilia (14%) and increased ESR to 2[illegible]. She was found to be diabetic with FBS of 262 mg% and spillage of sugar in the urine. Her kidney function was somewhat reduced with BUN of 27 mg%, urine albumin 50 mg%, creatinine clearance 40.5%, urea clearance 30 mg%. The AG ratio was 1.13 (not unusual in the Marshallese), cholesterol 264 mg%, triglycerides 148 mg%, electrolytes and liver test generally normal. The syphilis serology was reactive (titer of 2) but was not considered significant in view of past history of yaws in so many of these people. Her stools were positive for trichuris trichura.

HOSPITAL COURSE: She remained generally asymptomatic during her 8-day hospital stay here except for a slight cough and non-specific muscle pains at times. With the finding of diabetes, she was placed on a 1400 calorie (ADA) diet. Since she continued to spill some sugar

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Utirik 2212
(NAME)
8-45-38R
(UNIT NO.)

in the urine, she was placed on 15 units NPH Insulin daily which controlled her diabetes. With the diabetes controlled, the slightly impaired kidney function was not considered serious enough to preclude surgery and she was discharged June 10th for transfer to Cleveland.

At Cleveland Metropolitan General Hospital, thyroid surgery was performed by Dr. Brown Dobyns. The day before surgery she was given 25 μCi ^{131}I in order to do autoradiographic studies on the removed thyroid tissues. At surgery, a left lobectomy was performed removing several cystic nodules from that lobe. A diagnosis of Hurthle cell adenomata on frozen section prompted the complete removal of that lobe. A further adenoma was removed from the isthmus and several tiny nodules were removed from the right lobe. The patient withstood surgery well and her convalescence was uneventful. She was continued on insulin and dietary treatment.

FINAL DIAGNOSIS: Mixed follicular cell and Hurthle cell adenomata of the thyroid.

Borderline hypertension with slightly reduced kidney function.

Diabetes mellitus

DISCHARGE MEDICATION: Under normal circumstances with a left thyroid lobectomy and subtotal on the right, somewhat depressed metabolism might be expected and 0.1 to 0.2 mg of L-thyroxin would be given daily. However, since this lady is older, has diabetes and a tendency to hypertension, it was deemed advisable to observe her for a few months before making the final decision on treatment. She was advised to attend the diabetic clinic at the Majuro hospital and maintain her diet. The practitioner accompanying the patients was advised to continue the insulin therapy. If the patient later was moved back to her isolated home at Utirik Island, the treatment would have to be re-evaluated.

Robert A. Conard

Robert A. Conard, M.D.

RAC:bwa
Dict: 6/27/73
Typed:6/29/73

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
— Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Utirik 2221
(NAME)

8-45-37R
(UNIT NO.)

ADMITTED: June 2, 1973 DISCHARGED: June 10, 1973

MEDICAL HISTORY: This 71-year-old Marshallese woman was found to have a small thyroid nodule at the time of the regular annual examination of the Marshallese exposed to radioactive fallout this past March. The nodule was pea-sized, freely movable, and in the midportion of the right lobe. There was no lymphadenopathy noted. She was brought to the U.S. and admitted to this Hospital for thyroid studies and evaluation for thyroid surgery. Her previous thyroid history had been negative and she had always appeared euthyroid with low-to-normal thyroxin levels. Because of her radiation exposure, surgical exploration was deemed advisable.

She was exposed on Utirik Island in 1954 to about 14 rads of gamma radiation from fallout with a thyroid dose of about 22 rads (partly from radioiodine absorption). She showed no effects of the slight exposure, and the principal medical findings over the 19-year period since exposure have concerned the development of essential hypertension with possibly slight kidney involvement. Complaints have largely centered around arthritic pains and stiffness of the knees and legs and the development of poor vision.

FAMILY AND SOCIAL HISTORY: Irrelevant.

PHYSICAL EXAMINATION: This slender, elderly, alert lady appeared healthy, euthyroid, and well-preserved for her age. The thyroid findings were as described above and during the March examination. Other findings included an early cataract formation of the left eye and the presence of hypertension (BP 200/96), and a moderately loud systolic murmur. The heart was not thought to be enlarged and there was no evidence of cardiac decompensation. Pain and stiffness on moving the knees and legs may have been associated with arthritic changes.

LABORATORY AND X-RAY DATA: The thyroid scan showed a "cold" nodule at the lateral border of the right lobe. Radioactive iodine uptake was low-normal, and TSH administration showed reduced thyroid reserve. Her serum was non-reactive to antithyroid globulin antibodies. Chest x-ray showed cardiomegaly and aortic sclerosis; slight increase in density near the cardiac apex "probably due to old inflammatory disease", and a slight deviation of the trachea. X-ray of the knees was negative. EKG was within normal limits. The hemogram was normal except for 8% eosinophils and an ESR of 38. Aldosterone level was normal. Some kidney dysfunction was evidenced by BUN of 27, urine albumin 50 mg% with 8-12 RBC/HPF, urea clearance 55% and creatinine clearance 39%. Other clinical chemistry tests were generally negative, including tests for liver function, electrolytes, lipids, and serum proteins. Stools were positive for ascaris lumbricoides and trichuris trichura. Syphilis serology was slightly positive (titer of 2) but this low level is not considered significant, particularly in view of possible yaws in the past which was prevalent in these people.

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Utirik 2221
(NAME)

8-45-37R
(UNIT NO.)

HOSPITAL COURSE: Her 8-day stay at this Hospital was uneventful and she remained generally asymptomatic except for a few non-specific muscle pains. Her cardiovascular/renal disease was not considered sufficiently advanced to preclude surgery and she was discharged June 10th,1973, and taken to Cleveland. Just prior to discharge, she was given 25 μCi of ^{131}I to ensure complete removal of the thyroid at surgery and for study of activity of thyroid tissues removed.

At the Cleveland Metropolitan General Hospital, Dr. Brown Dobyns performed thyroid surgery on June 11th. A 1.5 cm nodule was removed from the right lobe and a right lobectomy was performed. The pathological diagnosis was "thyroid adenomata".She withstood surgery well, with no complications. Post-operative convalescence was uneventful and she was discharged June 18th for return to the Marshall Islands. The hospital summary and pathological reports from the Cleveland Hospital are inserted in her BNL record.

FINAL DIAGNOSIS: Thyroid adenomata.

Hypertension.

Reduced kidney function, secondary to hypertension.

DISCHARGE MEDICATION: In view of her low thyroid reserve and right lobectomy performed, supplementary thyroxin treatment was considered but, in view of her hypertension and age, it was considered better to observe her and see if supplemental therapy was necessary. She will, therefore, be observed at regular intervals on her return home to check on her thyroid status.

Robert A. Conard

Robert A. Conard, M.D.

RAC/bwa
Dict: 6/27/73
Typed:6/28/73

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap unexposed 829
(NAME)

08-45-39R
(UNIT NO.)

ADMITTED: 6/2/73 DISCHARGED: 6/10/73

During the March medical survey in the Marshall Islands, this 36 year-old Marshallese woman who is part of the comparison population unexposed to fallout was found to have a thyroid nodule and was brought to the United States and admitted to this hospital for thyroid studies and evaluation of physical status for possible thyroid surgery.

MEDICAL HISTORY

During the March examination a thyroid nodule, a discrete spherical mass was detected which was moderately firm, movable and slightly tender to palpation. No lymphadenopathy was noted. She appeared euthyroid and previous examinations showed no evidence of thyroid abnormalities.

Along with the population exposed to fallout she has been examined as part of the comparison population for the past 16 years. She is the mother of 11 children and has been healthy. With only a few findings of any significance - slight anemia at one time, one miscarriage, cervical erosion and occasional fungus infections of the skin. Her FAMILY and SOCIAL HISTORY are non-contributory.

PHYSICAL EXAMINATION:

This slender, well nourished Marshallese lady appeared alert and euthyroid. The thyroid findings were the same as those reported above during the March examination. The remainder of the physical examination was essentially negative except for slight abdominal tenderness and discomfort which she experiences prior to menstruation.

LABORATORY AND X-RAY DATA:

Thyroid scans showed a 2 cm, poorly functioning nodule in the lower left lobe of the gland, Radioactive iodine uptake was normal and response to TSH stimulation was good. Serum was nonreactive for antithyroid globulin antibodies. Tests X-Ray and EKG were normal. The hemogram showed slight lymphocytosis and an elevation of the ESR to 38 (which is not unusual in the Marshallese.) In view of her previous anemia it was interesting that her Fe and TIBC were within normal limits. Tests for liver and kidney function, electrolytes and lipids were normal. Serum proteins were somewhat high (9G) but the electrophretic pattern was not unusual for the Marshallese (high gamma globulin levels). The stools were negative for ova and parasites.

HOSPITAL COURSE:

During the early part of her 8-day stay, here, she had premenstrual abdominal discomfort and anorexia. With onset of menstruation these symptoms disappeared and she remained a symptomatic for the rest of her hospital stay here. Just prior to discharge on June 10th, for travel to Cleveland for surgery she was given 25 μ Ci ^{131}I orally in order to carry out autoradiographic procedures on thyroid tissues to be removed at surgery.

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap unexposed 829
(NAME)

08-45-39R
(UNIT NO.)

HOSPITAL COURSE CONTINUED: Thyroid surgery was carried out June 12th at the Cleveland Metropolitan General Hospital by Dr. Brown Dobyns. A discrete 2 cm diameter mass was found in the left lobe and removed along with subtotal lobectomy. The remainder of the thyroid appeared to be normal and no lymph nodes were noted. A pathological diagnosis was PAPILLARY CYSTIC adenoma and 2 small thyroid adenomata were also found. She withstood the operation well and her convalescence was uneventful. She was discharged June 18th and returned to the Marshall Islands. Copies of the Hospital Summary and Pathological Reports from the Cleveland Hospital are included in our records.

FINAL DIAGNOSIS: Papillary cystic adenoma and thyroid adenomata.

DISCHARGE MEDICATION: Since thyroid function was good in this patient and so little thyroid tissue removed at surgery no supplemental thyroxin or other treatment was prescribed. She will be followed in subsequent examinations in the Marshall Island.

Robert A. Conard

Robert A. Conard, M.D.

Received: June 29, 1973
Typed: July 2, 1973
er

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 83
(NAME)

08-50-54 R
(UNIT NO.)

CIRC 63

ADMITTED: 27 May 1974 DISCHARGED: 3 June 1974

This 20-year-old Marshallese boy was admitted for thyroid studies and consideration for possible surgery in Cleveland.

MEDICAL HISTORY: In September 1973, a small nodule was noted in the left lobe of the thyroid. During the recent March 1974 survey, an additional slight enlargement of the right lobe was palpated. He has always appeared euthyroid and the T4 levels have been within normal limits. This boy was one of 4 Rongelap children exposed in utero. He was exposed near the end of the second trimester to 175 rads whole body gamm radiation from accidental fallout, and the thyroid also received an indeterminate dose from radioiodines which had been absorbed by the mother. When first seen, 3 months after birth, the only possible indication of radiation exposure was a transitory depression of his blood platelets. He has been examined regularly by our medical team and no serious findings have been noted. He has had the usual childhood infections, and on one occasion he had a brief hospitalization for acute URI. His growth and development have been normal.

PHYSICAL EXAM ON ADMISSION: This Marshallese boy is well-developed and well-nourished. He appears euthyroid. Physical exam is generally negative except for the thyroid findings. A small, 2 mm, freely moveable nodule was noted in the left lobe and a bumpy irregularity in the upper right lobe. No tenderness of the gland or lymphadenopathy was noted.

LABORATORY & X-RAY DATA: Thyroid scans showed a discrete area of decreased radioactivity in the middle portion of the left lobe. ^{123}I uptake was 24.6%, T4 level was 6.7 µg% in March. The hemogram and blood chemistry data were within normal limits.

HOSPITAL COURSE: The hospital course was uneventful. He ate and slept well and was completely asymptomatic. The patient was given 20 µCi ^{131}I just before discharge to Cleveland for autoradiographic analysis of excised lesions.

At the Cleveland Metropolitan General Hospital on 4 June 1974, thyroid surgery was performed by B. M. Dobyns, M.D. There was a 3-4 mm mass in the left lobe near the isthmus, a 1 cm soft mass in the superior pole and the right lobe contained a 1 cm soft mass. These were removed and the histological diagnoses were benign adenomas, one of which was trabecular in pattern. His recovery from surgery was uneventful with no complications. By 9 June the wound was healing nicely and the patient was discharged for travel back to the Marshall Islands. A summary of his hospitalization including surgical and

BNL 720A

pathological reports from the Cleveland Hospital are being incorporated into the chart of this hospital.

DISCHARGE MEDICATION & INSTRUCTIONS: The patient was resumed on thyroxin medication (Synthroid®) 2.1 mg/weekly. Dr. Knudsen, the resident physician in the Marshall Islands, will carry out careful followup examinations on this patient.

DISCHARGE DIAGNOSIS: Benign thyroid adenoma, surgically removed.

Robert A. Conard

Robert A. Conard, M.D.

cfh
Received: 6/25/74
Typed: 6/27/74
ac: R. Conard on 7/1/74

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap unexposed 841
(NAME)

08-50-53 R
(UNIT NO.)

CIRC 63

ADMITTED: 27 May 1974 DISCHARGED: 3 June 1974

This 41-year-old Marshallese woman was admitted to this hospital for thyroid studies due to nodularity in the gland, and evaluation for possible thyroid surgery.

MEDICAL HISTORY: In March 1974, during routine physical examination, the patient was found to have a firm nodule in the lower left pole of the thyroid gland and possibly another tiny nodule in the upper right lobe. The nodules were non-tender and no lymphadenopathy was noted. She appeared to be euthyroid. The patient is one of the unexposed people in the Rongelap compariosn population. Since exams began in 1957 on this group, her health has been generally good, but with an increasing tendency to obesity and a recent development of hypertension. Her weight increased from 145 lb in 1957 to 170 lb at present. The past few years she has had frequent headaches, and the BP has become elevated to around 170-180/100-115. She has had about annual pregnancies, resulting in 9 healthy children, 2 miscarriages, and 1 child dying at the age of 1 year. Except for occasional joint pains and presence of worms in her stools, her history is otherwise negative generally. She has had no symptoms referable to kidney disease.

PHYSICAL EXAMINATION: The patient is somewhat obese and is 20-22 weeks pregnant. She appears euthyroid and generally in good health. Exam of the thyroid reveals a 1 cm nodule in the lower left lobe with a companion nodule also in the mid right lobe. The nodules were non-tender and no lymphadenopathy was noted. Her BP was 150-180/105-115. The heart and lungs appeared normal, as well as the peripheral vascular system. Funduscopic exam was generally negative except for slight increase in light reflex. The physical exam was otherwise negative except for the presence of a cystocele and rectocele.

LABORATORY & X-RAY DATA: No scans or thyroid uptake studies were performed because of her pregnancy. X ray of the chest showed the heart to be top-normal in size, but otherwise negative. EKG was normal. Cholesterol was 336 mg%, and triglycerides were 182 mg%. Creatinine clearance was slightly low. Aldosterone was 50 mg/24 hr. VMA was normal. The urine showed 20 mg albumin, but was otherwise negative. Stool examinations revealed the presence of whip worms and trichirus trichura.

HOSPITAL COURSE: During her hospitalization, the patient remained asymptomatic, and ate and slept well. She was completely ambulatory. Her BP varied from 130/90 to 160/110. From the lab findings a diagnosis of mild essential hypertension

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap unexposed 841
(NAME)

08-50-53 R
(UNIT NO.)

CIRC 63

seems appropriate and should not preclude surgery.

At the Cleveland Metropolitan General Hospital on 4 June 1974, thyroid surgery was performed by B. M. Dobyns, M.D. The gland was found to be 2-3 times normal size, soft and very vascular (presumably related to pregnancy). There were 2 adenomas, one in the right lobe and one in the left; one filled with colloid and the other with necrotic liquid. The pathological diagnoses were thyroid adenomas, one with cystic degeneration and the other "a regenerating-degenerating microadenoma". Recovery from surgery was uneventful and the wound healed nicely. There were no complications related either to hypertension or pregnancy. The patient was discharged for travel to the Marshall Islands on 9 June 1974. The hospital summary and a surgical and pathological report are being included in the chart of this hospital.

DISCHARGE MEDICATION: Dr. Knudsen, the resident physician in the Marshall Islands, was advised that the obstetrician on this case suggested that if hypertension persists after pregnancy termination, some form of medical management should be instituted. It was not considered necessary to start this patient on supplementary hormone therapy.

DISCHARGE DIAGNOSES:

1. Benign thyroid adenomas, surgically removed.
2. Essential hypertension (mild).

Robert A. Conard

Robert A. Conard, M.D.

cfh
Received: 25 June 1974
Typed: 27 June 1974
cc Dr. Conard 7/1

BNL 720A

HOSPITAL OF THE MEDICAL RESEARCH CENTER,
BROOKHAVEN NATIONAL LABORATORY
UPTON, NEW YORK 11973
Area Code 516 YAphank 4-6262

DISCHARGE SUMMARY

Rongelap 51	(NAME)
08-50-52 R	(UNIT NO.)
CIRC 63	

ADMITTED: 27 May 1974 DISCHARGED: 3 June 1974

This 45-year-old Marshallese female was admitted for evaluation of her thyroid status in anticipation of possible surgery.

MEDICAL HISTORY: In September 1973, a small nodule 0.5 cm in diameter was noted in the region of the lower left lobe of the thyroid. By March 1974, the nodule appeared to have grown slightly in size. She had always appeared euthyroid and her T4 levels were in the normal range. The patient was accidentally exposed to fallout radiation in 1954 at age 24. She received an estimated 69 rads of whole body gamma radiation which caused mild depression of her blood elements during the first few weeks after exposure. Fallout contamination of the skin caused mild, transitory, beta burns of the skin during the first few weeks also. She also absorbed internally some radionuclides, the most serious of which were radioiodines. She remained generally healthy until about 1970, except that she was somewhat underweight and had an early menopause (age 42), after having had one miscarriage and one child. Since 1971 she has complained of frequent bouts of coughing, dyspnea, night sweating at times, and chest pain. She has had white-to-yellowish sputum, but denies hemoptysis. She claims to become dyspneic on exertion, and during the bouts of coughing sleeps propped up on pillows.

LABORATORY & X-RAY DATA: Thyroid scan using ^{123}I shows a focal area of decreased radioactivity in the middle and lower 1/3 of the left lobe of the thyroid laterally. Her RAI uptake was 15.8%. Chest x ray showed increased lung markings, possible due to chronic bronchial disease. Numerous exams for AFB of the sputum were negative, both on smear and by culture. No other consistent pathogenic organisms were found in the sputum. The EKG showed some T-wave abnormalities, which were not necessarily considered significant. Her hemogram was negative except for increased eosinophils which may have been related to the finding of whip-worm and trichuris trichura in her stools. She had slightly increased blood proteins, particularly globulins, which is not an unusual finding in the Marshallese people. She had a positive syphilis serology, reactive, titer II. (In the Marshallese, infection with yaws, which was endemic years ago, not infrequently results in positive serology.)

HOSPITAL COURSE: During the first few days of hospitalization, the patient remained in bed a good deal of the time. She coughed frequently and had slight increase in respiration, bringing up a whitish-yellowish sputum. She became dyspneic on exertion. She had a low-grade fever in the evenings. Her appetite was poor. In view of possible TB infection, isolation procedures were instituted, though subsequent tests and sputum findings did not substantiate such a diagnosis. Considering the possibility of bronchial infection, she was given tetracyclines and Tedral for cough. She improved in the last few days of hospitalization

BNL 720A

and was able to get up and walk around, with an increase in appetite. She was considered fit to travel to Cleveland on 3 June 1974.

When the patient arrived at the Cleveland Metropolitan General Hospital she was improved. Her chest x rays were reviewed by a chest specialist and the EKG by a cardiologist. Their opinions were that the ? hilar shadows were not suggestive of tuberculosis but of bronchitis, and the T-wave changes in the EKG were not considered significant. Their conclusions were that these conditions did not preclude surgery. Positive pressure ventilation and other pulmonary exercises were instituted and resulted in removal of large amounts of bronchial mucus with marked improvement in the patient's status prior to surgery.

On 5 June 1974, thyroid surgery was carried out. The gland was found to be small, thin, and brownish in color and quite friable. A 1-cm mass was removed from the mid left lobe which was filled with necrotic fluid and a smaller 5 mm tongue of adenomatous tissue removed from near the isthmus. The pathological diagnoses for both masses were "adenomas".

The patient's postoperative convalescence was uneventful with no pulmonary problems. A positive serological test for syphilis was reported. This was confirmed on re-check.

DISCHARGE MEDICATION & INSTRUCTIONS: Dr. Knudsen, the resident physician in the Marshall Islands, was notified of the return of this patient and advised about the positive serological test and the desirability for continued postural drainage procedures. She was resumed on thyroid hormone treatment (Synthroid®) and given a supply to take back to the islands with her. She will be given careful followup exams.

DISCHARGE DIAGNOSES:

1. Benign thyroid adenomas, surgically removed.
2. Chronic bronchitis.

Robert A. Conard

Robert A. Conard, M.D.

cfh
Received: 6/25/74
Typed: 6/27/74
cc - Dr. Conard on 7/1

BNL 720A

Appendix 11

Hospital Summary of Leukemia Case

Admitted on 10-2-72 to National Cancer Institute, Hematology and Supportive Care Branch

The patient is a 19-year-old man from the Marshall Islands, who transfers from the hospital of the Brookhaven National Laboratory with a tentative diagnosis of acute leukemia.

PRESENT ILLNESS:

This patient has been followed with yearly physicals and blood counts by the Medical Research Center of the Brookhaven National Laboratory since age one, at which time he suffered an accidental radiation exposure of 175 total body rads. He had a transient leukopenia one month following exposure but since then has had normal blood counts at his yearly examinations. At the time of a routine evaluation in August 1972, his peripheral blood white count was 1,200, platelet count 119,000, and red blood cell count 4.4 million. Attempt at a bone marrow aspiration at that time was unsuccessful, and on September 29, 1972, the patient was flown to the Brookhaven Medical Research Center for further evaluation. He had not complained of easy fatigability, spontaneous or unusual bruising or bleeding, joint or muscle aches, or an increased number of infections. At the time of admission on September 29, the hemoglobin was 12.5, white blood cell count 700 with 26% polys, 28% lymphocytes, and the remainder abnormal monocytic precursors. Urinalysis, liver function tests, and prothrobin time were all within normal limits. A bone marrow aspirate in the left posterior iliac crest was performed and revealed a predominance of early myeloid precursors with some dissociation of cytoplasmic and nuclear maturation and presence of Auer rods in the cytoplasm of myeloblasts. On September 30, the patient had an asymptomatic temperature elevation to 101°F. Physical examination then revealed slight injection of the pharynx and a chronically scarred left tympanic membrane. Chest x-ray was normal. Cultures were taken, and the patient was begun at that time on treatment with keflin and gentamicin. He was placed on isolation. On October 1, the patient was afebrile, and arrangements were made to transfer him here.

PAST HISTORY:

Operations: Subtotal thyroidectomy.

Accidents: None

Medicines: Occasional "Anacin" tablet; synthroid, 0.3 mg. daily from 1968 until May 1972.

FAMILY HISTORY:

Mother, father, six brothers, and three sisters are all living and in good health in the Marshall Islands.

SOCIAL HISTORY:

The patient was born and has lived all his life in the Marshall Islands except for one visit to Boston, where he was hospitalized for a thyroidectomy in 1967. He graduated from high school last year. He has working knowledge of English, although he is not fluent.

REVIEW OF SYSTEMS:

Head: The patient gets a rare fronto-occipital headache, which is relieved by Anacin. The frequency of these headaches has not changed recently. He has no known seizure disorder, no history of syncope. Ears - No complaints. Eyes - Vision good. The patient does not wear glasses. Nose and Mouth - Negative.

Pulmonary: No chronic cough, no shortness of breath.

Cardiovascular: Negative.

Gastrointestinal: Negative.

Skin: The patient has had multiple skin infections on the legs, often resulting from cuts and scratches. In addition, he has had boils on the buttocks over the last few months and a mildly prutitic, depigmenting rash over the thorax for the last six months.

Endocrine: Subtotal thyroidectomy 5 yrs prior to admission for multiple benign adenomas. On synthyroid since.

PHYSICAL EXAMINATION:

Vital Signs: Blood pressure 110/60; pulse 90; respirations 16.

General Appearance: This is a well-developed, muscular, young man in no distress.

Head, Eyes, Ears, Nose and Throat: The head is symmetric. There is no bony or scalp tenderness. Pupils are equal, round, and reactive to light and to accommodation. The extraocular movements are intact. Visual fields are intact to confrontation testing. There is no nystagmus. The fundi are normal. The Weber test does not lateralize. The right tympanic membrane is slightly scarred and retracted; the left tympanic membrane is distorted, dull, particularly in the postero-inferior quadrant, with a slight tan exudate at the base. The pharynx is normal. There is slight inflammation surrounding the right mandibular third molar, which is partially covered by a soft tissue flap.

Neck: Supple. There is no venous distention. Two well-healed horizontal surgical scars are present anteriorly over the neck. No palpable thyroid tissue is present.

Chest: The chest expands symmetrically. The lung fields are clear to percussion and auscultation. The lungs bases move to percussion.

Cardiac: The point of maximum intensity of the cardiac impulse is in the fifth intercostal space in the midclavicular line. Cardiac rhythm is regular. There is a Grade II/VI soft systolic murmur head best at the aortic area, radiating slightly toward the left sternal border but not toward the neck. The second heart sound splits normally.

Bones, Muscles, and Joints: The bones are not tender. There is no costovertebral angle tnederness.

Abdomen: The abdomen is soft, nontender. Bowel sounds are normal. Hepatic span to percussion is 10 cm. Liver spleen, and kidneys are not palpable, and there are no abnormal abdominal masses or bruits.

Genitalia: Normal male.

Rectal: Examination is normal.

Extremities: The extremities are normal, with multiple healed scars over the legs.

Skin: There is a depigmented geographic rash over the thorax and upper extremities, which is slightly scaling in places.

Neurological: Examination is entirely within normal limits.

IMPRESSION:

1. Acute granulocytic leukemia.
2. History of resection of thyroid nodule, clinically euthyroid now.
3. Tinea versicolor.
4. Mild cutaneous furunculosis.
5. Chronic left otitis media.
6. Partially erupted right lower third molar with periodontitis.

PROGNOSIS:

Fifty percent chance of achieving remission of his leukemia; median survival about one year in other patients with a similar disease.

James Mabry, M.D./C/10-4-72 JM:kr D-X 10-5-72

Admitted on 10-2-72 to National Cancer Institute,
Hematology and Supportive Care Branch
Expired on 11-15-72 from National Cancer Institute,
Hematology and Supportive Care Branch

HOSPITAL COURSE:

Problem No. 1 - Acute Proganulocytic Leukemia:

On the morning of the second hospital day the patient had a posterior iliac crest bone marrow aspirate revealing decreased normal marrow elements and infiltration by cells with prominent eosinophilic cytoplasmic granules and prominent nucleoli. Some of these cells had Auer rods in the cytoplasm. The impression was that the marrow showed changes of acute promyelocytic leukemia. A bone marrow biopsy showed hypercellularity and infiltration by abnormal cells. Despite extensive marrow involvement, however, the patient did not have severe anemia or circulating abnormal cells when he first presented here. On October 12 he was discharged, feeling well, to be followed closely in the Special Ambulatory Care Clinic. The plan was to treat him for bleeding episodes, the appearance of blast cells in the peripheral blood, splenomegaly, or deterioration of coagulation parameters.

Unfortunately, after only two days in the Outpatient Clinic, the patient's platelet count fell to 7,000, and he developed an earache and ahd a low-grade fever. He was therefore readmitted and on October 15 begun on therapy with cytosine arabinoside, 100 mg. per meter squared intravenously every 12 hours, and 6-thioguanine, 90 mg. per meter squared orally every 12 hours. The patient's base-line prothrobin time, partial thromboplastin time, thrombin time, fibrinogen, fibrin split products, and factor VIII were within the normal range; nevertheless, because of the association of intravascular coagulation with acute progranulocytic leukemia, he was treated prophylactically with heparin, 0.5 mg. per kg. intravenously every 6 hours for the first five days of chemotherapeutic drugs. On the first day of treatment the white blood count was 1,700 with 21% neutrophils, 56% lymphocytes and 20% abnormal precursors; platelets were 32,000 (after transfusion) and hemoglobin 10.3. His white count remained low throughout the remainder of his hosital course; abnormal forms disappeared from the peripheral blood on the ninth day of treatment; platelet transfusions were administered every two or three days in an effort to keep the platelet count above 20,000. Serial bone marrow examinations revealed: on day five, hypercellular marrow with 80% abnormal cells; on day seven, hypercellular marrow with 90% progranulocytes; on day twelve, hypercellular marrow with 95% progranulocytes; on day fourteen, hypercellular marrow with 82% progranulocytes; on day nineteen, normocellular marrow with 80% progranulocytes; on the day prior to death, after twenty-two days of treatment, a hypocellular marrow with persisting foci of abnormal promyelocytes. On the day prior to death the patient's hemoglobin was 8.3, white cell count, 2,100 with 100% lymphocytes, and platelet count 11,000. He had received 10 units of packed red blood cells and had received platelet transfusions on 18 days of his hospital stay. During the last two weeks of life he had very poor increments in platelet count after platelet transfusions, probably because HLA identical platelets were not available for transfusion.

Laboratory Values on Admission: BUN 13, creatinine 1.3, sugar 98, amylase 46, cholesterol 116, electrolytes normal, calcium, magnesium and phosphate normal, alkaline phosphatase 50, total protein 8.5, albumin 4.1, total bilirubin 0.1, SGPT, SGOT and LDH all normal, uric acid 5.8. While being treated with cytosine arabinoside the SGPT and SGOT rose out of the normal range and in the one week prior to death he had bilirubin elevations to as high as 6 as well. There was never any evidence of disseminated intravascular coagulation on twice weekly coagulation screening tests.

Problem No, 2 - Thyroid Status:

Clinically, the patient was euthyroid. Thyroxine level was 3.7. Throughout his hospital stay he was continued on L-thyroxine, 0.3 mg. daily.

Problem No. 5 - Otitis Media:

The patient was treated with oxacillin and gentamicin followed by ampicillin for a total of seven days with resolution of his left otitis.

Problem No. 6 - Periodontitis:

The dental consultant recommended managing his molar periodontitis with frequent local lavage, which was done under his supervision. The initial inflammation resolved after several days, but during the last three weeks of his hospitalization he had severe peridontal inflammation, worse on the right. In addition, a right subauricular swelling appeared late in the second hospital week and persisted until the time of death. Ear, nose and throat consultant thought this represented parotitis, but reactive adenopathy from the periodontitis could not be excluded. During the last three weeks of hospitalization he was on nearly continual antibiotic treatment with oxacillin and gentamicin or keflin and gentamicin. On November 8 Proteus mirabilis and Pasteurella multicida were cultured from the blood. These organisms had previously been cultured from the mouth as well, and a likely source of sepsis was his periodontitis. He continued to be febrile throughout the rest of the hospital course, but subsequent blood cultures were sterile.

Problem No. 7 - Pneumonitis:

On November 7 the patient had gram-negative sepsis; on November 8 he complained of a brassy cough; on November 9 he was generally tachypneic and quite anxious, with cyanotic nail beds. Physical examination revealed right axillary rales and chest x-ray showed a patchy alveolar infiltrate in the right upper, middle and lower lobes. Arterial oxygen saturation was 45 mm. of mercury on room air and pCO_2 and 20 mm. of mercury. Cultures of the scanty blood-tinged sputum grew only a few colonies of Klebsiella. Over the next two days he had increasingly severe respiratory distress with gradual opacification of both hemithoraces on chest x-ray. His sputum became frankly bloody. On November 12 he was intubated by the nasotracheal route and placed on a volume cycled respirator. He was begun on treatment empirically with pyrimethamine and sulfadiazine for the possibility of Pneumocystis carinii pneumonitis. Management

during the last two days was complicated by the patient's agitation and inability to cooperate with the respirator, so that he was eventually given curare. The terminal event at 2:40 a.m. on November 15, 1972, seemed to be an intrapulmonary hemorrhage followed by hypoxemia and hypotension.

FINAL DIAGNOSES:

1. Acute progranulocytic leukemia
2. Resected thyroid nodule.
3. Tinea versicolor.
4. Mild cutaneous furunculosis.
5. Chronic left otitis media.
6. Periodontitis with subsequent sepsis.
7. Pneumonitis with terminal intrapulmonary hemorrhage

James Mabry, M.D./C/12-5-72
JM:nht K-I 12-11-72

Attachment: Admission History and Physical Examination

Appendix 12

Personnel Monitoring

A. Urine, 1970-1974

Year	Subject No.	Vol., ml	Ca, mg/liter	^{90}Sr, pCi/liter	^{137}Cs, nCi/liter
			Rongelap		
1970	7	2500	180	2.2	1.5
	9	2680	68	1.0	0.80
	11	1100	91	1.4	1.0
	34	270	220	4.2	3.1
	48	920	34	5.8	3.1
	53	980	170	7.3	2.5
	67	455	75	6.4	5.2
	70	135	52	2.4	1.3
	73	525	97	2.3	4.1
	80	1540	200	2.0	1.7
	805	275	110	7.4	4.4
	811	265	600	6.5	3.9
	835	330	110	5.7	4.1
	881	540	74	1.7	2.8
	882	1180	76	1.0	0.87
	934	920	150	2.5	3.5
	948	800	180	2.0	0.77
	1050	620	120	1.6	2.1
	1520	355	220	3.3	4.0
	1523	1520	220	3.4	2.6
				$\bar{X}$ = 3.5	$\bar{X}$ = 2.7
1971	34	625	130	3.7	5.9
	41	590	130	2.1	1.2
	61	1260	67	2.6	0.45
	66	560	160	2.4	2.7
	67	260	870	8.3	4.6
	73	830	210	2.6	2.1
	77	700	87	3.1	2.7
	802	210	160	9.7	3.7
	825	470	110	6.3	2.5
	827	330	200	2.5	2.0
	834	320	280	3.3	2.2
	864	830	140	2.6	2.2
	932	1220	55	1.3	1.4
	948	440	33	0.90	0.34
	1520	470	240	3.7	2.5
				$\bar{X}$ = 3.67	$\bar{X}$ = 2.4
1972	1	580	100	2.3	1.2
	14	960	91	2.6	2.6
	17	680	23	2.2	5.9
	34	85	84	<1.0	6.1
	48	250	29	1.7	7.0
	73	980	86	3.4	4.2
	845	385	360	2.3	2.9
	878	290	60	2.2	5.0
	882	470	110	1.3	1.3
	896	330	190	6.4	2.1
	1050	350	150	1.3	2.9
	1517	510	40	1.5	1.0
	2228	240	190	2.4	0.8
	2257	330	170	1.6	1.2
	2136	625	200	0.9	1.3
	2172	640	66	0.6	0.6
	2193	120	160	<0.8	0.7
	2195	470	56	1.0	0.5
				$\bar{X}$ = 2.4	$\bar{X}$ = 2.6

Appendix 12 (continued)

A. Urine, 1970-1974 (continued)

Year	Subject No.	Vol., ml	Ca, mg/liter	^{90}Sr, pCi/liter	^{137}Cs, nCi/liter	$^{239,240}Pu$, pCi/liter
			Rongelap			
1973	5	417	200	8.0	1.8	0.02
	12	180	210	7.7	2.7	0.05
	17	200	74	8.3	7.7	0.09
	48	410	32	2.8	1.9	0.02
	64	170	430	12.5	5.8	0.05
	66	432	190	2.8	2.1	0.05
	73	440	270	3.8	3.7	0.02
	80	215	620	5.1	5.3	0.06
	832	37	130	4.3	6.2	1.7
	83[illegible]	120	73	4.8	6.0	0.11
	843	125	490	10.9	8.1	0.11
				$\bar{X}$ = 6.45	$\bar{X}$ = 4.56	$\bar{X}$ = 0.21
1974	5	1160	350	0.9	0.7	
	41	840	560	2.0	0.7	
	66	800	1010	1.4	0.7	
	80	310	1100	5.8	4.0	
	855	260	170	3.8	4.9	
	869	680	540	4.2	35.0	
	1001	620	230	1.7	1.8	
	1525	220	140	2.3	8.9	
	1050	460	1120	2.3	1.8	
	7	530	760	1.9	lost	
	73	440	1160	4.5	1.6	
	834	580	2000	6.9	2.4	
	878	540	295	2.3	2.6	
	932	370	460	3.1	1.8	
			$\bar{X}$ = 706.8	$\bar{X}$ = 3.1	$\bar{X}$ = 5.5	
			Utirik			
1974	2212	37	220	0.4	0.7	
	2164	840	1304	2.2	1.7	
	2102	700	910	1.0	1.3	
	2160	400	480	0.7	1.6	
	2129	840	640	0.6	1.1	
	2102	190	240	0.7	0.9	
	2111	200	705	5.1	2.8	
	2124	700	1305	1.0	0.9	
	2172	1190	1020	0.6	0.9	
	2137	130	440	2.4	1.8	
	2150	740	820	0.9	1.4	
			$\bar{X}$ = 734.9	$\bar{X}$ = 1.4	$\bar{X}$ = 1.4	

A. Urine, 1970-1974 (continued)

Year	Subject No.	Vol., ml	Ca, mg/liter	^{90}Sr, pCi/liter	^{137}Cs, nCi/liter	^{238}Pu	^{239}Pu
			Bikini				
1970	Pooled	3640	120.0	1.2	0.100	0.002	0.002
	Pooled	3365	120.0	1.3	0.130	0.003	0.003
	urine G	1100		2.2		0.013	0.020
	urine M	930		1.9		0.015	0.024
	HASL* control	3000	160.0	1.0	0.012	0.003	0.003
	HASL control	1000		1.6		0.014	0.022
1971	Pooled	3920	54.0	0.96	0.217		0.004
	Pooled	2960	74.0	0.89	0.194		0.004
	Pooled	3300	110.0	1.22	0.211		0.004
	Pooled	500	100.0	3.9	0.110		
1972	Pooled	2700	204.0	4.2	0.910		
1973	Tonne	260	120.0	8.9	2.1		
	Jawel	280	100.0	5.7	1.1		
	Acme	250	240.0	5.5	2.6		
	Jone & Cheako	65	160.0	7.4	4.1		
	Nebal	150	230.0	6.2	0.9		
	Ruth	115	360.0	11.6	2.1		
	Pelepel-E-P	350	80.0	4.8	1.2		
	Bima Bikini, M-7	485	120.0	2.2	0.6		
	Distian Rep., family	300	210.0	5.6	1.5		
	Samwel	380	300.0	5.4	0.5		
	Aprizi	460	79.0	2.0	0.4		
	Ameta	220	130.0	18.9	1.3		
	Santos	410	100.0	1.9	0.4		
	Jormea	390	200.0	7.8	2.0		
			$\bar{X}$ = 173.5	$\bar{X}$ = 6.7	$\bar{X}$ = 1.5		
						$^{239,240}Pu$	
1974 Spring	Andrew	275				0.02	
	Edmila	220				0.06	
	Akiji	350				0.01	
	Tatus	370				0.01	
	Roja	350				0.01	
	Joji	1050				0.01	
	Libwe	1380				0.02	
	Bina	1230				0.004	
	Jormea	780				0.01	
	Beno	490				0.009	
	Mayao	155	290.0	1.2	1.0		
	Wanna	90	160.0	$\leq$0.1	1.0		
	Tojiro	105	440.0	3.0	0.6		
	Bonn	95	300.0	2.5	0.5		
	Tonny	50	160.0	$\leq$0.4	0.4		
	Enlik	140	220.0	2.4	0.8		
	Bear	200	360.0	3.2	1.7		
	Samuel	160	320.0	4.6	1.2		
	Ruth	300	380.0	3.8	3.2		
	Aetae	150	490.0	$\leq$0.1	0.5		
	Bokrok	110	290.0	$\leq$0.2	1.5		
			$\bar{X}$ = 310.0	$\bar{X}$ = 2.0	$\bar{X}$ = 1.1	$\bar{X}$ = 0.016	

*Health and Safety Laboratory, AEC, New York, N.Y.

B. Gamma Spectographic Data, 1974

Subject No.	Age	Wt., kg	Ht., cm	Potassium, g	nCi	^{137}Cs nCi/kg body wt.
			Rongelap males			
963	48	61.8	154.4	153.9	764	12.40
4	60	64.1	162.8	125.0	466	7.26
915	80	56.8	157.1	104.3	358	6.30
878	50	74.5	167.3	122.5	539	7.23
16	62	57.7	156.0	113.9	257	4.44
5	22	46.4	152.0	121.3	403	8.69
11	65	46.4	152.0	95.7	283	6.10
881	41	86.8	166.9	157.6	441	5.08
1517	53	64.1	154.6	150.5	442	6.89
41	64	52.3	159.1	109.9	307	5.86
814	22	59.5	161.7	158.0	969	16.3
863	24	66.4	165.4	185.5	590	8.89
7	57	54.1	155.5	123.7	509	9.41
850	60	59.1	160.5	130.6	444	7.52
834	40	63.6	156.3	159.4	721	11.34
845	52	68.6	161.2	157.6	368	5.36
882	41	55.5	161.3	133.3	243	4.37
82	70	53.6	151.1	101.3	400	7.46
80	66	56.4	147.9	105.5	287	5.09
27	48	66.4	156.8	153.3	666	10.03
1524	31	60.9	164.0	129.5	326	5.35
73	38	70.5	173.0	182.7	664	9.42
		$\bar{X}$ = 61.2		135.2	475	7.76
						(4.37 - 16.28)
			Rongelap females			
812	20	51.8	157.0	100.6	516	9.97
816	23	54.5	154.5	80.5	252	4.63
821	23	46.8	148.8	94.9	201	4.30
825	29	65.5	157.5	109.3	187	2.85
1050	39	75.9	155.9	112.3	393	5.16
14	42	60.0	150.4	64.1	373	6.22
1541	47	65.9	151.5	105.8	888	13.46
1525	31	57.7	134.4	63.2	249	4.32
843	45	53.6	146.0	80.6	209	3.90
1001	40	58.2	151.7	99.9	346	5.94
1	64	70.5	141.4	68.2	252	3.58
851	65	65.9	148.0	82.6	204	3.09
896	32	50.0	140.4	83.5	249	4.98
70	35	46.3	150.3	77.9	165	3.56
64	42	65.9	152.5	81.1	314	4.77
932	39	53.6	147.0	84.2	145	2.71
832	36	55.9	147.7	73.0	217	3.87
48	26	50.5	154.8	88.2	462	9.16
67	34 (8 mo preg)	80.5	156.7	109.4	266	3.31
66	50	63.6	155.0	82.7	283	4.45
835	40	60.0	147.0	72.7	190	3.16
78	59	69.1	156.3	76.5	431	6.24
58	79	51.8	143.2	57.2	202	3.90
859	81	52.3	143.7	62.7	298	5.69
					$\bar{X}$ = 304	$\bar{X}$ = 5.13
						(2.71 - 13.46)

B. Gamma Spectographic Data, 1974 (continued)

Subject No.	Age	Wt., kg	Ht., cm	Potassium, g	nCi	^{137}Cs nCi/kg body wt.
			Utirik males			
2125	56	69.5	171.5	144.4	184	2.65
2123	34	61.8	176.8	162.2	305	4.94
2137	38	76.8	165.5	160.2	526	6.84
2102	22	60.9	165.1	157.8	230	3.77
2167	38	92.7	168.6	155.8	228	2.46
2152	38	84.1	160.6	165.9	222	2.64
2166	57	54.5	160.4	144.3	230	4.21
2233	21	61.4	173.5	156.3	222	3.62
2185	50	59.5	159.5	156.4	208	3.46
					$\bar{X}$ = 262	$\bar{X}$ = 4.05
						(2.64 - 6.84)
			Utirik females			
2139	44	61.4	152.0	87.3	141	2.29
2172	32	89.5	159.0	128.9	147	1.64
2210	20	65.0	152.7	98.1	208	3.20
2160	24	68.2	137.5	108.5	132	1.93
2220	44	65.9	150.4	98.1	130	1.97
2244	63	77.3	153.5	74.9	74	0.96
2229	35	81.4	159.1	98.9	121	1.48
2164	36	77.7	147.8	84.5	128	1.64
2254	25	37.7	145.0	81.1	145	3.85
2161	48	34.5	131.2	65.8	103	2.99
2228	27	59.1	152.7	92.4	178	3.01
2193	50 (preg)	85.0	151.5	80.2	133	1.56
2212	53 (ctd. at BNL)	72.7	151.2	88.7	89	1.23
					$\bar{X}$ = 133	$\bar{X}$ = 2.13
						(0.96 - 3.85)

B. Gamma Spectographic Data, 1974 (continued)

Subject No.	Months on Bikini		Age	Wt., kg	Ht., cm	Potassium, g	^{137}Cs nCi	^{137}Cs nCi/kg body wt.
					Bikini males			
1	60		42	77.3	158.0	170.3	168	2.18
8	60		49	67.7	168.1	149.0	71.8	1.06
18	48		30	59.5	167.0	158.5	103	1.73
7	48		30	75.0	165.0	156.1	124	1.65
5	36		25	82.3	168.1	169.9	93.3	1.13
4	24		47	78.6	165.5	172.4	402	5.11
11	24		43	56.8	161.8	140.6	55.9	0.98
14	24		31	78.6	170.0	(197.6)	222	(2.82)
16	24		60	83.6	162.3	142.9	77.5	0.93
17	24		39	63.6	166.0	160.8	122	1.92
10	26		30	60.5	155.1	129.6	80.7	1.33
9	24		55	76.3	161.9	165.2	50.5	0.66
6	14		22	70.0	161.2	147.8	94.5	1.35
15	9		17	50.0	165.7	125.5	77.3	1.55
13	8		44	60.0	157.6	133.4	155.5	2.59
3	6	(from Rongelap)	49	85.1	165.9	162.5	290	3.40
12	4		16	70.0	172.4	168.3	75.5	1.08
2	3		54	100.5	170.1	158.8	42.9	0.43
							$\bar{X}$ = 128	$\bar{X}$ = 1.84
								(0.43 - 5.11)
					Bikini females			
8	60		54	54.5	149.6	88.7	30	0.55
5	36		23	96.4	155.4	124.1	73	0.76
7	36		29	64.5	151.9	110.6	108	1.68
13	36		15	46.8	146.4	85.6	36	0.78
9	24		45	70.5	149.3	101.5	116	1.65
10	24		27	57.7	142.7	106.9	25	0.44
11	24		28	46.8	145.8	85.2	92	1.96
6	18		44	50.0	146.0	94.6	58	1.16
12	12		13	49.1	145.1	106.4	77	1.74
1	7		31	77.3	149.9	91.4	252	3.26
2	4		45	82.3	150.7	59.4	18	0.22
3	4		60	73.6	152.5	93.7	33	0.44
4	4		20	62.3	156.9	94.0	29	0.47
						$\bar{X}$ = 95.6	$\bar{X}$ = 73	$\bar{X}$ = 1.15

Appendix 13

Report of The Ad Hoc Committee to Evaluate
The Radiological Hazards of Resettlement of The Bikini Atoll, May 1968

The Committee was convened to consider the question of whether the Bikini Atoll is safe for human habitation with respect to the radiological hazard. The detailed history of the various relocations of the Bikini natives is described in the appended material provided by Mr. Tobin (Appendix I).

We have examined the documents listed in Appendix II. In addition, we spent one and one-half days in detailed discussions with members of the 1967 Bikini Survey Team and other experts as shown in Appendix III. On the basis of the information provided, we have reached the following conclusions and recommendations:

1. The exposures to radiation that would result from the repatriation of the Bikini people do not offer a significant threat to their health and safety.
2. Such exposure may and should be further reduced by the following simple measures:
    a. Restrict rehabilitation for the present to the islands of the Bikini-Eneu complex. (See App. IV)
    b. Establish a village and immediate food crops on Eneu. No radiological precautions will be needed on Eneu because of its very low contamination level. (See App. V)
    c. Any village construction on Bikini Island should involve the covering of the site with coral rock as is the local custom.
    d. Radioactive scrap metal should be removed from the islands adjacent to former shot sites.

e. The population of land crabs should be sharply reduced because of their high content of Sr^{90}.

f. If pandanus trees are planted on Bikini Island, two inches of topsoil should be removed from the planting sites. The area of removal from each site should be equal to the area covered by the crown of mature trees.

3. Determinations should be made of body burdens of Cs^{137} and Sr^{90} at the end of the first year of residence on the Atoll and as appropriate thereafter. Baseline surveys prior to relocation would be desirable. Resurveys of environmental radiation levels on the Bikini Atoll and estimates or radionuclides in food should be made periodically.

4. Special efforts should be made to ensure a balanced and adequately nutritious diet. A dietary supplement of powdered milk would materially reduce Sr^{90} uptake by relieving the calcium deficiency usually associated with their diet.

Respectfully submitted:

Shields Warren
John C. Bugher
Robert A. Conard
John B. Storer
Paul Tompkins
John H. Harley
Charles L. Dunham
S. Allan Lough

Appendix 14

Gamma Radiation Measurements on Bikini Island, 1974

External gamma exposure rate measurements were made by N. A. Greenhouse and V. A. Nelson in the village area on Bikini Island during December 1974. The measurements were made with a Reuter-Stokes environmental radiation monitor, model RSS-111, which uses a pressurized ion chamber as a detector. The response of this instrument is relatively independent of gamma-ray energy (±5%) for incident photons from about 0.2 to 4 MeV. (The largest contributor to the gamma background at Bikini is ^{137}Cs with a gamma energy of 0.667 MeV.) The results, which include cosmic-ray background (∿4 μR/hr; ∿35 mR/yr), are given in the table below.

Measurements made through the interior of Bikini Island along the "center baseline" road indicated gamma exposure rates from 3 to 6 times as high as the village averages. A detailed survey of the interior is planned for early 1975.

	Gamma exposure rate ∿1 m above floor or ground, μR/hr		Annual gamma exposure, mR	
Location	Inside	Back yard	Inside	Back yard
House #4	7.8	13.2	68	116
House #20	8.0	7.7	70	67
House #28	9.3	15.5	81	136
House #40	9.3	15.5	81	136
Average			75	114