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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)

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

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THIRTEEN-YEAR SURVEY

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BENTLEY P. COLCOCK, M.D.,[4] AROBATI HICKING, PRACTITIONER,[5] AND MENASSA EMIL, PRACTITIONER[5]

with the technical assistance of

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SEBIO SHONIBER,[5] KOSANG MIZUTONI,[5] NELSON ZETKEIA,[5] WENTOLIN GOMEZ,[5] AND JUDE JOMULE[5]

FOURTEEN-YEAR SURVEY

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AROBATI HICKING, PRACTITIONER,[4] AND CHARLES F. DEMOISE, PH.D.[1]

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WILLIAM A. SCOTT,[1] DOUGLAS CLAREUS,[1] ERNEST LIBBY,[5] SEBIO SHONIBER[5], AND NELSON ZETKEIA[4]

FIFTEEN-YEAR SURVEY

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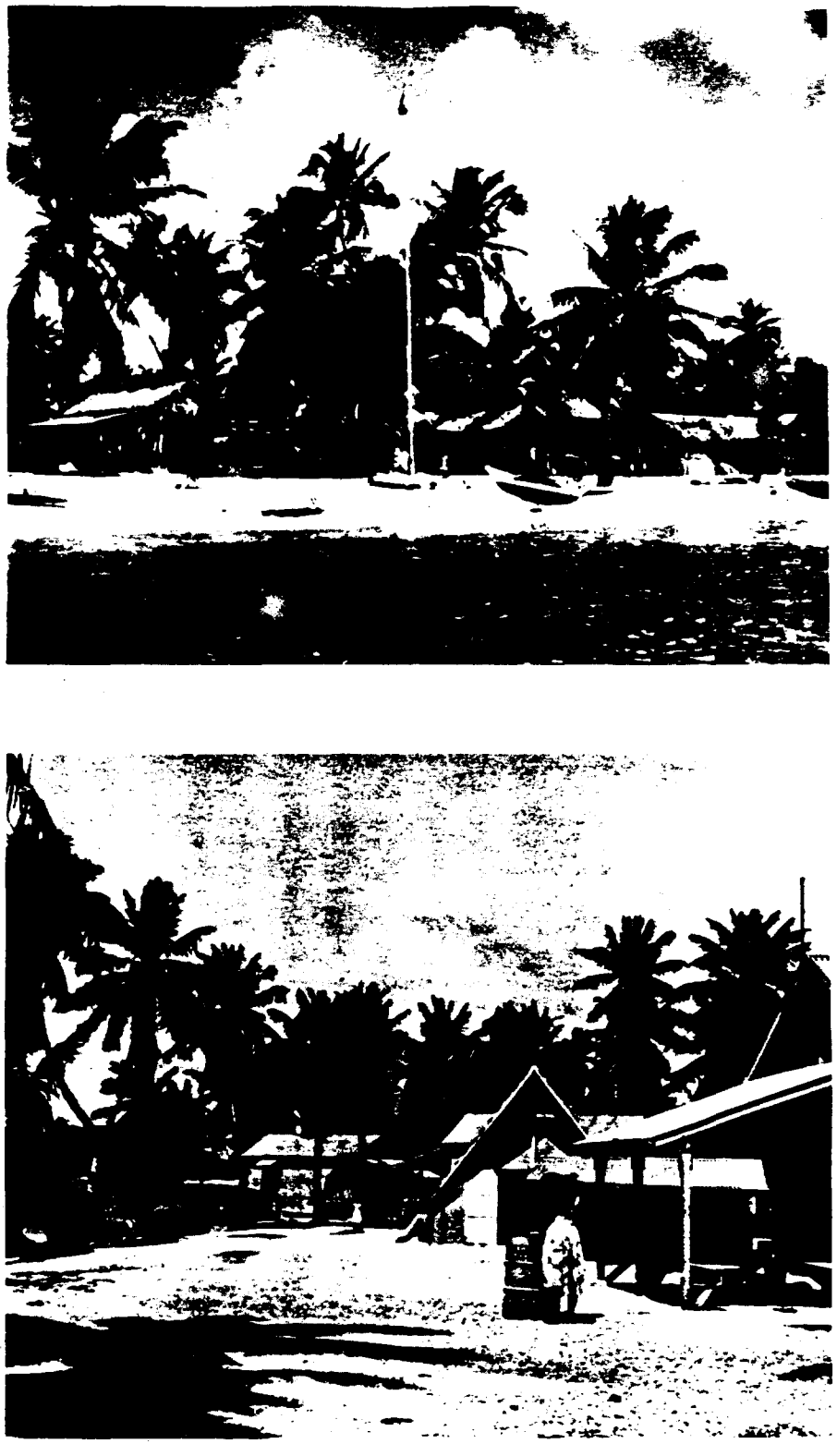


Rongelap Village in 1954 (above) and
after reconstruction in 1957.

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)

Introduction

This report presents in detail the results of medical surveys carried out during the past 3 years (March 1967, 1968, and 1969) on the people of Rongelap and Utirik Atolls who had been exposed to fallout radiation in March 1954. Earlier findings are also reviewed. The last detailed report (BNL 50029)[11] covered the surveys of March 1965 and 1966, 11 and 12 years after the accident. In view of the seriousness of recent findings regarding development of thyroid abnormalities, continuation of annual medical examinations and publication of detailed reports is considered important.

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

The exposed people were evacuated from these islands by plane and ship about 2 days after the accident and taken to Kwajalein Naval Base about 150 miles to the south, where they received extensive examinations for the following 3 months. During this period vigorous efforts were necessary to decontaminate the skin completely.

In view of the generally negative findings on the American servicemen they were later returned to their duty stations. The Utirik people were also allowed to return to their home island, where radioactive contamination was slight enough to allow safe habitation. Because Rongelap Atoll was considered to be too highly contaminated, a temporary village was constructed for the Rongelap people (including the 18 from Ailingae) on Majuro Atoll several hundred miles to the south, where they lived for the following 3½ years and were examined at yearly intervals by a special medical

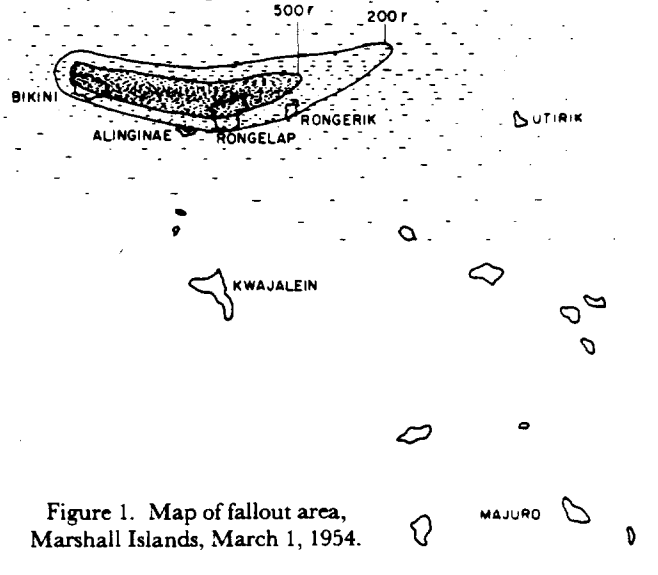


Figure 1. Map of fallout area, Marshall Islands, March 1, 1954.

Table 1

Summary of Fallout Effects

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

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

Table 2

Marshallese Populations Examined Since 1954

Group	Original number in group	Number living (1969)	Frequency of exams	Subject Nos.
Exposed				
Rongelap[a]	67	56	Annual	1–86
Ailingnae[b]	19	14	Annual	1–86
Utirik	157	127	3–4 years	2101–2257
Unexposed				
Rongelap	99	180[c]	Annual since 1957	801–1104 1500–1540
Rita	57		1955–1956	1000–1082
Majuro	115		1954 only	700–800
Children Conceived After the Fallout				
Of exposed parent(s)		89	Annual	87–181
Of unexposed parents		110	Annual	801–1104 1500–1540

[a]Includes 3 *in utero* children.
[b]Includes 1 *in utero* child.
[c]Individuals have been added since 1957 when this group was first available.

team. In July 1957, after careful evaluation of radioactive contamination, Rongelap Island was considered safe for habitation. A new village was constructed, and the Rongelap people were moved there by Navy ship. (See frontispiece.)

The annual surveys are carried out at Rongelap and also at Kwajalein and Majuro Atolls, where a number of Rongelap and Utirik people now reside. Examinations on Utirik Atoll are carried out about once every 3 years.

A group of more than 100 Rongelap people, who were relatives of the exposed people but had been away from the island at the time of the accident, moved back with the exposed people to their home island and have served as an ideal comparison population for the studies. The number has since increased to >200.

Since the accumulation of data from these surveys is becoming increasingly voluminous, survey reports published by this Laboratory are made as complete as possible and include a considerable amount of raw data, much of it in appendices, so that others may have access to complete data.

A summary of early and late findings covering the entire 15-year period is presented at the end of this report.

Reports have been published on the medical findings of surveys made at the following times after exposure: initial examination,[1] 6 months,[2] 1 year,[3] 2 years,[4] 3 years,[5] 4 years,[6] 5 and 6 years,[7] 7 years,[8] 8 years,[9] 9 and 10 years,[10] and 11 and 12 years.[11] A more complete list of reports, including outside publications, on the results of medical surveys of the Marshallese exposed to fallout, and including a section on some of the radiation ecological studies of these islands published largely by the University of Washington group, appears in BNL 50029.[11]

BACKGROUND

Several difficulties were encountered in carrying out the examinations. The language barrier was one, since very few of the Marshallese speak English, but usually some Marshallese were available who spoke enough English to serve as interpreters when necessary. The lack of vital statistics and demographic data on the Marshallese imposes serious limitations in interpretation and evaluation of the medical data. Trust Territory officials are attempting to improve registration of such data. The uncertainty on the part of many of the Marshallese as to their own ages is a problem, especially in the growth and development studies among the children and in the aging studies.

During the first 2 years two separate groups of Marshallese people were used for comparison purposes, but they were unstable, with a large attrition rate. At the time of the 3-year survey the Rongelap population at Majuro Atoll was found to have doubled during the preceding 12 months because of the influx of relatives who had come back from other islands to live with their own people. Those people had been away from Rongelap at the time of the accident and therefore were unexposed. The group matched reasonably well with the exposed group for age and sex. This group has gradually increased and is now more than twice the size of the exposed group. Table 2 shows the various Marshallese populations that have been examined since 1954.

ORGANIZATION OF THE 1967-69 SURVEYS

In 1967 and 1969 complete surveys were made of all of the comparison population as well as the exposed people. In 1968 only the exposed group was examined, in line with our policy of doing a complete survey only in alternate years. In 1969 the Utirik population was also examined, for the first time since 1966. The physicians and technicians participating in the different surveys are listed on the title page and are shown in Figure 2. These included, in addition to those from the United States, a sizable number of Micronesian medical personnel. The examinations were carried out at Majuro, Ebeye, and Rongelap, since a number of Roneglap people drift back and forth between these three islands, and at Utirik (see Table 3). The medical teams traveled to Kwajalein in the Marshalls by commercial plane and from there to the various islands by Micronesian cargo ship (Figure 3). Most of the supplies and medical equipment had to be carried with the team except for a considerable amount of permanent equipment, including two examination trailers, established at Rongelap. Table 4 shows the numbers of people examined during the 1967-1969 surveys.

Findings

GENERAL MEDICAL STATUS

During the past 3 years some improvement has been noted in the general health of the Rongelap people. This may be partly due to general improvement in sanitation and hygenic measures. Garbage disposal appears to have reduced the fly population, and children are using the outhouses rather than the beach for defecation. The people have more money to purchase such items as kerosene refrigerators, automatic washing machines, and new plastic water cisterns. Gastronenteritis and respiratory infections were still fairly frequent, but skin lesions due to fungus and impetigo appeared somewhat reduced. Fish poisoning still occurs sporadically among families who happen to eat a poisonous fish and will probably continue to do so, since the poisonous fish are very difficult to identify and may shift from one species to another at times. A rather serious epidemic of Hong Kong influenza occurred among the Rongelap people in 1968 and may have been responsible for the deaths of a 58-year-old exposed woman and of an unexposed boy who died of meningitis complicating the influenza. In June 1968 a few cases of infectious hepatitis occurred, and also a mild measles outbreak among the younger children. In August 1968 four patients were brought

Figure 2. Medical teams: upper, 1967; middle, 1968; lower, 1969.

Table 3

Present Known Location of Rongelap and Utirik People Under Study

	Rongelap and Ailingnae exposed*			Rongelap unexposed		Utirik exposed		
	Adults (age >19)	Children (age 15–19)	Children of exposed parent(s)	Adults (age >19)	Children (age <19)	Adults (age >19)	Children (age 15–19)	Total
Majuro	3	3	6	29	13	12	9	75
Ebeye	19	8	43	40	52	15	5	182
Rongelap	26	4	37	47	70	0	0	184
Utirik	0	0	0	0	0	68	12	80
Other atolls	5	2	3	19	20	5	1	54
Total	53	17	89	135	155	100	27	575

*Includes 4 children exposed *in utero*.

Table 4

Rongelap and Utirik Populations Examined,* 1967–1969

	Rongelap			Ailingnae			Utirik	Rongelap unexposed	
Age	1967	1968	1969	1967	1968	1969	1969	1967	1969
13, 15–20	16	12	13	4	3	2	21	32	24
21–30	9	11	12	3	3	4	23	15	14
31–40	9	10	9	–	–	–	9	23	24
41–50	7	4	6	1	1	1	14	14	15
51–60	5	6	7	3	3	2	15	7	8
61–70	3	3	3	1	1	1	4	13	7
>70	1	1	2	–	–	–	1	6	7

	Children of Rongelap exposed parent(s)			Children of Rongelap unexposed parents	
	1967	1968	1969	1967	1969
<13, 15	68	4	81	70	94

*A few cases did not have complete examinations.

Figure 3. Trust Territory cargo ship used in transporting medical team at anchor in Rongelap lagoon.

Figure 4. Marshallese children watching Western movie at Rongelap.

Figure 5. Marshallese technique of "herding" fish ashore. Fish are being caught for annual feast during medical survey.

Table 5

Mortality, 1954–1969, by Age as of 195[illegible]

Group	Age:	<10	11–20	21–30	31–40	41–50	51–60	61–70	71–80	>80	Total
Rongelap (175 rads)		0/19*	1/13	0/10	0/5	2/7	3/5	1/1	3/3	1/1	11/64
Ailingnae (69 rads)		0/6	0/1	0/1	1/5	1/1	0/1	3/3	—	—	5/18
Utirik (14 rads)		1/56	0/20	3/15	1/20	5/18	9/13	8/11	3/4	—	30/157
Unexposed**		0/56	1/28	0/30	2/18	4/21	3/17	4/17	1/1	1/1	16/189

*Mortality/number in group.
**This group was not examined until 1957 and includes occasional new subjects examined through 1966 (Subject 1511).

Table 6

Adult Mortality

Exposed				Unexposed			
Year	Subject No.	Age & sex	Probable cause	Year	Subject No.	Age & sex	Probable cause
1956	24	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

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

to the United States for extensive thyroid studies at Brookhaven National Laboratory and later surgery in Boston. In 1969 five further cases were brought over for thyroid studies and surgery in Cleveland.

The deaths during the past 3 years are included in Tables 5 and 6. Three exposed people died. The 58-year-old woman (#59) referred to above, who died of pneumonia complicating influenza, had had a partial thyroidectomy for benign thyroid adenoma the previous year. Another death, that of a 71-year-old woman (#13), was thought to be due to cancer of the uterus, and an 88-year-old man (#55) died of cardiovascular disease with heart failure. Among the unexposed population three older men died, one with diabetes (#853), one with heart failure (#860) and one from unknown cause (#964). An older woman (#936) died of complications of diabetes. A 24-year-old male (#967) died in an auto accident. The boy mentioned above died of complications of influenza. During the past 15 years, 16 deaths have occurred among the exposed Rongelap people. This represents 13.0 deaths per 1000 persons per annum compared with 8.3 per 1000 for the Marshall Islands as a whole (1960).

Table 7 lists the births and miscarriages during the last 3 years for both populations. The 33 births during the 3-year period in the exposed group, compared with 30 in the unexposed group, are in line with the birth rate in previous years and show no impairment of fertility in the exposed women.

Table 7

Births and Fetal Deaths[a] by Year

Year	Women aged 15–45	Total pregnancies	Live births	Children		Miscarriages	% Pregnancies terminating in miscarriage
				M	F		
			Exposed[b]				
1954[c]	19	1	0	0	0	1	100
1955	20	6	5	4	1	1	17
1956	20	6	4	0	4	2	33
1957	21	5	2	2	0	3	60
1958	22	14	8	4	4	6	43
1959	22	6	5	2	3	1	17
1960	24	10	9	5	4	1	10
1961	23	7	6	2	4	1	0
1962	24	4	4[d]	1	3	1	25
1963	27	8	7	3	4	1	12
1964	26	6	6	1	5	0	0
1965	30	6	6	3	3	0	0
1966	30	10	10	7	3	0	0
1967	30	9	8	3	5	1	11
1968	33	14	12[d]	10	2	3	21
			Unexposed				
1956	29	9	7	6	1	2	22
1957	30	11	9	4	5	2	18
1958	30	9	8	5	3	1	11
1959	29	10	9	4	5	1	10
1960	29	10	8	5	3	2	20
1961	29	10	10	9	1	0	0
1962	30	6	5	4	1	1	17
1963	32	6	5	2	3	1	17
1964	32	13	11	8	3	2	15
1965	32	10	8	6	2	2	20
1966	32	7	6	4	2	1	14
1967	38	9	8	4	4	1[d]	11
1968	46	14	13	9	4	1	7

[a]Includes stillbirths and neonatal deaths.
[b]Includes nonexposed females married to exposed males.
[c]Includes only children conceived after March 1, 1954.
[d]Includes twins.

The occurrence of 4 miscarriages or stillbirths in each of the two groups also conforms with the incidence in recent years, the exposed women showing no greater incidence than the unexposed women.

Utirik

Vital statistics over the 3-year period for the population at Utirik were not reliable since the Health Aide had left the island the previous year and taken the records. Apparently no unusual epidemics occurred among the island people, and they escaped the Hong Kong flu. At least 6 deaths occurred during the past 3 years, but the causes could not be definitely ascertained. Of the original 157 people on Utirik at the time of exposure, 127 are now living. In addition some 100 Utirik people live on the island who were not exposed to fallout but moved back later. Quite a few Utirik people now live on Ebeye and Majuro (see Table 3). Seven births were reported for the 3 years but probably more had occurred. The general health status of the Utirik people and the sanitary conditions of the island appeared to be about the same as 3 years before.

PHYSICAL EXAMINATIONS – CHILDREN

Pediatric examinations were conducted on the Marshallese subjects under the chronological age of 20 years with the exception of adolescent girls who were pregnant or who had had babies. The

Figure 6. Children participating in games at time of feast.

studies consisted of a brief interval history, routine physical examination, roentgenograms of the left hand and wrist, and body measurements. In 1967 and 1969, all children in the exposed and control categories as well as children born to exposed and unexposed parents were examined. In 1968, only the children in the Rongelap and Ailingnae exposed groups were seen. In 1969, Utirik exposed persons under 20 years of age were included. During these examinations, special attention was directed to the palpatory findings in the thyroid glands.

The significant findings for each of the three years are summarized in Table 8. Thyroid-related abnormalities are discussed in a subsequent section of this report. The pattern and intensity of the recorded abnormalities reflected primarily the expected fluctuations related to such factors as age, weather, habits, activities, community health problems, and normal developmental variations. With the exception of thyroid lesions and growth retardation, no correlation between pediatric findings and exposure to fallout radiation could be suggested.

The data on physical growth of children (measured primarily in terms of stature and body weight) examined in 1965, 1966, 1967, 1968, and 1969 are tabulated in Appendix 1. Growth data from prior examinations have already been published.[10] Statistical analyses have been done on the data obtained through 1967. The results showed no marked deviations from the trends noted previously.[12-14] Comparison of median statures and weights by age showed no statistically significant differences between the exposed and control groups for either boys (Figures 7 and 8) or girls (Figures 9 and 10). However, separate analysis of the group of children under 6 years of age at time of exposure to fallout radiation shows a significant retardation in statural growth persisting among the boys (Figure 11) but not among the girls (Figure 12). The growth curves for stature and body weight of children born to exposed parent(s) showed no significant difference from those of children born to unexposed parents (Figures 13 and 14).

The analysis of skeletal age data through 1967 also showed no statistically significant difference between the exposed and control groups of children (Figures 15 and 16). Skeletal ages were estimated from roentgenograms of the left hand and wrist by the technique of Greulich and Pyle.[15]

Table 8

Physical Findings, Pediatric Examination
(See Table 13 for thyroid abnormalities)

	Exposed				Control				Nonexposed, born of exposed parent(s)	
	Rongelap			Utirik	Born before 1 Jan 1955		Born after 1 Jan 1955			
	1967	1968	1969	1969	1967	1969	1967	1969	1967	1969
Number examined	19	15	16	25	29	18	66	82	67	77
Blood pressures taken	16	–	15	25	27	18	36	43	30	39
Hypertension	1	–	–	–	–	–	–	–	–	–
Keloids	1	–	–	1	–	–	–	–	–	–
Active skin infection	1	–	3	1	3	–	11	25	13	22
Molluscum	1	–	–	–	–	–	3	–	9	4
Tinea versicolor	2	1	3	1	4	2	4	1	–	3
Vitiligo	1	1	–	–	1	–	–	1	–	1
Warts	4	1	–	–	1	–	–	3	4	5
Papilloma	–	–	1	–	–	–	–	–	–	–
Café-au-lait spots	–	1	–	–	–	–	–	2	–	–
Folliculosis	–	–	–	–	–	–	–	–	1	1
Otitis media	6	2	–	3	9	5	9	3	8	5
Respiratory infection	1	–	1	–	7	3	11	7	16	8
Hypertrophic tonsils	–	1	2	7	4	6	7	20	4	17
Dental caries	2	–	3	3	4	2	16	20	21	19
Cheilosis	–	–	–	–	1	–	1	1	1	4
Conjunctivitis	–	–	–	–	–	1	1	–	–	–
Thrush	–	–	–	–	–	–	–	–	1	–
Chickenpox	–	–	–	–	–	–	–	1	–	–
Adenopathy	1	–	2	–	–	3	6	7	5	8
Palpable liver	–	–	–	–	–	–	1	2	6	2
Palpable spleen	–	–	–	–	–	–	–	1	3	–
Umbilical hernia	–	–	–	–	–	–	2	6	–	1
Systolic murmur	6	6	3	3	16	6	43	34	34	26
Anisocoria	–	–	–	–	1	–	–	–	–	–
Pes excavatus	–	–	–	–	1	–	–	–	–	–
Wrist deformity	–	–	1	–	–	–	–	–	1	–
Club foot	–	–	–	–	–	–	–	–	–	1
Dwarfism	–	–	–	–	–	–	–	–	1	–
Polio residual	–	–	–	–	–	–	–	–	1	1

The lines on Figures 15 and 16 represent the best fitting linear relationships plotted by the least-squares method. The points representing the markedly growth-retarded boys and showing their early response to thyroid hormone administration can be recognized by comparison with Figures 31 to 33. The graphs continue to indicate that in the Marshallese children the skeletal age values at comparable chronological ages are lower (by about 6 to 12 months) than the norms for American children presented by Greulich and Pyle.[15]

The interpretation of growth data from the exposed Rongelap children has been complicated by partial or total thyroidectomies in those children developing nodules (see Thyroid Findings, below) and by the administration of thyroid hormone to the whole exposed Rongelap population since September 1965. The response of certain individual hypothyroid children to thyroid hormone is readily seen in the growth curves (Figures 31 and 32). The increase in yearly growth rate in males exposed at <5 years of age is significantly greater after thyroid treatment ($p<0.05$) than in unexposed males of comparable age during the same period (based on an analysis of variance using weighted squares of means of yearly growth increments).

In order to probe the feasibility of conducting psychometric tests, the IPAT (Institute of Personality and Ability Testing) Culture Fair Intelligence Test, Scale 2, Form A[16] was given in 1969 to a group of Marshallese subjects on Rongelap Island and to another group on Ebeye Island. This particular test is designed to be comparatively immune to "contamination by accidents of social, educational, and racial background,"[17] and has been used before in several groups of Micronesians.[18]

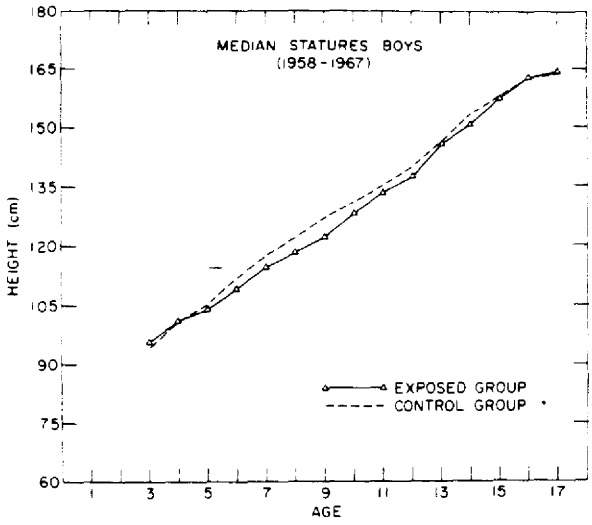
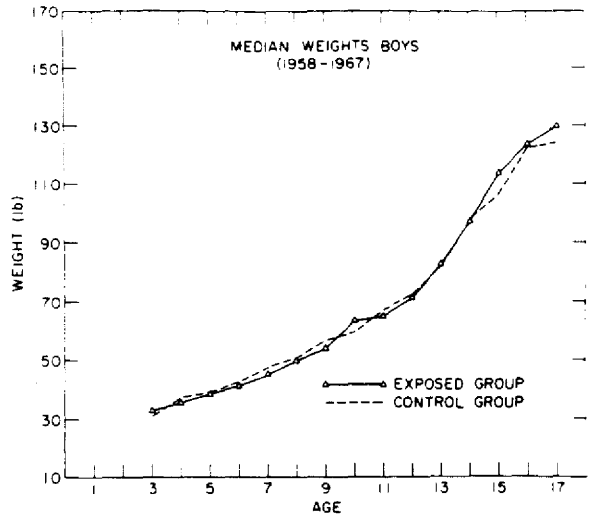


Figure 7.



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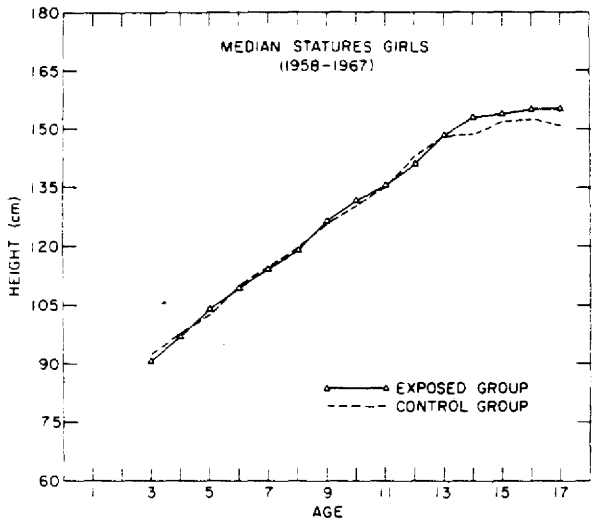

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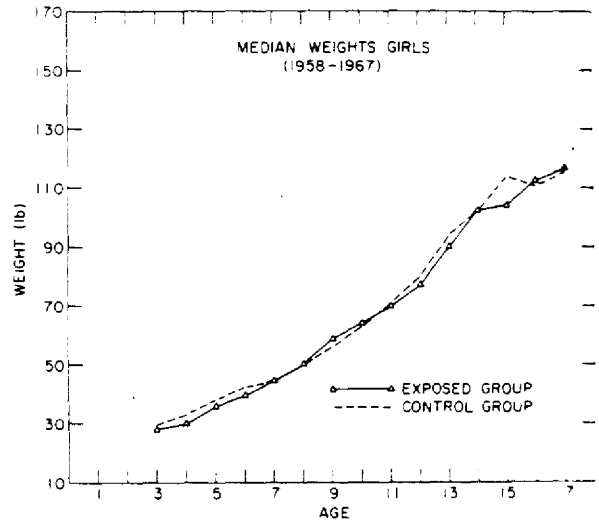
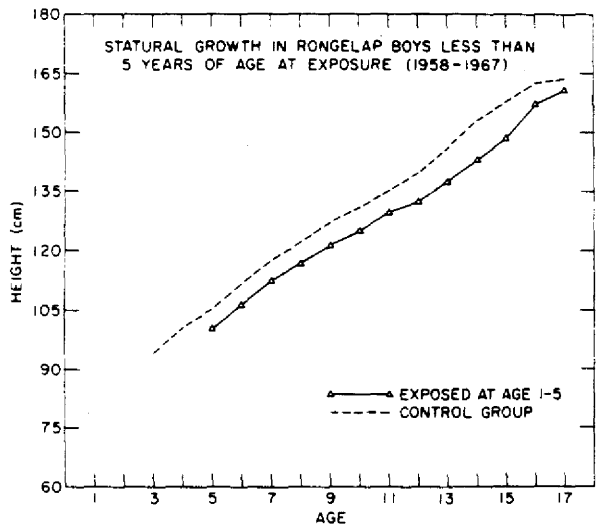


Figure 10.



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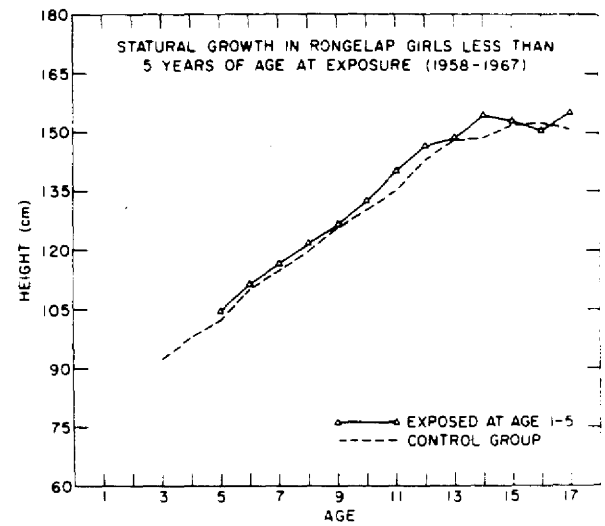

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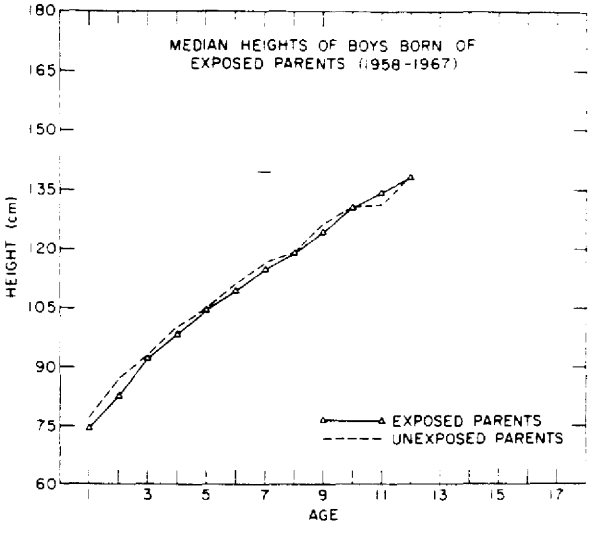

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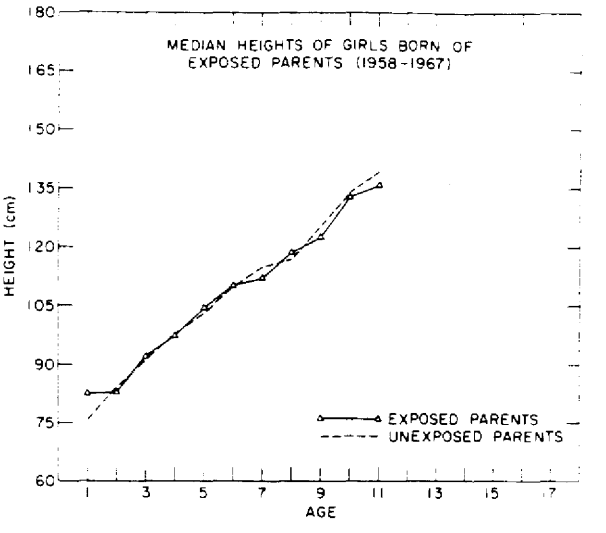

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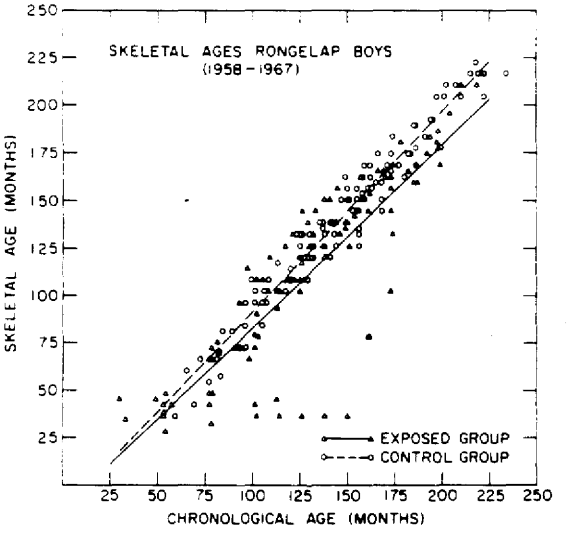

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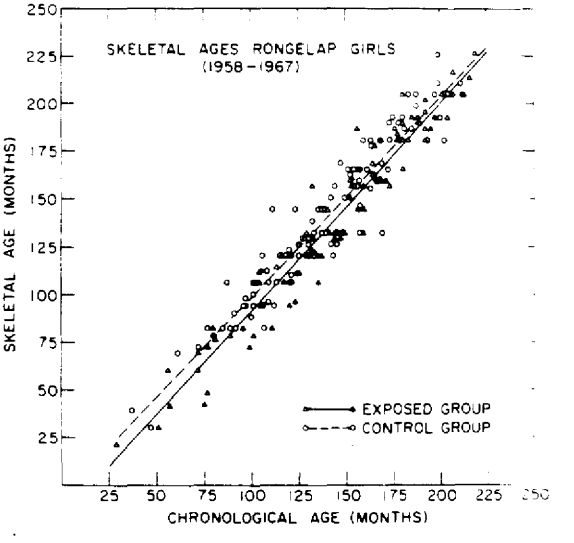


Figure 16.

The procedures were discussed beforehand with the Peace Corps personnel supervising educational activities on each island, and they explained the techniques and objectives to Marshallese teachers who actually conducted the tests. The tests were given in the regular classrooms, and prescribed time limits were used.

The test results are recorded as raw scores in Appendix 2. The small numbers precluded any statistical analyses of these results, but it is apparent that the Ebeye group consistently scored considerably higher than the Rongelap group and that the scores for both groups fell far below those recorded as "norms" in the manual. Observations during the testing indicated that, at Rongelap, neither the Marshallese teacher nor the subjects were very familiar with written examinations. Motivation was poor and understanding of the tests seemed marginal. The situation seemed better at Ebeye (Kwajalein Atoll), where more formal school classes are held. The exigencies of a field operation precluded improvement of test conditions that would have permitted a more leisurely and better understood examination. Under the circumstances, no quantitative interpretation of these test results seems justifiable.

Table 9

Physical Findings in Rongelap and Utirik Adults, 1967-69

	Rongelap exposed (49 examined)		Rongelap unexposed (77 examined)		Utirik exposed (71 examined)	
	Subject Nos.	%	Subject Nos.	%	Subject Nos.	%
Anemia	11, 70	4.1	835, 843, 865	3.9	2254	1.4
Aphakia	58, 68, 80	6.1				
Arteriosclerosis (mild)	4, 7, 41, 58, 63, 78, 79	14.2	884, 1515	2.6	2114, 2139, 2140, 2161, 2200, 2212, 2221, 2224	11.3
Arteriosclerosis (moderate to severe)	1, 11, 16, 60, 68, 80, 82	14.2	856, 859, 878, 889, 908, 915, 929, 947, 961, 975	13.0	2110, 2169, 2211, 2244, 2258	7.1
Asthma			844, 916	2.6	2200	1.4
Atrial fibrillation	80	2.0	858, 948	2.6		
Bradycardia	27 (with hypertension) 80 (with atrial fibrillation)	4.1				
Cardiac enlargement	60, 76	4.1	858, 859, 917, 947, 948		7.8	
Cervical erosion or laceration	1, 12, 15, 24, 39, 45, 49, 58, 61, 63, 64, 71, 72, 74, 78	30.6	823, 829, 832, 846, 867, 914, 916, 932, 938, 956, 959, 1001, 1505, 1520	18.1	2139, 2149, 2162, 2194, 2208, 2246	8.5
Cystourethro-rectocele	1, 14, 18, 34, 45, 49, 63, 64, 78	18.4	852	1.3		
Deafness	1, 60	4.1	858, 878, 884	3.9		
Diabetes mellitus	27?, 41	4.1	852, 855, 898, 915, 918, 956, 1041, 1042	10.4		
Epilepsy			875	1.3		
Hernia			948, 1517	2.6	2109, 2169	2.8
Hypertension (>140/90)	1, 4, 11, 58, 60, 77	12.2	856, 859, 885, 898, 899, 908, 947, 948, 982	11.7	2128, 2140, 2158, 2169, 2193, 2194, 2200, 2208, 2215, 2216, 2221, 2252, 2258	18.3
Leprosy, arrested	77	2.0				
Obesity	1, 49, 60, 61, 64, 67, 71, 74, 78	18.4	849, 815, 859, 880, 881, 898, 934, 942, 943, 951, 959, 970, 982, 1005, 1050	19.4	2107, 2128, 2158, 2189, 2195, 2196, 2215, 2224, 2246	12.7
Osteoarthritis	60, 79	4.1	858, 859, 878, 884, 896, 898, 915, 922, 928, 935, 947, 961	15.6	2161, 2169	2.8
Primary fibrous dysplasia	34, 58, 63, 64, 78	10.2				
Prostatic hypertrophy	68, 82	4.1	915	1.3	2211	1.4
Proteinuria (>100 mg)			855, 1526	2.6		
Rheumatic heart disease	76	2.0				
Rheumatoid arthritis	68	2.0				
Spermatocele	4	2.0				
Syphilis(?), arrested	11, 59	4.1	846, 859, 864, 880	5.2		
Tumor benign (except thyroid)	11 (knee) 14 (epidermoid cyst) 63 (giant tumor, finger) 70 (neurofibroma, neck)	8.2			2140 (r. ankle) 2208 (nose, abdomen) 2212 (forearm) 2252 (chest)	5.6
Tumor malignant	13 (uterus)	2.0				
U.R.I.	24, 39, 40, 74	8.2				
Varicocele	40, 41, 76	6.1	918, 948	2.6		

PHYSICAL EXAMINATIONS - ADULTS

The positive clinical findings in adult Rongelap and Utirik people are listed in Table 9. The general health status and incidence of physical abnormalities of the exposed Rongelap people was about the same as of the unexposed people on the island except for thyroid abnormalities, which are described separately below. The varying frequencies reported from year to year on such abnormalities as prostatic hypertrophy, cervicitis, and arteriosclerosis probably reflect to some extent differences in clinical evaluation among the examining physicians. During the past 3 years, skin lesions appeared to be less prevalent, perhaps because of better hygienic conditions (the importance of daily baths with soap and water has been stressed). Special ophthalmological examinations were carried out in 1967 and slit-lamp observations for enumeration of subcapsular flecks of the lens in 1969. These findings are described separately below.

As part of the cancer survey, x rays of the chest were taken every 2 years (half the group in alternate years) on both exposed and unexposed Rongelap people. The roentenograms showed about equal incidence of cardiovascular findings in the two groups. Two cases with inactive tuberculous pulmonary lesions were noted, but no evidence of primary or metastatic pulmonary malignant lesions was seen. Papanicolaou smears were taken from the vaginal region of all women examined. Evidence of inflammatory reactions and infections with trichomonas and bacteria were common. Only one smear in an unexposed woman was suggestive of cancer of the uterus. The medical authorities in the Marshall Islands were notified of this case. Except for cancer of the thyroid (described below) only one case of cancer was noted during the 3 years, in a 71-year-old exposed woman (#13) who died with bleeding from the genital tract thought to be due to cancer of the uterus. In 1968 one 34-year-old exposed Ailinginae woman (#70) was found to have a hard non-tender tumor several centimeters in diameter lateral to the lower left lobe of the thyroid. She was brought to the United States with several other Rongelap people who were to undergo thyroid surgery. Surgical removal of this tumor revealed a benign neurofibroma. A number of other benign soft-tissue tumors were noted during the examinations.

Examination of residual "beta burns," present in about 20 cases, showed only minimal scarring, atrophy, and pigment changes. The lesions have exhibited little or no change during the past several years. No further increase has been seen recently in benign moles which were noted several years ago in areas of the neck that had sustained "beta burns." No evidence of any malignant skin changes have been seen.

OPHTHALMOLOGICAL FINDINGS, 1967 SURVEY

Ophthalmological examinations were carried out* in 1967 on 63 exposed people, 62 children of exposed parents, and 185 people in the comparison population, a total of 310 (see Table 10).

As noted in previous surveys, as compared with American populations there was an increased incidence of large corneas and enlarged, tortuous, and bizzare-patterned retinal vessels, and a lower incidence of myopia, strabismus, amblyopia esophoria, retinitis pigmentosa, and congenital glaucoma.

The incidence of arcus senilis is higher in the Marshallese than in similar age groups in the United States, which is in keeping with the general observation that the Marshallese age faster than Caucasians. The incidence was higher in the exposed group (30%) than in the unexposed group (13%); however, on the basis of previous studies, the difference is probably not significant.

Although diabetes mellitus has a moderately high incidence in the Marshallese, only one case of diabetic retinopathy was noted. This is in keeping with the observation that diabetes in the Marshallese occurs largely in older individuals. The incidence of pinguecula and pterygium is high in the Marshall Islands. The incidence of pterygium was slightly higher in the exposed group.

Abnormalities of the crystalline lens, characterized below, are greater in the Marshall Islanders than in similar age groups in the United States. The abnormalities observed consist of polychromatic sheen changes, lenticular opacities of all degrees, and cataracts. The polychromatic sheen varied from a few fine granules to large granular plaques situated on the posterior lens capsule in the zone of specular reflection. In minimal cases the granules were yellowish and in some cases appeared slightly darker with a "beaten brass" color. Where the granules had coalesced into a

*By Dr. Austin Lowrey, a member of the 1967 survey team.

Table 10

Ophthalmological Survey, 1967

	Exposed (63)*		Children of exposed (62)		Unexposed (185)	
	No.	%	No.	%	No.	%
Anisocoria	1	1.5	2	3.2	1	0.54
Arcus senilis	20	30			25	13.5
Argyll Robertson pupil	1	1.5				
Chalazion			1	1.6		
Choroiditis (old, healed)	1	1.5			5	2.7
Conjunctivitis	4	6.0	1	1.6	6	3.2
Corneal scars	3	4.5	1	1.6	1	0.54
Corneal pigment	1	1.5				
Drüsen	1	1.5			2	1.08
Lens:						
Polychromatic sheen						
Right eye	11	16.5			22	11.88
Left eye	10	15			21	11.34
Lenticular opacities						
Right eye	8	12			18	9.6
Left eye	7	10.5			16	8.56
Cataracts, senile						
Right eye	5	7.5			17	9.8
Left eye	4	6			14	7.56
Aphakia·						
Right eye	2	3			4	2.16
Left eye	2	3			1	0.54
Leprosy, eye signs of	1	1.5				
Macular degeneration	7	8.2			3	1.62
Nystagmus			1	1.6	1	0.54
Pinguecula						
Right eye	5	7.5			13	7.2
Left eye	4	6.0			13	7.2
Pterygium						
Right eye	13	19.5			33	17.82
Left eye	13	19.5			28	15.12
Proptosis	1	1.5			2	1.08
Phthisis bulbi					1	0.54
Positive Rhomberg	1	1.5				
Retinal arteriosclerosis	9	13.5			18	9.72
Retinal scars	1	1.5			3	1.67
Retinal hemorrhages	1	1.5			2	1.08
Retinopathy, diabetic					1	0.54
Ocular muscle imbalance						
Phorias						
Esophorea						
Exophorea	2	3	1	1.6	8	4.32
Tropias						
Esotropia						
Exotropia	1	1.5	1	1.6	1	0.54
Uveitis					2	1.08
Vitreous opacities	11	16.5			23	12.42
Difficulty with night vision					2	1.08

*Number of people examined in each group.

plaque, greenish and bluish hues were noted, hence the name polychromatic sheen. These lens opacities are not comparable to the subcapsular flecks of the lens recorded in the 1969 survey and described below.

Whether the polychromatic sheen seen following irradiation has a unique and specific character is still a debatable question. Some investigators contend that similar appearing changes can be detected in patients with retinitis pigmentosa and the early stages of cataracts, which might be a complication of endogenous ocular systemic disease. Such polychromatic sheens were seen in 10% of the unirradiated Rongelap group and 16% of the exposed group. Again, the difference is thought to be too small to reflect irradiation exposure with any degree of certainty, particularly in view of the slightly greater number of older people in the exposed group. The incidence of lenticular opacities was also slightly greater in the exposed group than in the unexposed group.

Corneal pigmentation, noted in previous examinations, was noted in only one case in the 1967 survey. This pigmentation was characterized by a fine, dark, linear sheet of pigment lying close to or on Bowman's membrane in the horizontal axis, between the limbus and pupillary edge. Possibly these changes may have been iduced by beta radiation from contaminated material which collected on the margins of the eyelids at the time of the fallout.

Several findings may be residuals of the 1963 poliomyelitis epidemic. There were two cases of 7th nerve weakness involving the eyelids and an increase in the number of cases of esophoria and esotropia (ocular muscle imbalances) in post-poliomyelitis cases.

In the 1967 survey it was gratifying to find only one possible case of poor night vision attributable to inadequate vitamin A intake. This indicated improved nutrition with regard to vitamin A (yellow fruits and vegetables: squash, papaya, bananas, pandanus, yams, etc.).

RESULTS OF SLIT-LAMP MICROSCOPIC EXAMINATIONS,* 1969 SURVEY**

Study of the Marshallese persons exposed to radioactive fallout in 1954 for effects within the optic lens is of particular interest because (1) a broad span of ages was involved, and (2) the highest gamma exposure was slightly less than the lowest (radiotherapeutic) exposure at which Merriam found an effect on the lens.[19] The 1967 lens examinations of the Marshallese had shown the incidence of polychromatic sheen and lenticular opacities to be slightly higher in the exposed than in the nonexposed population. The differences were not thought to be significant, and the higher mean age of those exposed was pointed out (see preceding section).

The subjective nature of observations of the lens usually made with the slit-lamp microscope argues for serial examinations with increasing time post irradiation, because slight differences between groups in one examination may become significant with multiple examinations. A further obvious value to serial examinations is that of following the time course of lens changes within and between groups. (In a nonirradiated population, an approximate time course of lens changes may be derived by a single examination of individuals covering a broad span of ages.) Assuming an influence of age, the status of an irradiated lens will depend on three primary factors: (1) the radiation characteristics (dose, quality, and dose rate), (2) the age of the individual at exposure, and (3) the interval between irradiation and examination. For a radiation accident involving a single (relatively brief) exposure, a subsequent single examination can provide only one point on the time course of lens change at each age involved.

The Marshallese experience provided an adequate number of control individuals covering a broad span of ages, a lesser number of individuals exposed to 175 rads, and insufficient numbers for meaningful comparison at lower exposure groups (doses of [illegible] rads). Conclusions from this examination must therefore be limited to status of the nonirradiated lens as a function of age, and age-specific comparisons between [illegible] these people and those exposed to 175 rads 15 years previously.

It was intended during the 1969 medical survey to determine quantitatively the minute discrete opacities in the anterior and posterior subcapsular regions, which are similar to those that can be found in the posterior subcapsular region in the lens of the mouse (see Figure 17), and which may represent short defective portions of single lens fibers (see Figure 18). These defects are present in

*By Dr. John L. Bateman, a member of the survey team.

**Research supported in part by Grant RH99, Division of Radiological Health, Bureau of State Services, U.S. Public Health Service.

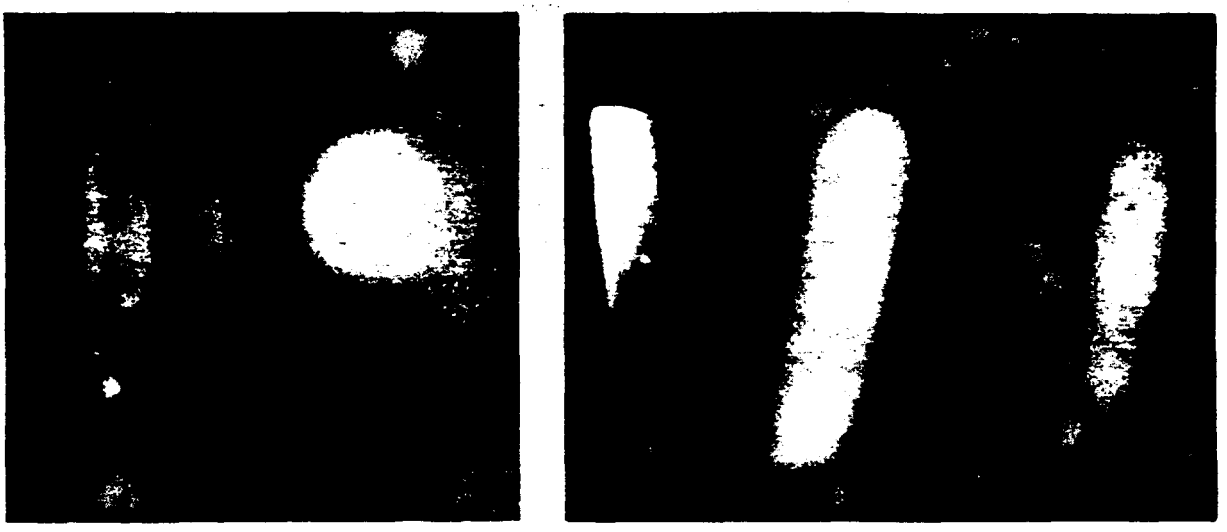


Figure 17. Photographs of the optic lens taken through the slit-lamp microscope at 312 magnification and enlarged 2⅓ ×. Lens flecks are visible in and to the right of the light arc which lies at the left in each photograph, created by impingement of the lamp beam on the lens posterior capsule. Left: Mouse, nonirradiated, one year old. Right: Human, a 39-year-old Marshall Island female exposed to 175 rads of fallout radiation 15 years previously.

nonexposed animals (increasing with age) but have been found in significantly greater numbers in mice exposed to even very low doses of radiation.[20]

Methods

Slit-lamp microscopic examination after iris dilatation with tropicamide 0.5% (Mydriacyl) was made of people within the several exposure groups as follows: 175 rads (midline gamma dose in air), 41 persons; 70 rads, 10 persons; 14 rads, 15 persons; nonexposed, 80 persons. The observer was unaware of the group to which each individual belonged until after the examinations. First a brief general inspection (at a magnification of 200 or 312 ×) was made of the cornea, aqueous humor, lens, and anterior vitreous humor. Representative photographs were taken, usually at 312 ×. Finally counts were made of minute discrete opacities (flecks) falling within the 2.5-mm-diameter circle projected into the viewed field by an ocular reticle. Counts were made within the anterior and posterior subcapsular regions of each lens at a location about one-half lens radius below the midline. This location was selected to minimize the corneal light reflex as the narrow slit-lamp beam was swept across the counting region. Axial traverse of the focus during counting gave depth to the counting area, effectively creating a cylinder of 2.5-mm diameter and extending between the lens capsule and lens nucleus.

Eight examinations were unsatisfactory because of advanced age cataracts, lens extractions, or corneal defects.

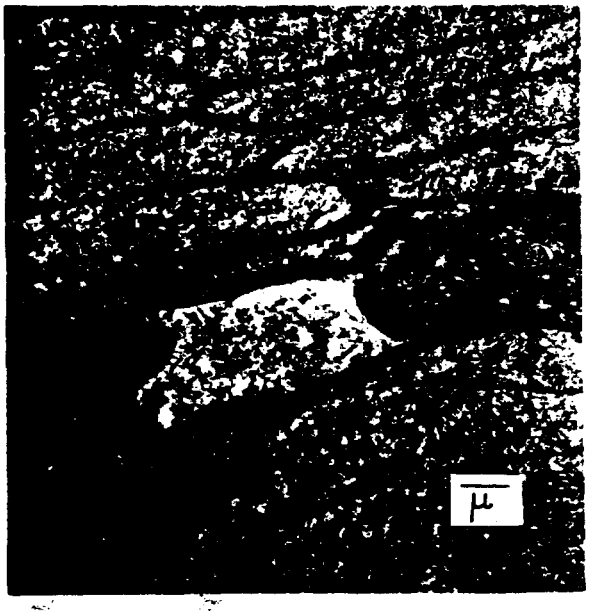


Figure 18. Electron micrograph of optic lens fibers of a 21-week-old mouse exposed to 3000 rads of ^{60}Co gamma rays at 6 weeks of age. The fibers are seen in cross section, and a defective fiber lies near the center of the field. (From H.A. Johnson, *Acta Iberica Radiol. Cancerol.* 19: 247-54, 1964).

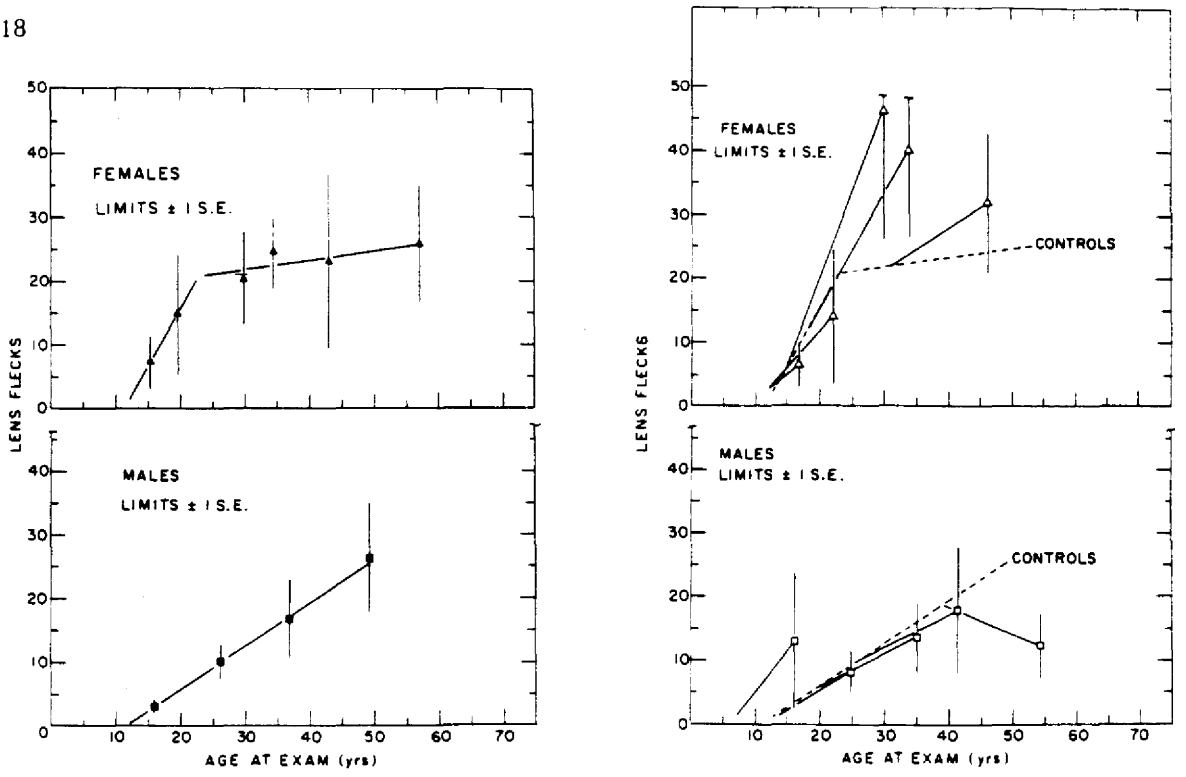


Figure 19. Counts of subcapsular flecks (made as described in the text) averaged within each exposure group for persons of similar age. Left: Unirradiated Marshallese people. Right: People exposed to 175 rads fallout radiation in 1954.

Results

The fleck counts were analyzed separately for males and females, and, within each exposure group, were averaged for consecutive intervals of age to produce age-specific subgroups. The average fleck count and statistical limits extending one standard error to either side were then computed for each subgroup. (Persons over 61 years of age were omitted.) The results are shown in figure 19 for the controls and for the irradiated individuals. Each subgroup of the latter is connected by a solid line to its presumed location on the (broken) control curve at irradiation 15 years prior to this examination. The individual fleck counts are listed in Appendix 3.

In the nonexposed females the number of lens flecks rose rapidly during adolescence but at a much slower rate thereafter. Females aged 13 to 20 years at exposure exhibited the greatest difference from their nonexposed counterparts. (Fleck counts were not obtained on persons <15 years of age, but would likely be lower than in persons >15 years of age.) Females who were mature at exposure also yielded fleck counts higher than those of comparably aged nonexposed females, but the difference was less. (Two fleck counts >150 were excluded from analysis.)

Nonexposed males had fleck counts that increased with age, but in contrast to that for nonexposed females, the rise was slower and appeared to be linear with time. Males exposed to 175 rads were the smallest group, which may contribute to the erratic fleck counts obtained.

The levels of confidence were generally low because of the nature of the examination and the numbers of persons examined. Confidence at the 95% level was found only between nonexposed males averaging 16 years of age and (1) the oldest nonexposed male subgroup, and (2) 175-rad exposed females older than 25 years at examination. Although some fine structure has been implied, the results for each group can be represented by a straight line.

Discussion

The primary finding in this survey was the continuous increase in lens flecks with age in the nonexposed males and females. Whereas the increase for females was more rapid during adolescence, lens flecks in males rose at a lower but constant rate; consequently, fleck counts in males did not equal

Table 11

Estimated Body Burdens of Radioisotopes in Rongelap People on Day 1 From Radiochemical Urine Analyses

Isotope	Activity, μCi
^{89}Sr	1.6 - 2.2
^{140}Ba	0.34- 2.7
Rare earth group	0 - 1.2
^{131}I (in thyroid gland)	5.6 -22.4
^{103}Ru	0 - 0.013
^{45}Ca	0 - 0.019
Fissile material	0 - 0.016 (μg)

those in females until the age of 50. The normal rise in lens flecks with increasing age is similar to that found in the mouse, in which it is felt to represent the gradual accumulation of defective lens fibers descended from abnormal but viable cells residing in the germinal zone of the lens epithelium. The progressive increase in the nonexposed individual suggests that some fraction of these cells has been abnormal from the time of lens formation. In distinction to that in the (female) mouse, the rise of lens flecks in the human appears to have a rapid phase coinciding with adolescence in the female, a period of life during which the ratio of estrogen to androgen may be elevated.

The second finding of interest is the greater radiosensitivity (for the production of lens flecks) in females, particularly those aged 13 to 20 years at the time of radiation exposure. As noted above, this is a period of rapid rise in lens flecks in the normal, and possibly of relatively high estrogen level. Experimental evidence pertaining to the influence of estrogen on radiolethality in the mouse[21] suggests the possibility that gonadal hormones may have been at least partly responsible for the difference in radiosensitivity between the sexes, and also for the difference in incidence between adolescence and maturity in the female. Such a mechanism might imply varying radiosensitivity in the female as a function of stage in the menstrual cycle or pregnancy at the time of irradiation. Little information was available as to menstrual status at the time of fallout exposure. Of three women who had been pregnant when they received 175 rads, two have fleck counts higher than would be expected for their age, but the fleck count in the third person was below that expected. These data serve only to suggest the possibility of a gonadal hormone role in radiosensitivity of the optic lens, and definitive answers must be sought elsewhere.

Finally, it should be emphasized that this survey has involved the counting of small numbers of discrete opacities of diameters measurable in microns. These rare, minute defects, possibly involving only portions of single lens fibers, are extremely unlikely to be capable of any effect on visual function. The dense subcapsular and diffuse nuclear changes commonly seen in persons of ages beyond the sixth decade are far more impressive and functionally important.

THYROID FINDINGS

Estimation of Dose of Radiation to the Thyroid Glands

The calculations of dose to the thyroid from the absorption of radionuclides in the fallout unfortunately could be made only from radiochemical urine analyses obtained several weeks after the accident. These calculations are summarized briefly here (see BNL 50029[11] for details). In addition to ^{131}I the isotopes ^{133}I, ^{135}I, and to a lesser extent ^{132}I in the fallout contributed significantly to the thyroid dose. The only direct data available on the Rongelap people are radiochemical analyses of pooled urine samples taken 15 days or longer after the fallout. At 15 days the urine still contained small amounts of ^{131}I. The dose to the thyroid from ^{131}I on the first day of the fallout was estimated to be 11.2 μCi (5.6 to 22.4 μCi) assuming that 0.1% (0.05 to 0.2%) of the maximum thyroid burden (not corrected for physical decay) was excreted in the urine on the 15th day. Table 11 shows estimated body burdens of various isotopes on day 1. The dose of 160 rads to the adult thyroid was calculated from oral intake and inhalation of the combined various iodine isotopes, considering their fission yield, the average energy deposited in the thyroid per disintegration, and the time of absorption. The dose to the thyroid glands of children 2 to 4 years of age was then calculated by means of these factors with consideration of pulmonary function and the thyroid size of the child of that age. The main source of iodine ingestion was considered to be water, and since it was being rationed at the time of fallout it was assumed that the children drank the same amount as adults and therefore had the same thyroid burden of radioiodines. The small size of the children's thyroid resulted in a substantially larger dose. The total estimated

Table 12

Iodoprotein Levels (IP) in Populations Under Study (μg%) 1966 – 1969

Nonexposed and low-dose groups (no thyroxine treatment)

Ailingnae			Utirik			Rongelap nonexposed		
Subject No.	Age (1969) & sex	IP	Subject No.	Age & Sex	IP	Subject No.	Age & sex	IP
6	16 M	5.8	2101	62 M	7.9	813	15 M	1.9
8	16 F	2.8	2146	51 F	7.0	816	19 F	6.4
70	30 F	1.9	2119	33 F	8.1	822	22 M	1.1
59	40 F	2.5	2166	54 M	7.9	833	36 M	1.5
		Av. 3.2	2125	52 M	8.5	835	35 M	2.3
					Av. 7.9	843	40 F	3.2
						855	64 M	7.3
						858	74 F	14.5
						914	34 F	9.2
						931	15 M	0.7
						932	34 F	6.7
						938	30 F	1.9
						956	60 F	6.4
						1532	19 F	3.5
								Av. 4.8

Rongelap people not on thyroxine						Thyroidectomy					
With thyroid lesions (before surgery)			No thyroid lesions			Subtotal			Total		
Subject No.	Age & sex	IP	Subject No.	Age & sex	IP	Subject No.	Age & sex	IP	Subject No.	Age & sex	IP
3	16 M	1.3	32	18 M	1.7	17	17 M	3.5	17	18 F	2.9
5	16 M	1.3	27	27 M	3.3	23	19 M	1.7	21	18 F	1.7
2	17 M	2.0	7	51 M	1.7	33	16 F	1.3	64	45 F	4.1
23	19 M	1.8	68	59 M	3.7	54	16 M	0.9	3*	16 M	1.3
72	21 F	9.2	34	60 F	3.4	65	16 F	3.3	5*	16 M	1.3
15	22 F	10.2	11	65 M	3.6	19	18 M	0.9			Av. 2.6
36	22 M	1.0			Av. 2.9	42	18 F	4.2			
18	36 F	1.0				20	22 M	1.3			
64	45 F	4.5				61	23 F	6.7			
		Av. 3.6						Av. 2.6			

*Placed in this category because of nearly complete atrophy of the thyroid gland.

dose from the various iodine isotopes to the child's gland was about 1000 rads, with a range of 700 to 1400 rads. The glands received an extra 175 rads from external gamma radiation.

Previous Thyroid Studies

Even before the development of thyroid nodules was noted, the evaluation of thyroid status of exposed individuals received considerable attention, since it was recognized that the slight growth retardation noted in some of the children might be related to radiation effects on that gland. However, on the basis of physical examinations and serum PBI and cholesterol determinations each individual examined at that time was believed to be euthyroid. It soon became apparent that the average serum protein bound iodine in the Marshallese of both Rongelap and Utirik was higher than

normal and that from 16 to 64% of the natives on Rongelap and 90% on Utirik showed values that were above the normal range by American standards.[22] No significant differences in the PBI levels were noted between the group that had been exposed to radiation and the unexposed group. Chemical studies of the sera revealed that the high PBI levels could be accounted for largely on the basis of high iodoprotein levels. Lack of recognition of this fact may have masked an incipient deficiency of the thyroid hormone in some cases during these early studies.

More recently, after development of thyroid abnormalities in the exposed Marshallese, it was possible to compare serum iodoprotein levels in cases with thyroid hypofunction with those in euthyroid populations. In Table 12 iodoprotein levels are compared for the Ailingnae, Utirik, Rongelap unexposed, and Rongelap higher exposure groups with and without thyroid abnormalities. The highest iodoprotein values were noted in the Utirik population. The Rongelap unexposed population had higher values than the exposed population. The reasons for this are not apparent. Among the exposed population those with thyroid abnormalities showed about the same mean level as those with no thyroid abnormalities. Following thyroidectomy the iodoprotein levels were slightly lower, but no difference was seen between those with subtotal and those with total thyroidectomy. These data indicate that radiation exposure may have resulted in a slight reduction of iodoprotein levels in the exposed population. Thyroidectomy, partial or complete, resulted in only a partial reduction in the iodoprotein levels; therefore, it appears that the major portion of iodoproteins are produced at extra-thyroidal sites.

Urine iodine levels were in the normal range in the Rongelap people, which indicates adequate intake in the diet, but the values were not quite as high as expected in individuals living close to the sea and eating seafood. ^{132}I uptake studies showed a depressed thyroidal iodine uptake rate and renal excretion rate in the Marshallese (unexposed people) compared with Americans. No explanation for these findings is available. These data are described in detail in the preceding report.[11]

Development of Thyroid Lesions

Nine years after the accident an asymptomatic thyroid nodule was detected during routine annual physical examination in a 12-year-old exposed girl ($\pm$17), and the following year a 13-year-old ($\pm$21) and a 14-yr-old girl ($\pm$69), both exposed, were also found to have nodules of the gland.[23] Since then increasing numbers of thyroid abnormalities have appeared in the exposed Rongelap people. In 19 people nodularity of the gland has been the prominent finding, and in 2 boys ($\pm$3 and 5) atrophy of the gland has developed. The nodules were usually multiple and non-tender and varied in consistency. Surgical exploration, described below, has been carried out in 18 of the 19 nodular thyroid glands. Benign adenomatous lesions were found in all these cases, and malignant lesions were also present in 3 of them. One adult with somewhat less significant nodularity of the thyroid is still under observation. Table 13 outlines the findings on individual cases. Appendix 4 lists thyroid information on the entire exposed Rongelap population (including PBI, cholesterol, etc.). In view of the seriousness of these thyroid developments a panel of experts advised that the more heavily exposed Rongelap people be given supplemental thyroid hormone. This treatment was instituted in September 1965.

Table 14 lists the incidence of benign nodules (including atrophy of the gland) and malignant lesions and the estimated dose of radiation to the thyroid glands in the various populations under study. The highest incidence of thyroid lesions (89.5%) has been noted in the heavily exposed Roneglap group who were $<$10 years of age at the time of the accident. The absence of lesions in people of the same age in the lesser exposed and unexposed groups is most notable. The incidence of thyroid lesions in those exposed as adults in the more heavily exposed group is considerably lower than in those exposed as children but is higher than in the adult population of the Utirik or unexposed groups. (One individual was found to have an adenomatous thyroid lesion in the Ailingnae group.)

The first case of carcinoma of the thyroid was discovered in 1965 in a 41-year-old woman ($\pm$64) in the heavily exposed group, 11 years after exposure.[24,25] At that time the relationship of radiation exposure to appearance of this lesion was seriously questioned, although such lesions are rare in the Marshallese. However, in September 1969 surgical exploration of the thyroid on 5 Marshallese with palpable nodules revealed malignant lesions in 3 additional people. Two of these were women in the more heavily exposed Rongelap group, a 36-

Table 13

Thyroid Abnormalitites in Exposed Rongelap People, 1969
(Arranged in order of appearance of abnormality)

Subject No. and sex	Present age, yr	Age at exposure	Time development: Year	Time development: Age	Findings, present status (1969)
3 M	16	1	1965	12	Hypothyroid, PBI <2μg% March 1965; retardation of growth preceded these findings by a number of years. Growth spurt and improved appearance on thyroxine.
5 M	16	1	1965	12	Hypothyroid, PBI <2μg% March 1965; retardation of growth preceded these findings by a number of years. Growth spurt and improved appearance on thyroxine.
17 F	18	3	1963	12	Adenomatous goiter; total thyroidectomy, 1964. No recurrence, euthyroid on thyroxine.
21 F	18	3	1964	13	Adenomatous goiter; total thyroidectomy, parathyroidectomy, 1964. No recurrence, euthyroid on thyroxine.
69 F	19	4	1964	14	Adenomatous goiter; partial thyroidectomy, 1964. Slight roughening right lobe persists (1968). Euthyroid on thyroxine.
2 M	16	1	1965	12	Adenomatous goiter, partial thyroidectomy, 1965. Slight roughening of right lobe and isthmus (1968). Euthyroid on thyroxine.
20 M	22	7	1965	18	Adenomatous goiter, partial thyroidectomy, 1965. No recurrence. Euthyroid on thyroxine.
64 F	45	30	1965	41	Mixed papillary and follicular carcinoma with localized metastasis. Total thyroidectomy (surgical and radioiodine) 1965. No recurrence. Euthyroid on thyroxine.
72 F	21	6	1965	17	3-mm nodule left lobe. Nodule not palpable 9/66. Recurrence 1969, increased size. Mixed follicular and papillary carcinoma. Lymph node metastasis. Adenomatous nodules were also present. Complete thyroidectomy.
42 F	18	3	1965	14	2-mm nodule right lower lobe. 3/66 nodular enlargement (~1½× normal) entire gland; firm 5-mm nodule right lobe. 7/66 subotal thyroidectomy: adenomatous goiter. No recurrence. Euthyroid on thyroxine.
61 F	23	8	1965	19	6 to 8-mm smooth nodule left lower pole. 3/66 1-cm nodule left lobe. 7/66 subtotal thyroidectory: adenomatous goiter. No recurrence. Euthyroid on thyroxine.
40 M	44	29	1965	40	2-mm nodule right lower pole. 3/66 no nodules detected (reduced on hormone treatment?). 1969 suspicion of slight recurrence.
59 F	*	44	1965	55	5-mm nodule midline. 3/66 same. 7/66 subtotal thyroidectomy: adenomatous goiter.
54 M	16	1	1966	13	Nodular enlargement (~1½× normal) left lobe and isthmus with 2-mm firm nodule. 1968 nodule left lobe 2 cm, thyroidectomy. No recurrence. Euthyroid on thyroxine.
19 M	18	5	1966	15	Multinodular soft enlargement entire gland (~1½× normal). 1-cm nodule right lower pole. 1968 new 1-cm nodule left lobe. Thyroidectomy 1968. No recurrence. Euthyroid on thyroxine.
36 M	22	7	1966	19	Slight nodular enlargement, entire gland. 1-cm nodule, not clearly demarcated, at left lower pole. Many tiny nodules over surface of gland. Nodules enlarged 1969. 9/69 partial thyroidectomy: adenomatous goiter, degenerating follicular adenoma.

Table 13 (continued)

Thyroid Abnormalitites in Exposed Rongelap People, 1969
(Arranged in order of appearance of abnormality)

Subject No. and sex	Present age, yr	Age at exposure	Time of development: Year	Time of development: Age	Findings, present status (1969)
33 F	16	1	1966	13	9/65 questionable irregular gland. 3/66 definite 5-mm nodule left lobe. 7/66 subtotal thyroidectomy: adenomatous goiter, Hürthle cell adenoma. No recurrence. Euthyroid on thyroxine.
65 F	16	1	1966	13	9/65 questionable small nodule. 3/66 5-mm nodule right lobe. 7/66 right subtotal thyroidectomy: adenomatous goiter. No recurrence. Euthyroid on thyroxine.
23 M	19	4	1967	17	1.5 to 2-cm nodule right lobe. 1968 slight increase in size of nodule. Subtotal thyroidectomy 1968. No recurrence. Euthyroid on thyroxine.
15 F	22	7	1968	21	Slight nodularity both lobes. 1968-1969 nodularity increase. Subtotal thyroidectomy 1969. Benign adenomatous nodules.
18 F	36	21	1969	36	Knarled mass right lobe. Complete thyroidectomy. Follicular carcinoma of right lobe. Adenomatous nodules were also present in remainder of gland.

*Ailingnae group; received 70 rads gamma radiation. This patient died of influenza-pneumonia in 1968. Another 77-year-old woman in the Ailingnae group (#43) died in 1964 of pneumonia, and, though no chemical evidence of thyroid adenomata was noted prior to death, histological examination of her thyroid revealed adenomatous changes. A 29-year-old female (#70) from this group had a neurofibroma removed from the left side of the neck, near the thyroid, in 1968.

Table 14

Thyroid Nodules (Plus Hypothyroidism), March 1969

Group	Age at exposure	Estimated thyroid dose,[a] rads	Percent thyroid lesions[b]	Percent malignancy[b]
Rongelap	<10	500–1400	89.5 (17/19)	5.3 (1/19)
(175 rads γ)	>10	160[c]	8.8 (3/34)	5.9 (2/34)
	all	–	39.6 (21/53)	5.7 (3/53)
Ailingnae				
(69 rads γ)	<10	275–550	0.0 (0/6)	–
	>10	55	12.5 (1/8)	–
	all	–	7.1 (1/14)	–
Utirik				
(14 rads γ)	<10	55–110	0.0 (/40)	–
	>10	14	5.1 (3/59)	1.7 (1/59)
	all	–	3.0 (3/99)	1.0 (1/99)
Rongelap				
unexposed	<10	–	0.0 (0/61)	–
	>10	–	2.3 (3/133)	–
	all	–	1.5 (3/194)	–

[a]Dose from $^{131, 132, 133, 135}I$.

[b]Based on present population.

[c]Children 10 to 20 years of age at exposure received up to about 500 rads.

Figure 20. Marshallese thyroid cases (1968) at BNL Medical Research Center with Dr. Conard and interpreter. Extensive examinations at BNL were followed by surgery in Boston.

Figure 21. Medical consultants at the Hospital of the Medical Research Center at Brookhaven to confer on Marshallese thyroid cases (1969). From left to right: Drs. R.A. Conard, BNL; J. Robbins, NIH; H.L. Atkins, BNL; B.M. Dobyns, Western Reserve; and B.P. Colcock, Lahey Clinic.

year-old female (#18) who was 21 years of age at exposure and a 21-year-old female (#72) who was 6 years of age at exposure. This latter case presented the first malignant thyroid lesion noted in the group of heavily exposed children, who have the highest incidence of benign lesions. These recent findings greatly increase the concern about radiation-induced neoplasms in this population. The third malignant lesion was in a woman from Utirik Island. Since the dose of radiation received by that group was very low, it is highly improbable that this lesion is attributable to radiation exposure.

Surgical Exploration of Thyroid Nodules

Thyroid operations have been preformed at the following times: 3 in 1964,* 3 in 1965,** 5 in 1966,** 4 in 1968,** and 5 in 1969.† (Hospital summaries of cases operated in 1968 and 1969 are presented in Appendix 5.)

At surgery the gross appearance of most of the thyroids was lobulated, but in addition they contained grossly discrete masses (see Figures 22 to 24). The benign thyroid lesions exhibited multiple nodules varying in size from a few millimeters to several centimeters in diameter. They varied from soft to firm in consistency, and were hemorrhagic or in many instances cystic. Some thyroid glands had increased fine vascularity over the surface similar to that noted in thyroids previously treated with large doses of ^{131}I for hyperthyroidism. Some of the recent patients were given small tracer doses of radioiodine the day before surgery so that the radioactive content of the nodular tissue could be measured at the time of surgery. The discrete lesions in many cases showed ^{131}I uptake different from that of the extra-nodular tissue, most discrete benign lesions showing less uptake (Figure 25). Radioiodine in malignant tissue was found to be nil compared with that in surrounding normal tissue.

Microscopic examination of the benign lesions revealed marked variation in size of follicles. The cells of some follicles appeared atrophic, while others were hyperplastic, which was reminiscent of iodine deficiency goiter (Figure 26). In addition to the gross adenomatous masses, some of the 15 thyroids classified as benign contained multiple microscopic clusters of what appeared to be atypical proliferating cells here and there in the parenchyma (see Figures 27 and 28).

From the microscopic examination the thyroid carcinomas were considered of low grade malignancy, and they varied in structure from papillary to mixed papillary and follicular type (Figures 29 and 30). Benign adenomatous changes were also noted in the glands. All showed capsular invasion, and in two cases localized metastases to lymph nodes were present and in two other cases, blood vessel metastases. Total thyroidectomies were performed in all three cases of malignancy, and a left radical cervical lymph node dissection also was done in one case because of spread to lymph nodes. No metastases have been recognized beyond the cervical region in any patients.

Thyroid Function: Correlation With Retardation of Growth in Children

In some children with thyroid lesions, deficiency in serum thyroxine has been correlated with retardation of growth. The most striking instances of hypothyroidism were in two boys who showed marked retardation of statural growth and bone age. By 1964, they had developed obvious atrophy of the thyroid gland with almost complete loss of thyroid function as evidenced by failure of the thyroid to take up much if any iodine even after TSH stimulation. By this time their blood had low thyroxine and very high TSH levels. They showed bony dysgenesis, sluggish Archilles tendon reflexes, puffy faces, and dry skin. Their response to thyroid hormone supplement as evidenced by growth spurt, improved appearance, etc., has been dramatic (see Figures 31 to 33). Several other children who displayed thyroid nodularity and whose statural growth was below average showed low or low-normal serum thyroxine values and poor radioiodine uptake after TSH stimulation indicating that their thyroids were functionally impaired and operating near their maximum capacity. Functional deficiency of the thyroid was not demonstrated in adults with nodules or carcinoma of the thyroid.

Influence of Physiological Stress on Thyroid Abnormalities

An assessment was made of the relationship of the development of puberty to the occurrence of thyroid nodules. Degrees of pubescent changes have been recorded annually by a grading system. The two boys who showed greatest retardation of

*By Captain C.A. Broadus (MC)USN, U.S. Naval Hospital, Guam.

**By Dr. B.P. Colcock, New England Deaconess and Bapist Hospitals, Boston, Massachusetts.

†By Dr. B.M. Dobyns, Cleveland Metropolitan General Hospital, Cleveland, Ohio.

Figure 22. Exposure of thyroid at surgery showing nodules. The nodules were benign.

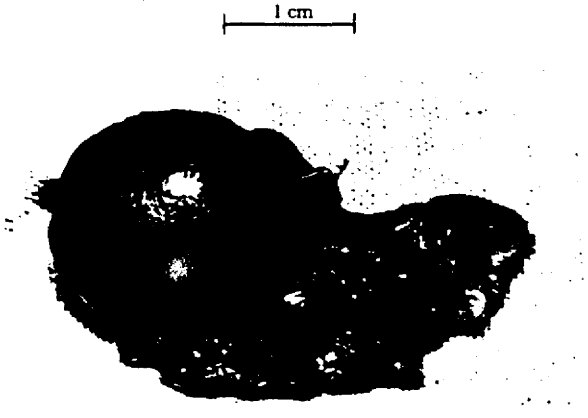


Figure 23. Excised thyroid showing benign nodules.

Figure 24. Excised thyroid showing malignant nodule in upper right lobe.

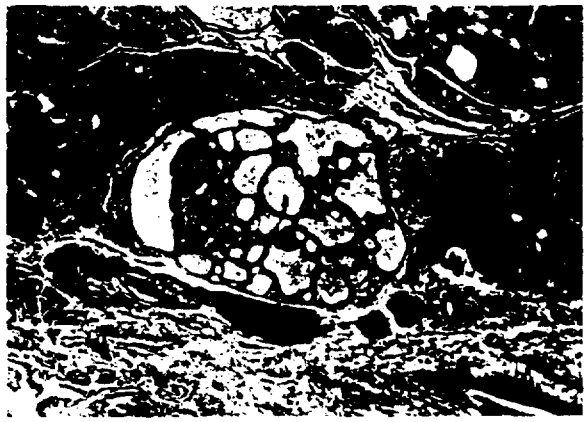


Figure 25. Autoradiograph of section through a nodule of a thyroid with benign lesions. Lack of grains (superimposed blackening) shows that adenoma in center is nonfunctioning. H. and E. stain; ×9.8.

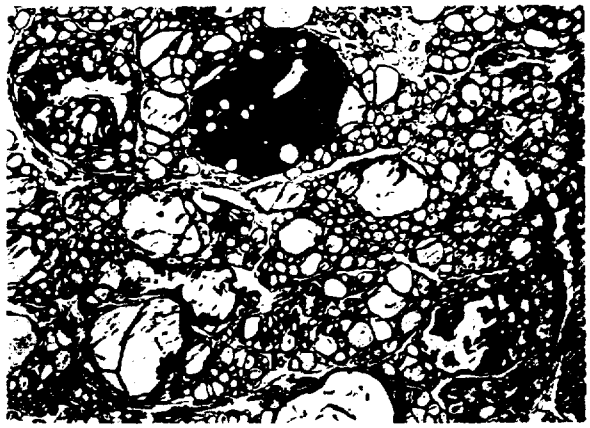


Figure 26. Section of thyroid showing 2 benign papillary adenomas. H. and E. stain; ×9.8.

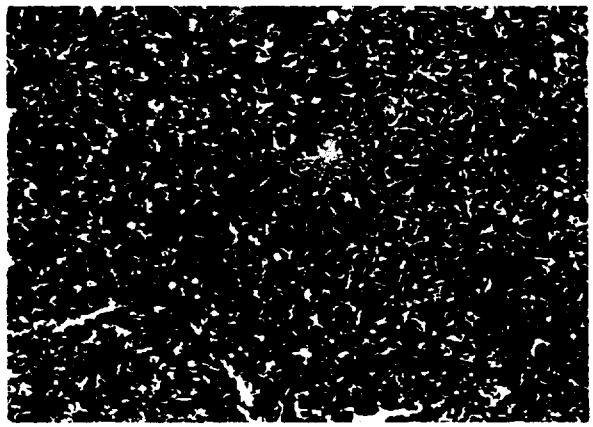


Figure 27. Multiple clusters of what appear to be atypical proliferating cells in a thyroid which contains several large discrete adenomas. The lesions were considered benign. H. and E. stain; ×14.

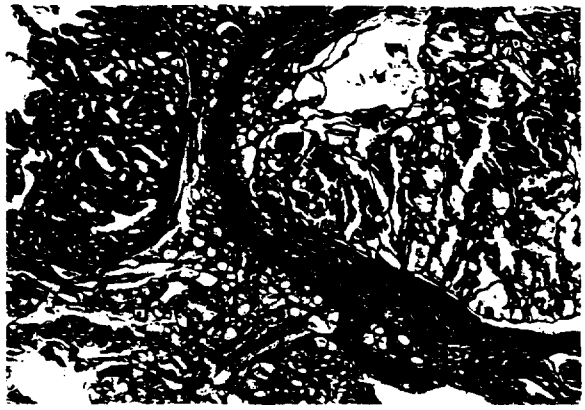


Figure 28. An area of atypical proliferating cells in a thyroid which had developed multiple discrete benign adenomas. H. and E. stain; ×49.

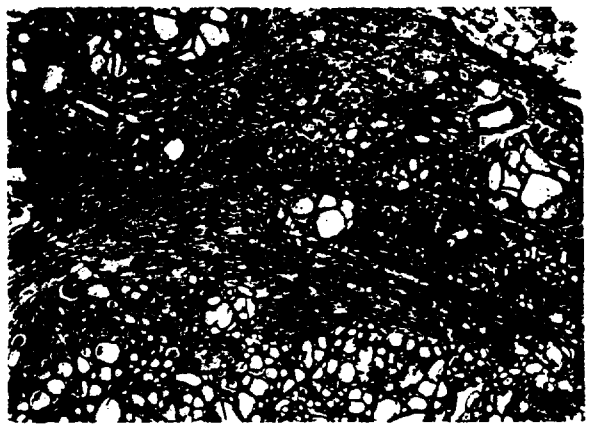


Figure 29. Follicular carcinoma of the thyroid showing capsular invasion in a 41-year-old women exposed to fallout. This represented a discrete mass, as shown in Figure 24. There was no lymph node metastasis. H. and E. stain; ×18.2.

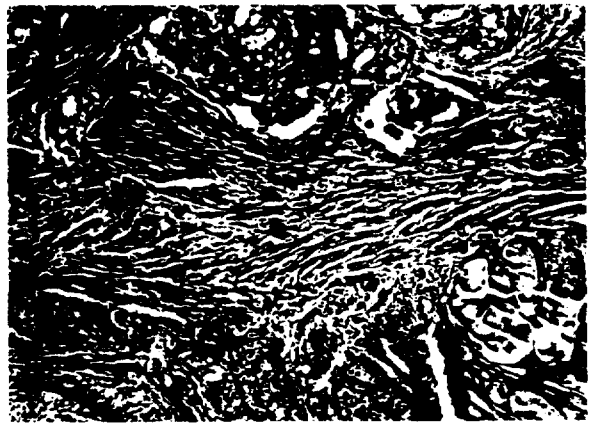


Figure 30. Papillary carcinoma demonstrating extensive connective tissue invasion within the lobe. This patient had multiple cervical lymph node metastases. H. and E. stain; ×46.9.

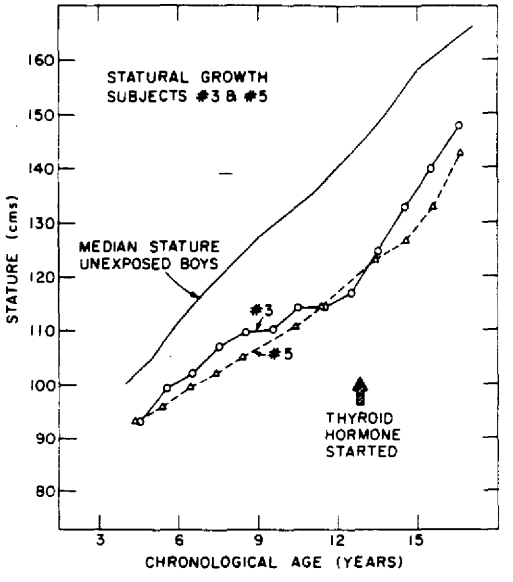


Figure 31. Relative patterns of statural growth (correlated with chronological age) in the two boys who had marked hypothyroidism. Comparison is with unexposed. Note enhanced growth after thyroid treatment.

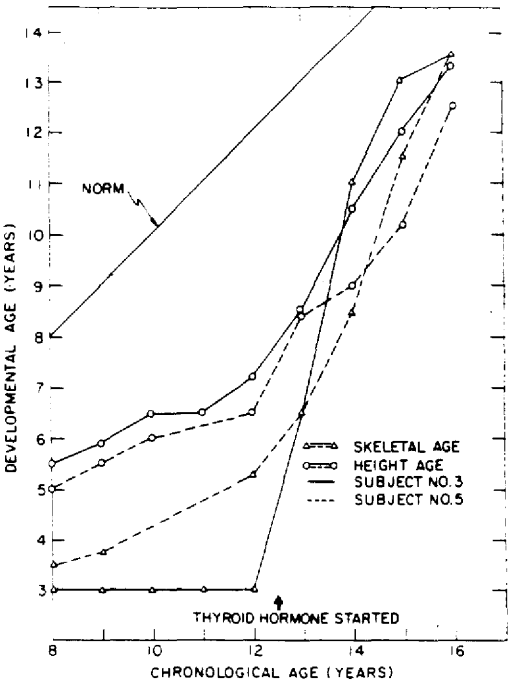


Figure 32. Relative patterns of skeletal maturation (correlated with chronological age) in the two boys who had marked hypothyroidism. Comparison is with unexposed boys. Note the dramatic change in slope of growth curves after thyroid hormone administration.

Figure 33. One of the two boys (#5) showing most retardation of growth with development of hypothyroidism. Left: near the beginning of thyroid hormone treatment (1966, age 13); right: after 3 years of treatment (1969), showing remarkable spurt in growth and development with dissappearance of hypothyroid symptoms.

growth had developed atrophy of the thyroid gland before puberty, and changes associated with puberty were delayed. There may be some association between the apparent increased demand for thyroid hormone at puberty and the appearance of thyroid nodules, since in 10 children (4 males and 6 females) thyroid nodules appeared near the expected or actual time of puberty. In 5 other children (2 males and 3 females) the nodules appeared 1 to 3 years after puberty and in the females were associated with pregnancies. In evaluating the influence of puberty and pregnancy it should be pointed out that the latent period between exposure and the development of thyroid abnormalities was fairly constant in all these children, varying between 10 and 13 years, so that the above findings may have been fortuitous. In the 4 women who developed carcinoma of the thyroid the possible influence of the stress of pregnancy must be considered, since all had multiple pregnancies in the years preceding the development of lesions.

Sex Incidence

The sex ratio of occurrence of benign thyroid lesions in the Rongelap population was 1.3 in females to 1.0 in males. This may be misleading since, in the group exposed at <10 years of age, all the females had lesions whereas 2 males did not. The fact that all 3 malignant lesions of the thyroid were in females is consistent with reported statistics showing a preponderance of such lesions in females.[26]

Discussion

By the time the first malignant thyroid lesion appeared in the more heavily exposed Rongelap group several years ago, numerous benign adenomatous thyroid nodules had appeared. The latter were suspected of being related to the radiation exposure, but such an etiological relationship of the single isolated malignant lesion found at that time was speculative. The finding of 2 additional individuals with thyroid carcinoma in this group (3 among 21 thyroid lesions in 67 Rongelap people exposed) makes the etiological role of radiation exposure increasingly probable. For the time being the single malignant thyroid lesion found in a woman from Utirik Atoll cannot be attributed to radiation exposure because of the low dose received there.

The significance of radiation exposure of the thyroid glands in the Rongelap people had not been fully appreciated until the appearance of thyroid lesions. More careful review of the dose calculations indicated that considerable exposures from radioactive iodine absorption had probably occurred, particularly in the children. The exposure of the Rongelap people was not comparable with exposure of populations due to fallout from reactor accidents in which radionuclides are absorbed chiefly via milk from cattle grazing on contaminated pastures. The Marshall Islands have no cattle and no local milk supply. (Mother's milk may have contributed to the radioiodine absorption in 2 children reported to have been nursing at the time of the accident.) But there was heavy contamination of food and water supplies on Rongelap and a relative abundance of radioiodines in the fallout. The dose to the thyroid glands was greater than that to other organs by a factor of 2 in adults and a factor of about 7 in children.

Numerous animal studies have demonstrated the role of radiation in the etiology of thyroid neoplasms.[27-29] In humans the development of thyroid nodules and cancer from x-irradiation,[30,31] particularly when the radiation occurs in infancy and childhood,[32-34] is well documented. Development of such lesions from radioiodines has also been seen in animals but less frequently in humans. Sheline et al.[30] reported 8 cases of nodular goiter in their follow-up study of 250 cases treated for hyperthyroidism. Six of these cases were irradiated before 20 years of age and 4 before 10 years of age. One showed a possible invasion of the thyroid capsule.

The incidence of thyroid nodularity in the exposed Marshallese is considerably higher than that reported by Pincus[32] and Hempelmann[34] in their studies of populations who had been exposed to therapeutic x-irradiation of the neck region at a young age. However, on a risk per rad basis, the incidence of 51 cases per 10^6 persons per rad per year for the Marshallese is quite comparable with 24 for one group and 64 for a second group calculated by Pincus and Hempelmann. This comparison seems to indicate similar effectiveness – per rad – of x-radiation and radioiodine exposure.*

The 3 malignant lesions of the thyroid reported here in the heavily exposed Rongelap people appear to be the first such cases clearly associated with radioiodine exposure except for one possibly malignant thyroid lesion reported by Sheline et al.[30]

*Mr. Keith Thompson of this Laboratory carried out the statistical analyses in the thyroid cases.

Based on the incidence of carcinoma of the thyroid among the 17,000 Marshallese reported by the Trust Territory, the expected incidence in the Rongelap high exposure group would be 0.056 cases over the 15-year period. The finding of 3 cases (5.6% incidence) thus represents a considerable increase over the expected number ($p<0.01$, χ^2 test). Among the 157 Utirik people about 0.14 cases would be expected, and 1 case is reported. It seems less likely that this single case would involve radiation etiology in view of the low dose received by this group and the fact that no nodular lesions had been noted among the children. Based on the present incidence of thyroid malignancy in the high exposure Rongelap group, the risk of this malignancy developing – per 10^6 persons per rad per year – is 3 cases for the children exposed at <10 years of age, 10 cases for the older people, and 5.6 cases for the group as a whole. The risk in the Marshallese children is not inconsistent with that reported by others.[34]

Radioiodine exposure has been generally considered less effective than x-radiation in producing thyroid lesions, primarily because few thyroid tumors have been noted following radioiodine therapy.[26] It seems likely, however, that the scarcity of such findings is related to the high doses of radiation used (5000 to 10,000 rads or more in the treatment of hyperthyroidism and 50,000 rads or more for ablation of the gland to ameliorate symptoms in certain diseases). Such doses probably are so destructive that they preclude proliferative activity and malignant transformation. The increasing incidence of hypothyroidism without tumor formation, years after treatment of hyperthyroid patients with radioiodines, illustrates this point. Tumor formation in animals is not always a dose dependent phenomenon.[35] Shellabarger et al.[36] showed that the incidence of breast tumors in rats reached a maximum at about 400 rads and fell off with higher doses. Lindsay et al.[29] reported that in rats doses of ^{131}I >200 to 400 μCi were less carcinogenic than lower doses. Mark and Bustad reported similar findings in sheep.[37] Although the dose to the thyroid gland in the Marshallese was generally considerably below the dose of ^{131}I used for therapy of hyperthyroidism, the doses received by some of the children were probably above the optimum carcinogenic range and therefore the true risk per effective rad may be greater in this group. The paradoxical finding of greater risk in the older group appears to be in line with this reasoning. The two stunted Marshallese boys who showed almost complete atrophy of the thyroid gland with no evidence of nodular development are probably comparable with cases of hypothyroidism developing in patients years after radioiodine therapy. The thyroid exposures of the Rongelapese were slightly different from those of patients treated with ^{131}I because their thyroids were not hyperplastic when exposed, at least part of the radioiodine isotopes were of shorter half-life than ^{131}I, and their exposure was complicated by gamma radiation.

Some factors secondary to radiation exposure might have enhanced the development of thyroid lesions in the Marshallese. Iodine deficiency or goitrogens in the diet did not appear to be among these. However, the physiological stresses of puberty and pregnancy may have played a role in the development of the lesions. For instance, the development of 10 cases of nodular goiter in the children during or near the time of puberty might indicate that this stress may have enhanced nodular development. In 3 females who later developed thyroid nodules the demand of multiple pregnancies may have been related. Since the latent period between exposure and nodule discovery varied only between 10 and 13 years, it may be argued that development of thyroid nodules was independent of these stresses. On the other hand it is noteworthy that all the women who developed malignant thyroid lesions had had muliple pregnancies.

The occurrence of thyroid nodules in the Marshallese was only slightly higher in females than in males (ratio of 1.3 to 1.0). This is similar to the nearly equal sex distribution reported by Toyooka et al.[33] for thyroid nodules developing in persons irradiated over the neck region in infancy. However, in the case of carcinoma of the thyroid the expected female preponderance occurred.[26]

The insidious development of growth retardation in some of the Marshallese children before clinical evidence of thyroid abnormality or deficiency was recognized demonstrates the apparent sensitivity of growth and development processes to borderline or subclinical thyroid deficiency. All possible steps are being taken to help the children adhere to the present thyroid treatment schedule so that they will achieve satisfactory growth and maturation.

Careful medical surveillance of these exposed people, including the Utirik, will be essential in future years. The latent period for the develop-

ment of cancer was 7 years in one case and 14 and 15 years in the other two. That the latent period may be longer is supported by Goolden's observation of the development of thyroid cancer 40 years after radiation exposure.[38] It may be that we are just reaching the critical period in the post-radiation observations.

SOME STUDIES OF IMMUNOLOGICAL ASPECTS OF AGING AND FALLOUT RADIATION EXPOSURE

Among the many studies on the Rongelap people, the investigation of the possibility that radiation causes premature aging has been of continuing interest, particularly because such findings have been reported in irradiated animals.[39] The 200 unexposed Marshallese people have served as an excellent comparison population for these studies since they are closely related and live in the same environment. For aging studies, during physical examinations measurements were made on characteristics considered to be criteria of aging.[40] Among these were skin elasticity and looseness and hair grayness; accommodation, visual acuity, and arcus senilis of the eyes; hearing loss; nerve and neuromuscular function, vibratory sense, and hand strength; response to light extinction test and rapid movement test; systolic blood pressure; and levels of blood cholesterol and body potassium (^{40}K). Most of these criteria showed varying degrees of correlation with age and afforded a means of arriving at a "biological age" score for each individual. However, none of the tests showed any significant indication of premature aging in the exposed group that might be associated with radiation exposure.

During the past 3 years these studies have been extended to include examination of some aspects of the immune status in the exposed and unexposed Marshallese populations which might be indicative of aging and/or radiation exposure. The studies reported here include measuring transformation and replication of circulating lymphocytes from phytohemagglutinin (PHA) stimulation in culture, quantification of the various serum proteins by electrophoresis, immunodiffusion studies for immunoglobulin levels, and routine enumeration of peripheral blood elements. In contrast to results of previous studies, some of the present tests showed differences between the exposed and unexposed groups that might be interpreted as radiation effects. Therefore the results on the unexposed population are treated separately here in order to determine the correlation of these criteria with aging in a normal Marshallese population. The results on the exposed group are compared with those on the unexposed group to evaluate possible radiation effects.

Materials and Methods

In Table 15 the numbers of subjects on whom the various tests were done are listed according to age decades.

Lymphocyte Cultures. Blood cultures were set up as follows. The buffy coat was separated from 5 ml of heparinized blood by sedimentation and centrifugation. The culture medium consisted of Eagle's minimum essential medium supplemented

Table 15

Numbers of Marshallese Subjects Tested in Various Studies
(Since the results for males and females showed no significant difference, they were combined)

Age group	Lymphocyte transformation (1968)		Serum proteins (1968)		Immuno-globulins (1968)		Blood elements (1967)	
	Unexposed	Exposed	Unexposed	Exposed	Unexposed	Exposed	Unexposed	Exposed
13–20	11	11	12	11	6	4	29	15
21–30	11	9	11	10	9	2	16	7
31–40	25	10	26	10	9	2	20	7
41–50	19	4	19	4	11	2	11	8
51–60	15	6	17	6	11	3	5	5
61–70	12	3	12	3	12	2	11	3
71–80	9	1	8	1	7	1	6	1
Total	102	44	105	45	65	16	98	46

with 1% glutamine, 15% fetal calf serum, penicillin (100 units/ml), and streptomycin (0.1 mg/ml). Five-ml cultures were seeded with 10^6 leukocytes/ml, PHA M Difco (0.32 mg/ml culture) was added, and the cultures were incubated at 37°C. At exactly 72 hr the cells were harvested, and the number of transformed lymphocytes (blastlike cells) was determined as follows. The cells were prepared for counting by the method of Stewart and Ingram.[41] A 1-ml aliquot of each culture was treated with a proteolytic enzyme (pronase) to remove cellular debris and a cytoplasmic stripping agent (cetrimid) to release intact nuclei. The nuclei were counted and sized with a Coulter electronic counter (Model A). Previous experiments[42] had shown that the transformed cells had nuclei larger than 47 cubic microns. The percent transformation was obtained by comparing the number of larger cells with the total number of cells present. With the above culture technique, the leukocytes removed from the buffy coat are predominantly lymphocytes, but with varying fractions of other leukocytes, principally neutrophils. Although the total number of cells was constant at the beginning of culture for each individual, the number of lymphocytes varied because of slight differences in differential counts. However, by 72 hr, when the final counts were done, practically all neutrophils had disappeared from the cultures so that the percentage transformation of lymphocytes was not significantly affected by this variable.

Serum Proteins. Serum was collected from non-heparinized aliquots of blood from each individual. Total serum proteins (g/100 ml) were determined with a refractometer (American Optical-TS). Separation of serum proteins into albumen and alpha-1, alpha-2, beta, and gamma globulin fractions was done by microelectrophoresis with strips of cellulose acetate (Phoroslides) and a Millipore cell (Millipore Corp.). Barbitol buffer (pH 8.6, ionic strength 0.075) was used with a run separation of 17 min at 100 volts. The protein bands were stained with Ponceau-S dye and then quantified by using a Beckman/Spinco Analytrol with a microzone scanning attachment.

Serum Immunoglobulins. Immunodiffusion procedures for the determination of immunoglobulins IgA, IgD, IgG, IgM, and kappa and lambda light chains were carried out by Dr. John L. Fahey and Dr. Roy Woods of the National Cancer Institute Immunoglobulin Center (Springfield, Virginia). The technique used for quantifying the serum immunoblobulins in antibody-agar plates has been previously described.[43]

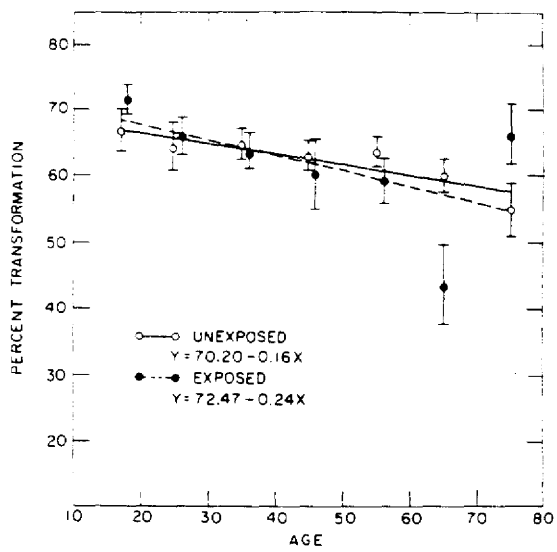


Figure 34. Age-related change in lymphocyte transformation in peripheral blood cultures showing the mean percent transformation for each decade with standard deviation.

Peripheral Blood Elements. The enumeration of peripheral blood elements was part of the routine medical examination of the Marshallese (see below, under Hematological Fndings). Leukocyte counts[44] were carried out electronically (Coulter A counter). Platelet counts[45] were done by phase microscopy. Differential counts of leukocytes (200 cells) were performed on Wright stained smears. Hematocrits were determined by the microcapillary method[46] and sedimentation rates by the method of Wintrobe.[47]

Statistical Analysis of Data. An analysis of variance was used to determine differences among groups for age, sex, and radiation exposure. These data were programmed and analyzed on a high speed digital computor. Since sex differences were not apparent, the results for males and females were combined and each criterion was analyzed for age correlation (r value). The level of significance (p) of differences between the exposed and unexposed groups (radiation effects) was determined; p values $\leqslant 0.05$ are referred to as "significant" in interpreting these findings.

Results

The results are summarized in Table 16, and the values of the various criteria are plotted as a

Table 16

Correlation of Criteria With Age and Radiation Exposure (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

function of age in Figures 34 to 37. Most of the changes generally reached a maximum in the 40 to 50-year age group with little further change in the older groups. Therefore most of the age-dependent correlation was due to differences between the younger (15 to 50 years) and older age groups (>50 years).

Response of Lymphocytes to Phytohemagglutinin Stimulation. The transformation of lymphocytes into blast forms as a result of PHA stimulation in peripheral blood cultures showed a decreasing response with increasing age (Figure 34) which was well correlated with age ($r = 0.89$). Lymphocyte cultures in the exposed group showed no significant differences from those in the unexposed group in response to PHA stimulation ($p > 0.68$).

Peripheral Blood Elements. The changes in various blood elements as a function of age are presented in Table 16 and Figure 35. In the unexposed population the decrease in lymphocyte levels showed the greatest correlation with age ($r = 0.91$) and appeared to reach a maximum in the 50 to 60-year age group. Slight depressions in platelet counts, white blood counts, and hematocrit were noted but were less strongly correlated with age. An increase in sedimentation rate, however, was fairly well correlated with age. In the exposed population the mean levels of neutrophils and platelets were significantly depressed ($p < 0.04$) below levels of the unexposed population, particularly in the older age groups. The other blood findings were not notably different.

Serum Protein Patterns. The results for serum proteins determined by electrophoretic analysis are shown in Figure 36 and Table 16. A slight increase was noted with increasing age in the unexposed Marshallese, but it was not statistically significant. The gamma globulins increased signifi-

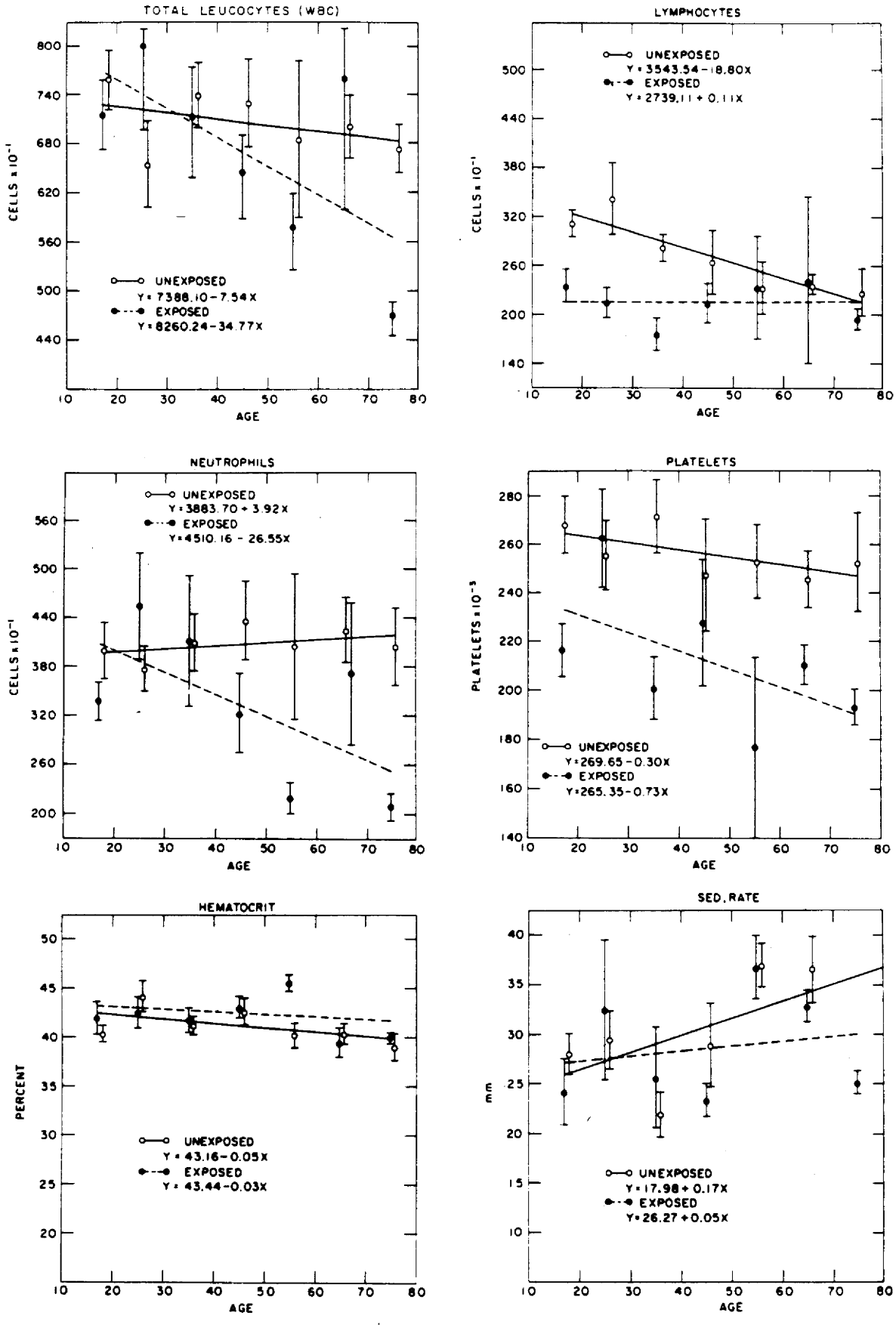
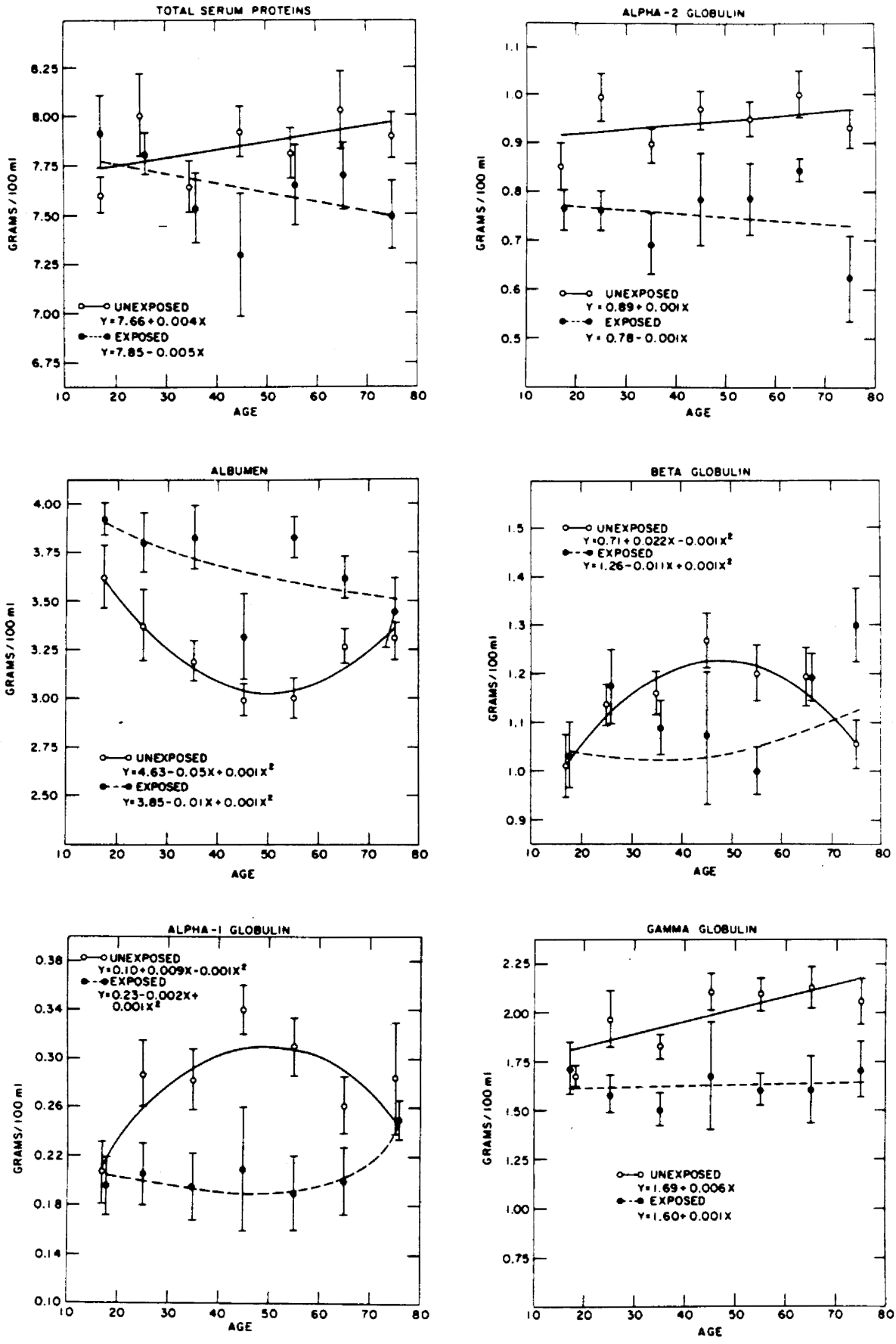


Figure 35. Age-related changes in the blood elements showing the mean level for each decade with standard deviation.



Figue 36. Age-related changes in serum proteins showing the mean level for each decade with standard deviation.

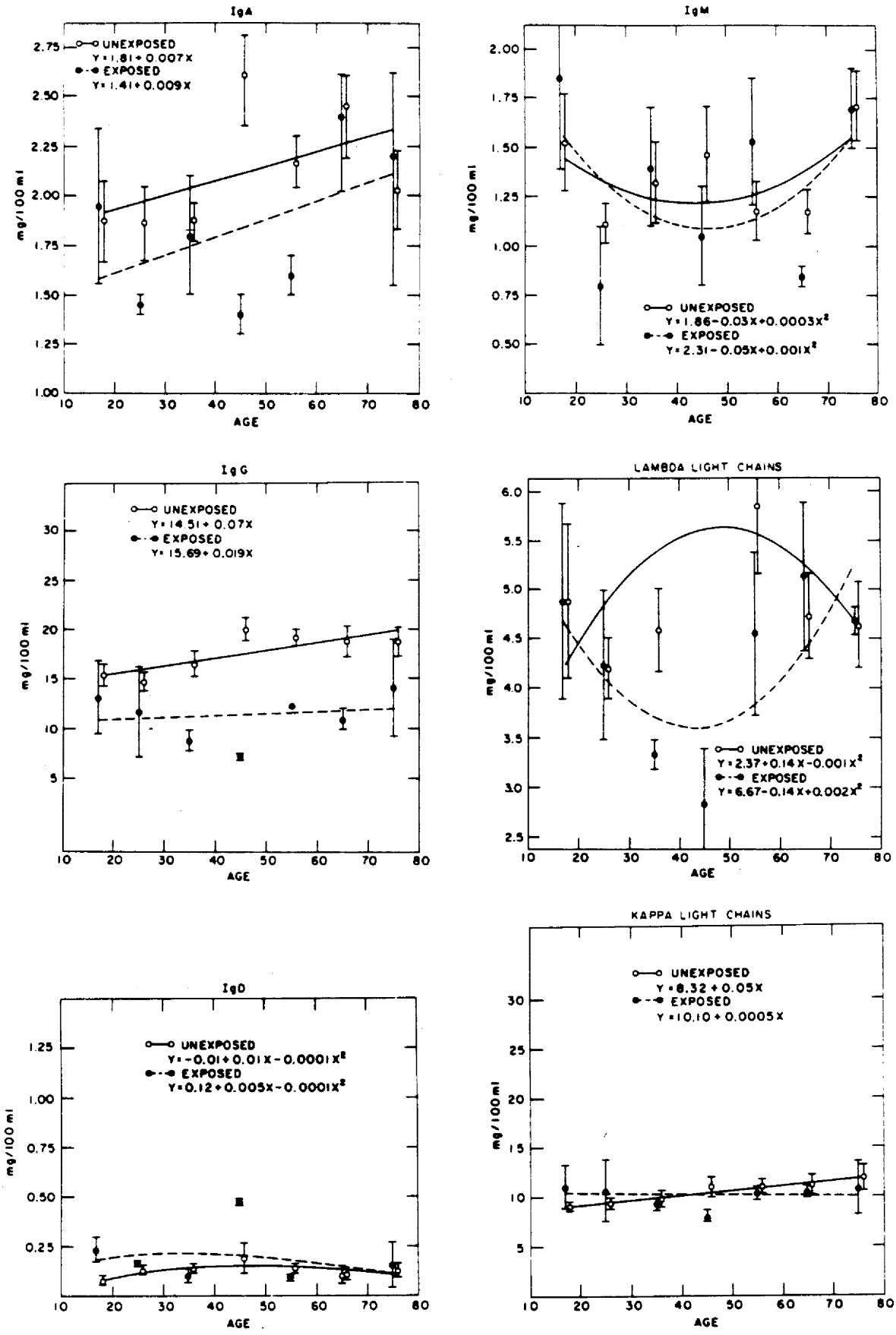


Figure 37. Age-related changes in immunoglobulins showing the mean level for each decade with standard deviation.

cantly in older people. Alpha and beta globulin levels tended to show some increase though the correlation with age was not significant. Albumin levels tended to decrease slightly in older people. Pronounced differences in serum protein levels were noted in the exposed population. The albumen levels were significantly higher and the globulin levels significantly lower than in the unexposed group. Alpha 1, alpha 2, and gamma globulins showed the most pronounced depression in the exposed group (Table 16). Serum proteins, particularly gamma globulins, showed greatest deficits in the older exposed age groups.

The results of the immunodiffusion studies are shown in Table 16 and in Figure 37. Parallel to the increase in serum gamma globulin levels, the immunoglobulins showed increasing values in the older age groups of the unexposed people. The increase in IgG moiety was most pronounced and showed significant correlation with age ($r=0.78$). The increase in the other immunoglobulins did not show a high correlation with age. The increase in the K light chains was highly correlated with age ($r=0.96$). Higher K/L ratios were noted in the older people though there was no significant correlation with age ($r=0.41$). In the exposed population all the immunoglobulins were depressed below levels of the unexposed group, the most pronounced depression being in the IgG and IgA moieties and in the L light chains.

Discussion

Though the role of immune mechanisms in the aging process has never been clearly defined, it is generally agreed that such mechanisms are impaired in senescence. Ram[48] pointed out that "... it is well established that the capacity for immune responses increases during neonatal and juvenile life to a maximum in the young adult animal, it remains constant for a time and then gradually decreases as the animal ages." The results of the present studies in the Marshallese people seem generally to support this thesis.

In connection with the results of these studies, it should be pointed out that the Marshallese people under study are not strictly comparable with a population in the United States because of certain environmental and racial differences. As is generally true throughout Micronesia, even though living standards have been improving, the people are subject to frequent infections such as gastroenteritis, upper respiratory infections, skin lesions, and intestinal parasites. Therefore it is not surprising that they have elevated gamma globulin levels. The A/G ratio is reduced or reversed in most of the people. The serum protein levels are generally elevated, which may in part be related to dehydration associated with a tropical environment where dependence for water is on rainfall which is scant at certain seasons. The specific gravity of the urine is frequently high, and reduced red cell and blood volume have been found. In spite of these factors the present studies should provide valid comparisons of the exposed and unexposed Marshallese populations, since both groups are generally exposed to the same environmental factors, and studies of genetically inherited characteristics show them to be a relative homogenous population.

These studies are believed to test the immunological status of the individuals to some degree, although interpretation of some of the results is not readily apparent. The results of the various tests for the unexposed population are discussed below in terms of correlation with aging. The differences in the results for the exposed population are then discussed with regard to radiation-induced effects.

In the unexposed comparison population it is obvious from the various graphs that, even in the tests showing changes more closely correlated with aging, the changes are maximum by the middle years (usually in the 40 to 50 age group) with little or no increase, or in some cases even slight reduction, after middle age. The reason for this is not apparent.

The response of lymphocytes to PHA stimulation in peripheral blood cultures was tested because of the active role of the lymphocyte in maintaining immunological integrity. PHA is generally believed to evoke a nonspecific blastogenic response in such cultures, since many more lymphocytes are transformed and proliferate with this agent than with specific antigens (such as tuberculin, tetanus, thyroid, pertussis, etc.). It is controversial whether or not PHA in this situation induces antibody formation. The exact mechanism of action of the mitogen is unknown. Conard and Demoise,[49] using autoradiographic and subcellular fractionation techniques, showed that a tritiated PHA was localized largely in the cytoplasm of transformed lymphocytes, with the greatest concentration in the mitochondrial fraction, which suggested that such organelles may be involved in initiation of lymphocyte transformation.

The Marshallese in this study showed a decreasing transformation of lymphocytes with PHA stimulation which was well correlated with increasing age. These results indicated that the percentage of lymphocytes that can respond to PHA was reduced as the Marshallese grew older. Such a finding appears to indicate a general decrease in immunological capacity of the lymphocytes, assuming that the response of these cells to specific antigens is similarly impaired.

Peripheral blood counts showed a decrease in lymphocytes well correlated with aging and compatible with cellular depletion and reduction in immunological capacity generally noted in the aged.[48] These results indicate that with aging there is on an absolute basis a greater loss of PHA-committed lymphocytes than of non-committed types. The slight decrease noted in the hematocrit and platelet levels may also be part of the phenomenon of age-related cellular depletion. Such reduction, however, was not noted in the case of neutrophils. The increasing sedimentation rate noted with aging in the Marshallese could be related to a cumulative effect of chronic infections and debility in the older age groups.

Electrophoretic studies of the serum showed protein levels (both albumen and globulin) well above the usually accepted norms for Caucasians. This may be related in part to dehydration and lowered blood volumes, as mentioned earlier. The increase in gamma globulin levels in the aging Marshallese is consistent with many reports in the literature.[50-53] Possibly the slightly lower albumen levels noted in the older Marshallese may be related to lowered food intake.

As expected from the serum electrophoretic data, the immunodiffusion studies showed increasing immunoglobulin levels with increasing age (Figure 37). The most pronounced and most age-correlated change was in the IgG group. Since the K light chains are twice as prevalent as the L light chains in the IgG immunoglobulins,[54] the significant increase in the K light chains paralleling the increase in the IgG group is not surprising. The K/L ratios in the Marshallese are similar to those in Caucasians[55] and show a slight but significant increase in older people.

The increase in immunoglobulins in the older Marshallese people is probably related to an accumulation of immunological reactions to infections. Parfentjev[56] suggested that the increase in gamma globulins he noted in aging dogs and chickens was related to continuous contact with infectious organisms resulting in hyperimmunization. Such a situation may be present in the Marshallese.

The increased gamma globulin levels seem to be incompatible with decreased immunological reactions in the aged. It has been suggested that the increased globulin levels may be partly related to the development of autoantibodies with age. Blumenthal and Berns[57] state that "... while antibodies to exogenous antigens decrease with age there may be an age-related increase in gamma globulins, presumably containing antibodies to endogenous substances." On the other hand the changes may be of a compensatory nature. Perhaps in older people immunoglobulins are conserved by some mechanisms which tend to decrease catabolism or excretion. Or the cells still capable of producing antibodies might be more active. If it had been possible to study age-related integrity of plasma cells and other lymphatic and reticuloendothelial tissues in these people, further light might have been shed on the problem. Finally, no attempt was made in the studies to separate "normal" older people from those with various morbid changes (if that is possible). Therefore some of the age-correlated changes may be associated with morbid processes. But are not these processes part of aging? It will be interesting in future studies of this population to see whether longitudinal changes on an individual basis agree with the cross-sectional results.

Interpretation of the differences between the exposed and unexposed populations is difficult because radiation-induced aging is a poorly understood phenomenon. It is generally considered to be a late effect of radiation, a manifestation of nonreparable injury, since aging effects are usually not recognizable early. The relative depression of the peripheral blood elements in the exposed Marshallese is probably a continuing manifestation of incomplete recovery from the hematopoietic injury originally sustained. Therefore, if such an effect is a part of the aging process, it has been present since exposure, since continuing depression of blood elements has been noted on annual examinations. The significant depression of the serum globulins and increase in serum albumens in the exposed population is a notable finding. The depression in the gamma and alpha globulins and particularly in the IgA moiety and to a lesser extent in the IgG group and the L light chains

seems to indicate a reduction in relative immunological capacity or at least lowered antibody reserves in the exposed people. The tendency for the depression of these moieties to be relatively greater in the older groups may imply a radiation-induced aging effect. Since such studies were not carried out earlier, it is not possible to say when these changes developed. However, at 3 years post exposure, when the people were challenged with tetanus toxoid, primary and secondary responses were found not to be significantly different in the exposed and unexposed populations.[5] Also, in spite of slight depression of blood elements and reduced serum globulin levels, the exposed Marshallese people, on the basis of our observations over a 15-year period since the accident, have shown no recognizable impairment of immunological capacity as evidenced by the incidence of, or susceptibility to, illness or diseases.[58] If the serum protein changes are a recent development, then such impairment may yet become apparent if the people are faced with a virulent antigenic challenge in the future.

The lack of any differences between the exposed and unexposed people in transformation of lymphocytes in response to PHA stimulation is somewhat unexpected in view of the above findings. However, the lymphocyte levels in the exposed group have not been depressed as severely as some of the other blood elements, which indcates that there may be no significant depression of lymphocytes capable of responding to PHA stimulation.

ESTIMATION OF INTERNAL BODY BURDENS OF RADIONUCLIDES IN MARSHALLESE

The last evaluation of body burdens of gamma emitting isotopes in the Marshallese was done in 1965 on 179 Rongelap people[11] in a shadow-shield whole-body counter with an 11½-in. NaI(Tl) crystal. Radioisotope levels in the exposed and unexposed people were indistinguishable by this method, both groups showing elevated total body burdens of 10 to 20 nCi ^{137}Cs and 1/1000 this amount of ^{60}Co. Also in 1965 radiochemical urine analyses were done on 24-hr samples from 23 people and on two pooled samples. The results showed no increase in internal body burdens since 1961; therefore, no whole-body counts were considered necessary during the next 3 years (1966-1969). However, urine samples were collected for radiochemical analyses* as follows: 24 in 1967, 22 in 1968, and 23 in 1969. The urines were analyzed first for ^{137}Cs by gamma spectroscopy by counting the wet ashed samples on top of an 8 × 4-in. NaI(Tl) crystal. Then the residues were dissolved and analyzed radiochemically for ^{137}Cs and ^{90}Sr. Calcium was determined by the oxalate-permanganate titration method.

Because of their high ^{90}Sr and ^{137}Cs content, coconut crabs on Rongelap Atoll have been banned for use as food. Several crabs were brought back for radiochemical analysis during the past 3 years.

Our group at BNL has been given the responsibility for monitoring the radioactive body burdens of the Bikini people when they return to live on their home island.[59] They are now living on Kili Island, several hundred miles to the south. In order to obtain base-line data, 24-hr urine samples were collected from 14 of the people at Kili in 1969 and brought back for radiochemical analyses.

Individual results of radiochemical urine analyses on the Rongelap and Bikini people for calcium, ^{90}Sr, and ^{137}Cs for 1967, 1968, and 1969 are listed in

*Radiochemical analyses were done at the Environment Studies Division, Health and Safety Laboratory, AEC, New York, by Mr. Edward P. Hardy.

Table 17

Summary of Marshall Island Radiochemical Urine Analyses, 1967–1969

	1967		1968		1969	
	^{90}Sr, pCi/liter	^{137}Cs, nCi/liter	^{90}Sr, pCi/liter	^{137}Cs, nCi/liter	^{90}Sr, pCi/liter	^{137}Cs, nCi/liter
Rongelap exposed	4.8	2.5	3.0	2.8	4.9	3.4
Rongelap unexposed	4.8	2.7	5.6	2.8	4.1	2.9
Kili					0.48	0.34

Appendix 6. The mean values are given in Table 17. The mean urinary levels of ^{137}Cs and ^{90}Sr for 1967-1969 are definitely lower than for 1965. The 1965/1969 ratio for ^{137}Cs (nCi/liter) was 6.3/3.2 and for ^{90}Sr (pCi/liter), 10.1/4.5. Data over the 3-year period 1967-1969 show some variation in both ^{90}Sr and ^{137}Cs levels for both groups but no definite increase or decrease; therefore, it seems justifiable to assume that the body burdens have not increased but have been roughly constant for the past 3 years. This indicates a state of equilibrium with minimal allowance for biological or physical decay.

The drop in the urinary excretion levels of these isotopes suggests an interesting speculation concerning the influence of change in environmental factors. The lowered levels during the past 3 years might be due to increased consumption of foods brought in from the outside with less dependence on home-grown items. The people received fallout compensation payments in 1966 and have had more money to buy imported foods.

The 14 Bikini people living at Kili Island had urinary levels of ^{137}Cs about the same as those of the Rongelap people living on the non-contaminated island of Ebeye and within the range of those of the Marshallese members of the medical team. The ^{90}Sr level of the Bikini group was well below that of the Ebeye people. Since Kili Island is far to the south of the Pacific atom bomb proving ground at Eniwetok and Bikini, there would be little contamination on that island; hence, as expected, the people have very low body burdens of these isotopes. When the people return to Bikini further tests of body burdens, including whole-body gamma spectroscopy, will be done on them and also on the Rongelap people. In preparation for the return of the people Bikini is undergoing a number of procedures intended to reduce the radiation contamination greatly. Among these are clearing away of scrub growth, planting of new coconut and pandanus trees after replacement of the top soil in the area, removal of top soil around the village area and covering with clean coral, disposal of radioactive materials and debris from all the islands of the Atolls, and reducing the coconut crab population. Most of the food for a long time will be brought from outside. Therefore, when the people return, the increase in body burdens is not expected to be anywhere near that measured in the Rongelap people on return to their island. The body burdens of radionuclides in the latter remained far below maximum permissible levels (see below, under Summary of Past Findings).

Table 18

Radionuclides in Coconut Crabs

Year	^{137}Cs, pCi/kg	^{90}Sr, pCi/g Ca
1961		1140
1962		1227 (1317; 1086; 1113; 1378)
1964	50,280 (39,292; 45,318; 66,234)	751 (865; 628; 780)
1965	12,700	724
1969	7,770 (8,540; 7,010)	700 (910; 500)

Table 18 shows the levels of ^{137}Cs and ^{90}Sr in the coconut crabs over the years. The ^{137}Cs levels have been dropping since 1960. The ^{90}Sr levels showed some reduction between 1961 and 1964 but very little after that. The disparity in reduction of the two isotopes may be caused by the molting crabs eating their shed shells, which contain high concentrations of ^{90}Sr. It is unfortunate that the high ^{90}Sr levels necessitate a continued ban of this favorite food, but the crabs are a delicacy rather than a dieary staple since they are not present in great numbers.

HEMATOLOGICAL FINDINGS

Peripheral blood studies were carried out on both the exposed and unexposed Rongelap people in 1967 and 1969 and on the exposed only in 1968. Blood studies were done on the Utirik population in 1969. Leukocyte and red cell counts were done electronically by Coulter counter.[44] Platelet counts were done by phase microscopy[45] in 1967 and by Coulter counter in 1969.[44] Differential counts were performed on Wright stained smears. Hematocrits were determined by the microcapillary method.[46] Sera or plasma samples were collected each year for studies in U.S. laboratories.

The hematological findings for the past 3 years are summarized in Tables 19 and 20 and in Figures 38 to 42. Individual counts are tabulated in Appendix 7 and mean counts over the years in Appendix 8. Total leukocyte counts in the exposed Rongelap people still averaged slightly below those of the unexposed population during the last 3 years, but lymphocyte counts since 1965 have averaged slightly higher than the unexposed mean

Table 19

Mean Levels of Peripheral Blood Elements of Populations Under Study

	Plat. ($\times 10^{-3}$)		WBC ($\times 10^{-3}$)			Neut. ($\times 10^{-3}$)		
	1967	1969	1967	1968	1969	1967	1968	1969
Rongelap exposed	240 (52)*	176(47)	7.2 (52)	7.0(48)	6.5(53)	3.7 (50)	3.8(48)	3.0(53)
Ailingnae exposed	221 (12)	201(10)	6.4 (12)	5.6(12)	5.8(10)	3.0 (12)	3.8(12)	3.2(10)
Utirik exposed	—	—	—	—	7.5(81)	—	—	3.8(79)
Rongelap unexposed	259(102)	191(85)	7.3(102)	—	6.6(99)	3.9(101)	—	3.1(96)
Rongelap children of exposed parent(s)	347 (56)	230(34)	9.9 (56)	—	9.8(65)	4.7 (55)	—	3.9(64)
Rongelap children of unexposed parents	355 (63)	241(45)	8.7 (63)	—	9.5(85)	3.9 (60)	—	4.4(84)

	Lymph. ($\times 10^{-3}$)			Hct. (%)			RBC ($\times 10^{-4}$)	
	1967	1968	1969	1967	1968	1969	1967	1969
Rongelap exposed	2.8 (50)	2.6(48)	3.0(53)	42.0 (52)	42.0(49)	42.9(51)	419 (52)	395(48)
Ailingnae exposed	2.9 (12)	2.2(12)	2.2(10)	38.2 (12)	40.2(12)	38.4(10)	433 (12)	391(10)
Utirik exposed	—	—	3.2(79)	—	—	42.3(81)	—	437(12)
Rongelap unexposed	2.7(101)	—	2.9(96)	41.3(102)	—	43.1(99)	422(102)	402(87)
Rongelap children of exposed parent(s)	4.1 (55)	—	4.9(64)	36.9 (55)	—	38.2(65)	410 (56)	398(63)
Rongelap children of unexposed parents	3.6 (60)	—	4.0(84)	35.5 (63)	—	36.9(85)	406 (63)	393(85)

*Number of people in group.

Table 20

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

	Plats.	Leuks.	Neuts.	Lymphs.
Rongelap				
1959	−5.9	−5.5	−8.2	−0.5
1961	−14.9 ($p<0.01$)	−5.9	−6.0	−5.5
1962	−8.0 ($p<0.1$)	−11.1 ($p<0.01$)	−13.6 ($p<0.05$)	−13.0 ($p<0.01$)
1963	−16.0 ($p<0.01$)	−3.5	−4.4	−3.9
1964	−4.9	−10.5 ($p<0.05$)	−23.5 ($p<0.01$)	−1.1
1965	+0.1	+1.4	−5.1	+5.7
1967	−7.7	−1.4	−5.1	+3.7
1969	−7.4	−1.5	−3.2	+3.4
Ailingnae				
1959	−4.4	−4.8	−6.3	−8.4
1961	−12.6 ($p<0.1$)	—	−13.6	+8.1
1962	−6.5	−15.1 ($p<0.05$)	−17.7 ($p<0.1$)	−11.0
1963	−23.4 ($p<0.01$)	−6.7	+3.9	−20.8 ($p<0.01$)
1964	+4.8	−22.1 ($p<0.02$)	−26.4 ($p<0.1$)	−11.1
1965	+24.8 ($p<0.01$)	−2.7	−2.6	−3.6
1967	−12.6	−12.3	−23.1 ($p<0.1$)	+7.4
1969	+5.9	−12.1	+3.2	−24.1 ($p<0.02$)
Utrik				
1963	+22.7 ($p<0.01$)	—	+2.0	−3.2
1969	—	+13.6	+22.6	+10.3

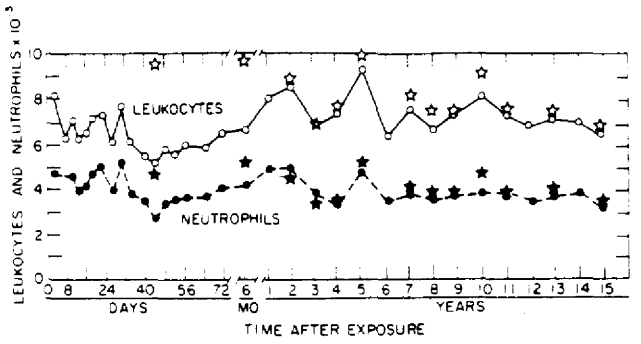


Figure 38. Mean neutrophil and white counts of Rongelap people from the time of exposure through 15 years post exposure. Stars represent mean values of comparison population.

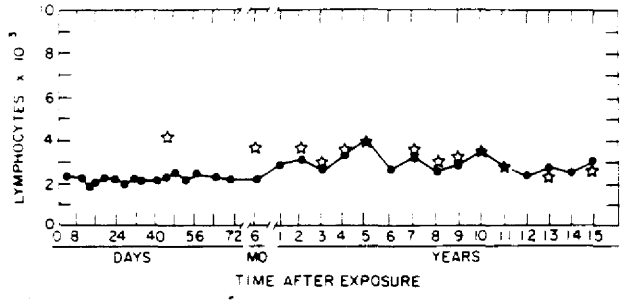


Figure 39. Mean lymphocyte counts of Rongelap people from time of exposure through 15 years post exposure. Stars represent mean values of comparison population.

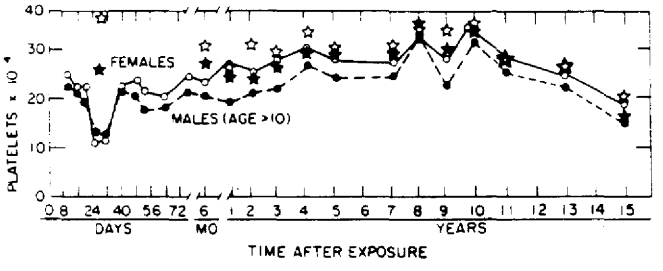


Figure 40. Mean platelet counts of Rongelap people from time of exposure through 15 years post exposure. Stars represent mean counts of unexposed comparison populations.

level. Neutrophil levels continued to show a slight depression in the exposed group, except that in 1969 the smaller Ailingnae group showed a slight increase. Platelets continued to have the most depressed levels of the peripheral blood elements in the higher exposure group. Percentage differences

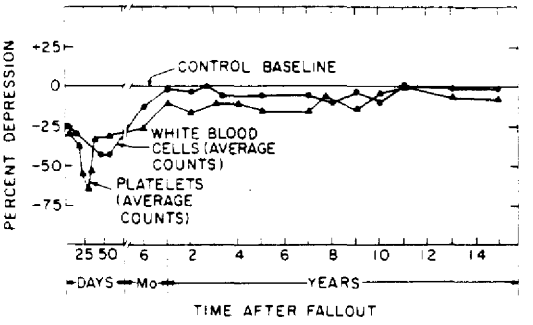


Figure 41. Percent difference in blood counts of exposed and unexposed Rongelap people over 15-year period since accident.

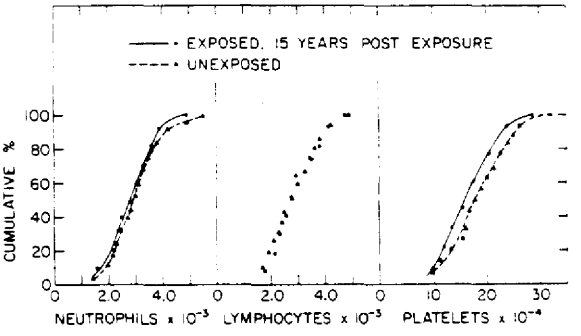


Figure 42. Cumulative percent distribution curves for blood elements of Rongelap people in 1969.

between the exposed groups and the comparison population are shown in Table 20 and Figure 41.

Generally higher counts were noted in the population of Utirik, which had received a very low dose of radiation, but it is difficult to compare the blood counts in the Utirik population with those in the Rongelap unexposed groups because of differences in environment and possibly in ethnic background.

Morphological study of blood cells revealed the continued presence of atypical lymphocytes, particularly in the exposed population. These cells are similar in size to large lymphocytes or slightly larger, the cytoplasm is more basophilic, and the nucleus is more lightly stained with no indentation of the nucleus. Atypical forms were present in 27% of the exposed Rongelap group, 30% of the children of exposed parents, 12% of the unexposed population, and 10% of the Utirik population. The higher incidence in the exposed Rongelap group could be correlated with radiation exposure, but the high level in the children of the exposed

population is difficult to explain. Evidence for residual bone marrow injury may also be associated with continued slight depression of the peripheral blood elements noted above, the finding of bizarre nuclear forms and binucleated forms in bone marrow examinations, and a slight increase in incidence of aneuploidy and 2-hit aberrations in chromosome studies of cultures of the peripheral blood lymphocytes.

Summary of Findings in Rongelap People Over the Past 15 Years

The effects of fallout radiation on the exposed Marshallese can be categorized as prodomal, acute, and late effects. Some special studies are also discussed below.

PRODROMAL RADIATION EFFECTS

During the first 24 to 48 hr after exposure, about ⅔ of the people experienced anorexia and nausea. A few vomited and had diarrhea, many also experienced itching and burning of the skin, and a few complained of lacrimation and burning of the eyes. None of these symptoms was noted in the Utirik people (14-rad group). Following this, the people remained asymptomatic until about 2 weeks after the accident when cutaneous lesions and loss of hair developed, due largely to beta irradiation of the skin. It was apparent when the people were first examined, a few days after exposure, that the lymphocytes were considerably depressed and that significant doses of radiation had probably been received. In addition to the whole-body dose of radiation and the beta irradiation of the skin, radiochemical analyses of the urine showed that measurable amounts of radioactive material had also been absorbed internally.

ACUTE EFFECTS

Penetrating Radiation

One of the earliest findings indicative of a significant exposure in these people was lowering of levels of *leukocytes* and *platelets* of the peripheral blood. This was most marked in the 64 people on Rongelap who had received 175 rads and less so in the other groups receiving less exposure. The hemopoietic depression was roughly proportional to the dose of radiation received. Even in the 157 Utirik pepole 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 chronological records of blood findings in the group exposed on Rongelap are presented in Figures 38 to 41 and Appendix 8.

Lymphopenia of about half the level of the comparison Marshallese population was evident when the people exposed on Rongelap were first examined on their arrival at Kwajalein 3 days after exposure (see Figures 39 and 41). In children <5 years of age the lymphocytes dropped to 25% of the levels in the comparison children, but showed a slight rise during the following weeks. The lymphocyte level showed a slight increase by 1 year. In the following year mean counts approached the levels of the comparison population but remained slightly below it.

Neutrophil levels fluctuated considerably during the first month; possibly this was related to the prevalence of beta burns of the skin during that period. Neutrophil depression became evident by 5 and 6 weeks post exposure (see Figures 38 and 41) with levels reaching about half that of the comparison population in the adults and slightly lower in the children <5 years of age. This degree of neutropenia was insufficient to result in any apparent increased infectious processes, and, indeed, it was noted that neutrophilic leukocytosis was possible in people showing casual infections at this time. Neutrophil levels recovered more rapidly than lymphocyte levels and reached near control levels by 1 year. Subsequent annual surveys have revealed that recovery does not appear to have been complete, particularly in younger and older age groups, during the 15-year period.

Early *platelet* counts showed less fluctuation than other blood counts and fairly consistently showed increasing depression, reaching levels of about 30% that of the comparison population by the 4th week. A spurt of recovery to about 75% of comparison levels occurred during the following few weeks, which was followed by slower recovery but with mean levels never reaching higher than 90 to 95% that of the comparison population during the 15 years post exposure (see Figures 40 and 41).

Erythropoietic depression has not been a consistent finding as with the leukocytes and thrombo-

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

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

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

Clinical findings, except for radiation-induced lesions of the skin, patchy epilation, and early gastrointestinal symptoms, revealed no clear-cut disease processes or symptoms which could be related directly to radiation effects during the first few years post exposure. No prophylactic or specific therapy for radiation effects was ever considered necessary or given. Epidemics of chicken pox and measles that occurred during the first year showed no greater incidence or severity in the exposed than in the unexposed Marshallese people.

During the first months post exposure about ⅔ of the exposed people exhibited *loss of weight* of several pounds. This may possibly have been related to their radiation exposure, although it was difficult to rule out possible effects due to change of environment.

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

Beta Irradiation of the Skin

It was impossible to get an accurate estimate of the radiation dose to the skin. Beta burns of the skin and epilation appeared about 2 weeks after exposure, largely on parts of the body not covered by clothing. About 90% of the people exposed on Rongelap had these burns, and a smaller number developed spotty epilation of the scalp. Most of the lesions were superficial; they exhibited pigmentation and dry, scaly desquamation, and were associated with itching and burning sensations. Rapid healing and repigmentation followed. Some lesions were deeper, showed wet desquamation, and were more painful. A few burns became secondarily infected and had to be treated with antibiotics. Repigmentation of the lesions gradually took place in most instances, and the skin appeared normal within a few weeks. However, in about 15% of the people, deeper lesions, particularly on the dorsum of the feet, continued to show lack of repigmentation with varying degrees of scarring and atrophy of the skin.

Numerous histopathological studies have been made,[1,4,5] and the changes found have been consistent with radiation damage.

Spotty epilation on the heads was short lived, regrowth of hair occurring about 3 months after exposure and complete regrowth of normal hair by 6 months. No further evidence of epilation has been seen.

An interesting observation noted during the first few months after exposure was the development of bluish-brown pigmentation of the semilunar areas of the fingernails and toenails in about 90% of the people exposed on Rongelap. By 6 months this pigmentation had disappeared, having grown out with the nail. The cause of this phenomenon is not known.

Internal Irradiation

Radiochemical analyses of numerous urine samples of the exposed population showed internal absorption of radioactive materials, probably brought about largely through eating and drinking contaminated food and water and to a lesser extent through inhalation. During the first few days when the body levels were at their highest, the maximum permissible concentrations were approached or slightly exceeded only in the case of strontium-89 and the isotopes of iodine. At that time the concentrations were believed to be too low to result in any serious effects. Body levels fell rapidly, so that by 2 and 3 years post exposure they were far below the accepted maximum permissible level; even by 6 months activity in the urine was barely detectable. No acute effects were observed that could be related to internal exposures. As seen later, however, the significance of

the radioiodine absorption from the fallout was not fully appreciated at the time of the early examinations. In addition to ^{131}I, the isotopes ^{133}I, ^{132}I and ^{135}I contributed significantly to the thyroid dose. On the basis of radiochemical urine analyses several weeks after exposure it was estimated that the adult thyroid received about 160 rads from radioiodines plus 175 rads gamma. The smaller glands of the young children, however, received an estimated 700 to 1400 rads plus 175 rads gamma.[11]

LATE EFFECTS

General Health

Except for growth retardation in some children and some thyroid abnormalities reviewed below, the general health of the exposed people has been good and about the same as that of the unexposed population. The incidence of the diseases usually encountered in these people was about the same in both groups.

Mortality

During the 15 years there have been 16 deaths in the exposed group (Rongelap + Ailingnae). This represents 13.0 deaths per 1000 per annum compared with 8.3 for the Marshall Islands as a whole (1960). None of these deaths could be attributed directly to radiation exposure. The higher mortality in the exposed group must be interpreted with caution since the numbers of people involved are too small for a sensitive statistical test and there were more older people in the exposed group.

Fertility

It has been difficult to evaluate the effects of exposure on fertility. During the early period after exposure there may have been some relative infertility. However, the birth rate of the exposed groups over the past 15 years indicates no noticeable effects on fertility (see Table 7) since it has been about the same as that of the comparison population. No radiation-induced sex ratio alteration has been seen.

Miscarriages, Stillbirths, Genetic Effects

The incidence of miscarriages and stillbirths in the exposed women was about twice that in the unexposed women during the first 4 years after exposure. No difference has been noted since then (Table 7). Genetic effects have not been specifically studied because of the small number of people involved. However, no apparent radiation-induced genetic changes have been found on routine physical examinations in the first-generation children of exposed parents, with the possible exception of suggestive evidence of increased miscarriages and stillbirths among the exposed women.

Malignancies

Six cases of cancer have developed in the exposed group, all in females, 3 of the genital tract that resulted in death and 3 of the thyroid gland. Because the thyroid gland received a larger dose, the malignancies of that gland cannot be compared directly with the others. Therefore the genital cancers in the females are less positively related to radiation exposure. Leukemia surveys, including physical findings, studies of leukocyte counts and morphology, alkaline phosphatase staining, and basophil counts of 4000 white cells, showed no evidence of leukemia or leukemic tendency in any of the exposed people over the 15-year period.

Degenerative Diseases

Cardiovascular and arthritis surveys, as well as the general results of physical examination, have not shown any apparent increased incidence of degenerative disease in the exposed people. The Marshallese people appear to have less hypertension on the whole than is noted in the continental United States. An increased incidence of diabetes of the old-age type has been noted in the Marshallese, but no more so in the exposed than in the unexposed population.

Growth and Development Studies

Anthropometric measurements and radiographic studies for bone age on the children have revealed slight retardation in growth and development in boys exposed on Rongelap at age <12 years, particularly in those exposed at age 12 to 18 months. Only slight immaturity was noted in similarly exposed girls. Male children born to exposed parents have shown slight growth retardation and slightly lower levels of peripheral blood elements compared with male children of unexposed parents, but the latter finding has not been evident since 1963. The slight growth difference did not appear to justify the conclusion that it is associated with exposure of the parent. In 1965 marked hypothyroidism with atrophy of the thyroid gland be-

came apparent in 2 boys exposed at 15 months of age who showed the greatest growth retardation. High TSH levels indicated a primary injury of the thyroid. Thyroid function tests on several other children with growth retardation showed evidence of thyroid deficiency (see below). Preliminary results of thyroid hormone therapy have indicated an improvement in growth in some of the children.

Thyroid Findings

By far the most significant of the late effects of fallout exposure noted in these people is a high incidence of thyroid abnormalities. Until the recent development of these abnormalities in 1965, the people were considered to be euthyroid and no abnormalities of that gland were noted. The Marshallese people were found generally to have high serum PBI levels and unusually high iodoprotein values. The latter finding may have masked earlier mild thyroid deficiency. Dietary and urinary iodine levels had been in the normal range. Serum cholesterol levels were not indicative of thyroid disease. Thyroid uptake studies were generally normal but with some pecularities. In 1963 a thyroid nodule was first detected in a 12-year-old girl, and since then increasing numbers of thyroid abnormalities have been noted. Between 1963 and 1966, 18 cases were detected, 15 in children exposed at <10 years of age and 3 in adults. Of these, 16 involved nodular glands, one of which was cancerous, and 2 involved complete atrophy (the growth-retarded boys mentioned above). No such abnormalities were found in the unexposed Rongelap children or the slightly exposed Utirik children, and only a low percentage of nodules was found in the older unexposed population. The exposed population was placed on thyroid hormone treatment in 1965. During the past 3 years further cases have developed; at present among the 66 exposed Rongelap and Ailingnae people now living (of the original 82) 3 have developed malignant lesions of the thyroid, 16 have developed benign nodules of the thyroid, and 2 have atrophy of the gland with hypothyroidism. These findings indicate the seriousness of the exposure to radioiodines in fallout. The majority of thyroid lesions have occurred in persons who were <10 years of age when exposed (90% of that group). Thyroidectomy, partial to complete, has been carried out on 18 Marshallese persons, the first 3 in Guam and the others in the United States following complete study at BNL. The results of these studies and the pathological findings, gross and microscopic, have been published in detail. The slight retardation of growth in some of the exposed children has been correlated with demonstrable deficiency of the thyroid hormone associated with radiation-induced lesions of the gland. The supplemental thyroid hormone treatment seems to be promoting skeletal growth in some of these children. A review of the case histories indicated a possible influence of puberty and pregnancy on the development of the thyroid lesions. The calculated risk of malignant lesions of the thyroid in the exposed Marshallese varied between 3 and 10 cases per 10^6 persons per rad per year for the different age groups. On the basis of these few cases, the risk of thyroid cancer from radioiodine exposure does not appear to be very different from that reported in persons following x-irradiation of the neck region in childhood.

Eye Findings

Ophthalmological surveys showed no remarkable differences between the exposed and unexposed groups except possibly for a slightly greater incidence of pterygia, pingueculae, and corneal scars and pigmentations in the exposed group. It is not known whether these findings are of any significance in relation to radiation exposure. As a whole, visual and accommodation levels in the Marshallese appear to be above the average in the U.S. population. In 1967 slit-lamp examinations of the lens showed a greater incidence of polychromatic sheen and lenticular opacities in the exposed than in the nonexposed population. The differences were not thought to be significant, particularly in view of the higher mean age of the exposed group. In 1969 minute flecks of the optic lens were enumerated in the Marshallese. (In mice low doses of ionizing radiation cause the number of flecks to increase significantly above that expected on the basis of age alone.) In both exposed and nonexposed Marshallese the number of flecks increased with increasing age. The exposed females who were adolescent at the time of exposure had considerably higher fleck counts than comparably aged unexposed females. Preadolescent exposed males had fleck counts twice those in nonexposed males. The presence of these flecks was not thought to imply any likelihood of reduction in visual function. It was suggested that variation in sex hormones might play a role in radiosensitivity of the lens.

Residual Skin Lesions

On recovery of acute beta burns some 20 exposed Marshallese continued to show residual scarring, atrophy, and pigment changes, and biopsy studies revealed residual microscopic changes. However, at no time have any of the lesions developed into chronic radiation dermatitis or shown any evidence either grossly or histologically of malignant change. An increased number of benign pigmented maculae and moles have been noted in previously irradiated areas of the skin, particularly in the neck region.

Hematological Findings

Mean peripheral blood counts in the Marshallese exposed group have tended to remain below those of the unexposed group following the initial acute depression. A slight degree of depression of white cells and platelets has consistently persisted (Figure 41). This implies a residual radiation effect on the bone marrow. Other evidence in support of this was the finding, on bone marrow examination of some of the exposed people, of an alteration in the myeloid-erythroid ratio (increased red cell precursors), presence of cells with abnormal chromatin material and double nuclei, and also increased mitosis. Examination of peripheral blood smears revealed increased numbers of atypical lymphocytes in the exposed group and an unexplained increase in these forms in the children of exposed parents. Also, chromosome studies of lymphocytes in cultured peripheral blood at 10 years post exposure revealed a high incidence of aneuploid cells and 2-hit aberrations in the blood of the exposed group.

Dental Findings

Dental examinations showed no significant differences in caries rate between exposed and unexposed groups. However, the incidence and severity of peridontal disease was slightly greater in the exposed group. It is not known whether this finding is related to radiation exposure. The poor oral hygiene generally observed in the Marshallese resulted in a high caries rate in the teenage children, severe peridontal lesions in the adults (heavy calculus and loss of alveolar bone), and edentulous mouths in the aged. Radiation exposure did not appear to have affected developing dentition in the exposed children.

Aging Studies

Aging studies have been included in several annual surveys to detect the possible influence of radiation on development of premature aging. In the earlier studies various parameters usually associated with aging were measured at the time of physical examination. The values of some of the parameters were estimated and scored on a 0 to 4+ scale (such as graying of the hair, senile changes of the skin, balding, etc.), and the values of others were measured (skin looseness, skin elasticity, accommodation of the eyes, visual acuity, arcus senilis, hearing, blood pressure, neuromuscular function, hand strength, vibratory sense, neuromuscular reaction time, body potassium by spectrographic analysis, etc.). Comparison of these values in exposed and unexposed individuals of the same age showed no apparent difference. Most of the measurements showed varying degrees of correlation with aging. A biological age score was calculated for individuals and groups by use of an average percentage score. During the 1967 and 1968 surveys the age-related and/or radiation-induced aging effect on immunological competence was tested in 100 unexposed and 50 exposed people (Figures 34 to 37). In the unexposed group decreasing immunological competence with aging was indicated by decreasing percentage transformation of peripheral blood lymphocytes by phytohemagglutinin stimulation, indicating decreasing immunological competence of lymphocytes; and by significant increase in gamma globulin, reflected also in increases in IgG and IgA immunoglobulins and K light chains. These latter findings were believed to be associated with age-accumulated effects of repeated infections and perhaps also with increased autoimmune reactions.

The irradiated population exhibited certain significant differences from the unexposed population which may indicate radiation effects. They had relative depression of platelet and neutrophil levels of the peripheral blood. As pointed out before, some slight degree of depression of peripheral blood elements has, however, been noted since exposure. Compared with the unexposed population the exposed group was also found to have reduced gamma globulin levels (including IgG, IgA, and K light chains on immunodiffusion analysis), more pronounced in older age groups, which may indicate a radiation-induced aging effect. Evidence, however, for relative loss of immunological capac-

ity in the exposed population, compared with the unexposed, has not been found on the basis of incidence or severity of diseases.

Absorption of Radionuclides From Environment

A low level of radioactive fallout contamination has persisted on Rongelap. Body burdens of the Rongelapese were evaluated by radiochemical urine analyses until 1957, when gamma spectroscopy by use of a low-level counting chamber was added to the techniques of radiochemical analysis. The return of the Rongelapese in 1957 to their home island (which after careful survey was considered safe for habitation, despite a persisting low level of radioactive contamination) was reflected in a rise in their body burdens and urinary excretion of certain radionuclides. During 4 years after the original contaminating event, additional weapons tests held in the area contributed slightly to the fission products in the environment. Since their diet includes a variety of imported foods, the people may have been delayed in reaching equilibrium with the environmental fission products.

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

Body burdens of ^{90}Sr were estimated from urinary excretion as determined by radiochemical analyses. Both the external dose measurements on Rongelap Island and the levels of radioactive isotopes in the food on the island indicated that some increase in ^{137}Cs, ^{65}Zn, and ^{90}Sr body burdens was to be expected when the people returned there in 1957. The ^{137}Cs body burden in 1958 was about 0.68 μCi, about 60 times as great as in 1957, and the urinary ^{137}Cs level rose by a factor of 140; the mean body burden for 1959 was 0.57 μCi. The mean body burden of ^{65}Zn estimated from whole-body counting data in 1958, after the return to Rongelap, was 0.36 μCi, 8 times as high as in 1957, and 0.44 μCi in 1959. In 1961 the mean ^{137}Cs body burden was 0.67 μCi, which is slightly higher than that of a similar group obtained in 1959; it was 300 times that of the medical team, measured at the same time for comparison. It appeared at this time that the people were approaching equilibrium with their environment. The ^{65}Zn level dropped to 0.071 μCi in 1959. With a larger detector and a longer counting time than previously employed, it was possible to identify and quantify ^{60}Co for the first time in these people; the mean level of ^{60}Co was about 11% of the ^{65}Zn level (7.6 nCi). A small amount of residual activity remained after subtraction of ^{40}K and the above radionuclides from the total spectrum. In 1960 the mean level of urinary excretion of ^{90}Sr was 7.2 pCi/liter, 14% higher than in 1959. In 1962 the mean urinary ^{90}Sr level was 114 pCi/g Ca, giving an estimated body burden of 12.0 nCi. Analysis of bone samples from a deceased Rongelap woman (1962) gave an estimated body burden of 11.4 nCi. These levels represent about a 6-fold increase in ^{90}Sr over the 1958 levels. The levels of ^{90}Sr in 1962 and 1963 hovered around the 12.0-nCi level in adults and about 22 nCi in children, about 5 and 10% respectively of the maximum permissible level (for members of the population at large).

The bone marrow dose from natural and residual radiation sources was estimated in 1958, a peak year, to be as follows:

	Dose to bone marrow, mrad/yr	
Internal		
^{90}Sr (11 nCi body burden)	11.3	
$^{137}Cs + ^{65}Zn$	120.0	
Natural (^{40}K, etc.)	44	
		175
External		
Residual gamma (0.03 mr/hr)	250	
Natural (cosmic, etc.)	134	
		384
Total		559

It thus appeared that by the early 1960's the body burdens of ^{90}Sr and ^{137}Cs in the Rongelap population had reached equilibrium with the environment. Little or none of the body burden in the exposed people at that time could be considered residual from the initial exposure, since little difference was noted between the body burdens of the exposed and unexposed groups living on Rongelap Island.

Radiochemical analyses of urine collected during the 1967-1969 surveys showed levels of ^{137}Cs and ^{90}Sr definitely lower than in 1965. It is speculated that these lowered levels may be explained on the basis of greater consumption of imported food with less dependence on home-grown items.

The ^{90}Sr content of coconut crabs has not become sufficiently lower over the years to allow lifting of the ban on eating them.

Since we have been assigned the responsibility of monitoring the body burdens of radioactive materials in the Bikini people when they return to their home island from Kili, radiochemical urine analyses were done on 14 Bikini people in 1969.[59] As expected, the levels of ^{137}Cs and ^{90}Sr were quite low. In view of the radiological decontamination being done on Bikini in anticipation of their return, no significant increase in body burdens of the people is likely to occur.

SPECIAL STUDIES

Genetically Inherited Characteristics; Blood Grouping Studies

A large body of data has been collected from genetic studies on the Marshallese people. The results not only are of great anthropological interest but also may show in time some possible genetic effects of radiation exposure in future generations. *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.[60] 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.[61] *Transferrins* in all sera were type CC, the common European type.[61] β-Aminoisobutyric acid urinary levels showed the Marshallese to be the highest excreters of this acid of any population thus far reported.[62,63] Levels in the exposed group were about the same as in the unexposed group, and no correlation was found with body burden level of radionuclides; this indicates that there is probably no correlation with radiation exposure. *Hemoglobin types* were considered normal (all had type 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% $Gm^{(a+1)}$ and nearly 100% $Gm^{(b+)}$. There was a complete absence of Gm^x and a high frequency of Gm-like (Gm^c).[64] 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.[65] 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. These data also may be useful as a base line, should genetic changes appear in later generations, possibly related to radiation exposure.

Other Laboratory Studies

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

Sodium levels in the urine and food indicated about the same consumption of NaCl as in Americans. The generally lower incidence of hypertension in the Marshallese might be related to the fact that the former native diet was probably lower in salt content than the present more Westernized diet.[66] It will be interesting to see whether the incidence of hypertension will later increase.

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

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

Serum vitamin B_{12} concentrations (1958, 1959) were generally significantly higher than American levels. The possibility of contamination of the samples with bacteria producing vitamin B_{12} must be considered, since myeloproliferative and liver diseases were not seen.

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

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

A survey for *intestinal parasites* (1958) showed 75% of the people to be infected with various

types.[67] For the three major pathogens found, the overall infection rates were: *Entamoeba histolytica*, 18.2%; hookworm, 5.5%; and *Trichuris trichiura*, 34.3%.

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

Complement fixation studies for parainfluenza 1, 2, and 3, respiratory syncitial, psittacosis, and Q fever showed antibodies to all groups of viruses except that for Asian influenza, which probably had not yet seriously involved the people of the Marshall Islands. The antibody titers appeared to be somewhat lower in the exposed people.

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

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

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References

1. Cronkite, E.P. et al., *The 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 5358, U.S. Government Printing Office, Washington, D.C., 1956.

2. 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.
3. Cronkite, E.P. et al., *Twelve-Month Postexposure Survey on Marshallese Exposed to Fallout Radiation*, BNL 384 (T-71), August 1955.
4. Conard, R.A. et al., Medical survey of Marshallese two years after exposure to fallout radiation, *J.A.M.A.* **164**: 1192 (1957).
5. 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.
6. Conard, R.A. et al., *Medical Survey of Rongelap People, March 1958, Four Years After Exposure to Fallout*, BNL 534 (T-135), May 1959.
7. Conard, R.A. et al., *Medical Survey of Rongelap People Five and Six Years After Exposure to Fallout*, BNL 609 (T-179), September 1960.
8. Conard, R.A. et al., *Medical Survey of Rongelap People Seven Years After Exposure to Fallout*, BNL 727 (T-260), May 1962.
9. Conard, R.A. et al., *Medical Survey of Rongelap People Eight Years After Exposure to Fallout*, BNL 780 (T-296), January 1963.
10. 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.
11. 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.
12. 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).
13. Sutow, W.W. and Conard, R.A., Effects of ionizing radiation in children, *J. Pediat.* **67**: 658-73 (1965).
14. Sutow, W.W. and Conard, R.A., The effects of fallout radiation on Marshallese, in *Monographs on Nuclear Medicine and Biology (Symp. on Effects of Radiation on Fetal and Juvenile Tissues, May 1969)*, Excerpta Medica Foundation, 1970.
15. 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.
16. Culture Fair Intelligence Test (A Measure of "G"), designed by R.B. Cattell and A.K.S. Cattell, distributed by Institute for Personality and Ability Testing, 1602 Coronado Drive, Champaign, Ill.
17. *Handbook for the Individual or Group Culture Fair Intelligence Test, Scale 2, Forms A and B*, Institute for Personality and Ability Testing, Champaign, Ill., 1960.
18. Jordheim, G.D. and Olsen, I.A., The use of a nonverbal test of intelligence in the Trust Territory of the Pacific, *Amer. Anthropologist* **65**: 1122-5 (1963).
19. 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).
20. Bateman, J.L. and Snead, Mary R., Current research in neutron RBE in mouse lens opacity, in *Proc. ORNL-AEC Symp. on Neutrons in Radiobiology, Oak Ridge, Tenn., Nov. 1969*, CONF-691106, p. 192.
21. Thompson, J.S., Reilly, R.W., Craword, Margaret, and Russe, H.P., The effect of estradiol and estriol on the survival of sublethally and lethally irradiated mice, *Radiat. Res.* **26**: 567-83 (1965).
22. Rall, J.E. and Conard, R.A., Elevation of serum protein-bound iodine level in inhabitants of Marshall Islands, *Amer. J. Med.* **40**: 883 (1966).
23. 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 (1965).
24. Conard, R.A., Rall, J.E., and Sutow, W.W., Thyroid nodules as a late sequela of radioactive fallout in a Marshall Island population exposed in 1954, *New Engl. J. Med.* **274**: 1392-9 (1966).
25. Robbins, J., Rall, J.E., and Conard, R.A., Late effect of radioactive iodine in fallout (Combined Clinical Staff Conference at the National Institutes of Health), *Ann. Intern. Med.* **66**: 1214-42 (1967).
26. Sloan, L.W. and Frantz, V.K., Thyroid cancer: clinical aspects, in *The Thyroid*, pp. 445-68, 2nd Ed., S.C. Werner, Editor, Harper & Row, New York, 1962.
27. Maloof, F., Dobyns, B.M., and Vicery, A., The effects of various doses of radioactive iodine on the function and structure of the thyroid of the rat, *Endocrinology* **50**: 612 (1952).
28. Doniach, I., Comparison of carcinogenic effect of x-irradiation with radioactive iodine on rat's thyroid, *Brit. J. Cancer* **11**: 67 (1957).
29. Lindsay, S. and Chaikoff, I.L., Effects of irradiation on thyroid gland with particular reference to induction of thyroid neoplasias: Review, *Cancer Res.* **24**: 1099 (1964).
30. 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 (1962).
31. Hollingsworth, D.R., Hamilton, H.B., Tamagaki, H., and Beebe, G.W., Thyroid disease: A study in Hiroshima, Japan, *Medicine (Baltimore)* **42**: 47-71 (1963).
32. Pincus, R.A., Reichlin, S., and Hempelmann, L.H., Thyroid abnormalities after radiation exposure in infancy, *Ann. Intern. Med.* **66**: 1154-64 (1967).
33. 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).
34. Hempelmann, L.H., Risk of thyroid neoplasms after irradiation in childhood, *Science* **160**: 159-63 (1969).
35. Upton, A.C., The dose-response relation in radiation-induced cancer, *Cancer Res.* **21**: 717-29 (1961).
36. 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).
37. Marks, S. and Bustad, L.K., Thyroid neoplasms in sheep fed radioiodine, *J. Nat. Cancer Inst.* **30**: 661-73 (1963).

38. GOOLDEN, A.W.G., Carcinoma of the thyroid following irradiation, *Brit. Med. J.* **2**: 954-5 (1958).
39. FURTH, J., UPTON, A.C., CHRISTENBERRY, K.W., BENEDICT, W.H., AND MOSHMAN, J., Some late effects in mice of ionizing radiation from an experimental nuclear detonation, *Radiology* **63**: 562-70 (1954).
40. CONARD, R.A., LOWREY, A., EICHER, M., THOMPSON, K., AND SCOTT, W.A., Ageing studies in a Marshallese population exposed to radioactive fall-out in 1954, in *Radiation and Ageing*, pp. 345-60, P.J. Lindop and G.A. Sacher, Editors, Taylor & Francis Ltd., London, 1966.
41. STEWART, C.C. AND INGRAM, M., A method for counting phytohemagglutinin-stimulated lymphocytes, *Blood* **29**: 628-39 (1968).
42. CONARD, R.A., Quantitative study of radiation effects in phytohemagglutinin-stimulated leukocyte cultures, *Int. J. Radiat. Biol.* **16**: 157-65 (1969).
43. FAHEY, J.L. AND MCKELVEY, E.M., Quantitative determination of serum immunoglobulin in antibody-agar plates, *J. Immunol.* **94**: 84-90 (1965).
44. RICHAR, W.J. AND BREAKELL, E.S., Evaluation of an electronic particle counter for the counting of white blood cells, *Amer. J. Clin. Pathol.* **31**: 284-93 (1959).
45. BULL, B.S., SCHNEIDERMAN, M.A., AND BRECHER, G., Platelet counts with the Coulter counter, *Amer. J. Pathol.* **44**: 678-88 (1965).
46. STRUMIA, M.M., SAMPLE, A.B., AND HART, E.D., An improved micro hematocrit method, *Amer. J. Clin. Pathol.* **24**: 1016-24 (1954).
47. WINTROBE, M.M., The blood as a whole, in *Clinical Hematology*, pp. 357-60, M.M. Wintrobe, Editor, Lea and Febiger, Philadelphia, 1967.
48. RAM, J.S., Aging and immunological phenomena - A review. *J. Gerontol* **22**: 92-107 (1967).
49. CONARD, R.A. AND DEMOISE, C.F., Preparation and purification of tritiated phytohemagglutinin and studies of cellular localization in human lymphocyte cultures, *Blood* **35**: 44-55 (1970).
50. KAREL, J.L., WILDER, V.M., AND BEBER, M., Electrophoretic serum protein patterns in the aged, *J. Amer. Geriat. Soc.* **4**: 667-82 (1956).
51. RAFSKY, H.A., BRILL, A.A., STERN, K.G., AND COREY, H., Electrophoretic studies on the serum of normal aged individuals, *Amer. J. Med. Sci.* **224**: 522-8 (1952).
52. GOLDBLOOM, A.A., New clinical and laboratory studies in the aged. V. Lipidogram by paper electrophoresis in normal patients 80-100 years of age, *Am. J. Dig. Dis.* **22**: 51-8 (1955).
53. DAS, B.C. AND BHATTACHARYA, S.K., Changes in human serum protein fractions with age and weight, *Can. J. Biochem. Physiol.* **39**: 569 (1961).
54. RITZMANN, S.E. AND LEVIN, W.C., Polyclonal and monoclonal gammopathies, in *Lab Synopsis, Vol. II.*, pp. 9-35, H.R. Dettlebach and S.E. Ritzmann, Editors, Hoechst Pharmaceutical Company, 1967.
55. FAHEY, J.L., Personal communcation, 1969.
56. PARFENTJEV, I.A., Changes in serum proteins during aging in humans and animals, *J. Gerontol.* **9**: 363-4 (1954).
57. BLUMENTHAL, H.T. AND BERNS, A.W., Autoimmunity and aging, in *Advances in Gerontological Research, Vol. I.*, p. 313, B.L. Strehler, Editor, Academic Press, New York and London, 1964.
58. 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*, pp. 325-36, International Atomic Energy Agency, Vienna, 1969.
59. Conard Named to Monitor Bikinians, AEC News Release #M-108, May 1, 1969.
60. SUSSMAN, L.N., MEYER, L.M., AND CONARD, R.A., Blood groupings in Marshallese, *Science* **129**: 644 (1959).
61. BLUMBERG, B.S., Biochemical polymorphisms in animals: haptoglobins and transferrins, *Proc. Soc. Exp. Biol. Med.* **104**: 25 (1960).
62. BLUMBERG, B.S. AND GARTLER, S.M., High prevalence of high-level β-aminoisobutyric acid excretors in Micronesians, *Nature* **184**: 1990 (1959).
63. GARTLER, S.M., An investigation into the biochemical genetics of β-aminoisobutyric aciduria, *Amer. J. Hum. Genet.* **11**: 257 (1959).
64. STEINBERG, A.G., STAUFFER, R., BLUMBERG, B.S., AND FUDENBERG, H., Gm phenotypes and genotypes in U.S. whites and negroes; in American Indians and Eskimos; in Africans and in Micronesians, *Amer. J. Hum. Genet.* **13**: 205 (1961).
65. BLUMBERG, B.S., DRAY, S., AND ROBINSON, J.C., Antigen polymorphism of a low density beta-lipoprotein: allotype in human serum, *Nature* **194**: 656 (1962).
66. MENEELY, G.R. AND DAHL, L.K., Electrolytes in hypertension: the effects of sodium chloride, *Med. Clin. N. Amer.* **45**: 271 (1961).
67. GOLDMAN, M. AND CARVER, R.K., An intestinal parasite survey on Rongelap Atoll in the Marshall Islands, *Amer. J. Trop. Med. Hyg.* **8**: 417 (1959).

APPENDIX 1

Pediatric Anthropometric Data (Height and Weight) on Rongelap Exposed Children, 1965 through 1969

				1965		1966		1967		1968		1969	
No.	Sex	Age at Exposure	Birth Date	Ht Cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb
83	M	In utero	6/8/54	137	74	144	86						
84	M	" "	5/31/54	129	64	133	65	138	71	141	77	148	---
85	M	" "	9/17/54	129	67	---	65	137	69	141	71	145	---
86	F	" "	10/17/54	126	53	131	---	138	67	144	76	147	---
54	M	1 y	2/21/53	145	89	152	101	159	112	165	123	166	135
65	F	1 y 2 m	12/52	130	59	136	70	141	77			145	90
5	M	1 y 4 m	10/52	116	54	122	56	127	66	132	73	143	---
3	M	1 y 5 m	9/52	117	80	125	63	133	71	140	97	148	---
2	M	1 y 4 m	10/52	138	71	144	80	148	91	159	110	164	120
6*	M	1 y 4 m	10/52	132	68	138	79	145	90	154	100	157	113
8*	F	1 y 8 m	6/52	147	90	154	96	153	96	153	100	154	---
33	F	1 y 7 m	7/52	156	100	159	106	161	109	160	114	160	---
42	F	3 y	3/51	145	80	148	81	149	92	149	95	149	96
21	F	3 y	3/51			150	96	150	102	150	101	151	---
17	F	3 y 4 m	10/50	155	110	157	105	157	115	158	116	158	118
23	M	3 y 5 m	9/50	154	102			166	136	169	140	169	148
32	M	3 y 6 m	8/50	141	76	148	88	156	102	161	116	163	121
44*	M	4 y	3/50			156	98	161	110				
69	F	3 y 7 m	7/50	158	109	160	114						
19	M	5 y 2 m	1/49	155	93	---	103	158	103				
48*	F	5 y 8 m	6/48	155	116	155	115						
72	F	7 y	3/47	157	136								
15	F	7 y	3/47	159	115								
20	M	6 y 9 m	5/47	162	119								
36	M	7 y 4 m	10/46	166	136								
61	F	8 y	3/46	155	166								

*Ailingnae Group

Pediatric Anthropometric Data (Height and Weight) on Rongelap Unexposed Children

1965 through 1969

			1965		1966		1967		1968		1969	
No.	Sex	Birth Date	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb
801	M	6/29/56	114	44								
802	M	3/16/56	123	52			133	66			143	110
803	M	3/18/56	123	50								
805	F	2/25/54	135	71			148	97			158	126
806	M	1/ /55	126	56							155	---
807	M	6/13/55	120	45			129	54			128	64
808	F	3/29/55	132	67			145	103			147	123
809	M	6/18/56	128	51			137	57			145	71
810	F	2/5/55	137	63							154	---
811	F	2/14/54	130	65			143	90			146	101
812	F	2/ /54	132	114			145	83			155	---
813	M	1/2/54	131	61			139	73			154	99
814	M	4/5/52	140	77							161	131
815	M	5/4/50	159	105			165	120			166	135
816	F	10/31/49	153	110								
817	M	10/19/50					170	161				
818	M	3/4/51	153	91							175	133
819	M	12/15/48	168	134								
820	M	10/25/48	164	125							165	132
821	F	8/1/47	--	132								
863	M	6/25/50	157	109			163	130			164	144
866	F	7/ /55	125	54			139	70			149	103
870	M	2/21/56	127	53			135	63			144	---
879	F	4/ /54	131	60			145	82				
891	F	5/15/48	153	97			153	100				
892	M	7/17/46	161	121								
900	F	7/ /57	126	51			138	68				
901	F	7/12/57	117	49			126	62			127	81
902	F	10/23/57	116	45								
903	F	11/19/57	111	43			122	48			133	50
904	M	12/22/57	118	49			129	62			138	71
905	M	10/23/57	112	39							126	---
906	F	3/1/58	113	39			123	48			132	63
909	F	3/11/50	142	94			143	99				
911	F	3/8/53	142	91			146	120				
912	M	6/1/53	135	63								
913	M	3/27/51	147	86								
919	M	3/9/48					158	92				
921	M	10/11/53	130	64			140	73			154	101
923	F	6/9/55	121	51			129	61			141	76
924	M	/55					126	57				
925	F	5/4/50	146	90			149	105			149	114
926	F	2/26/51	146	84			157	108			159	---
930	F	4/4/56	125	56								
931	M	/54	127	58			141	78			153	---
937	F	/53	143	87			157	125				
939	M	1/10/46	165	153								
940	M	9/19/48	159	116			160	121				
946	F	10/6/50	150	100			151	113				
952	M	9/26/56	119	48			126	55			135	---
954	F	6/ /56	112	41			124	51			134	---

Pediatric Anthropometric Data (Height and Weight) on Rongelap Unexposed Children

1965 through 1969 (cont'd)

			1965		1966		1967		1968		1969	
No.	Sex	Birth Date	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb	Ht cm	Wt lb
955	F	5/11/52	150	104			152	123			152	120
960	F	12/5/51	150	108			152	116				
962	F	11/3/52	135	70			147	98			150	119
972	M	7/1/55	126	51			133	59			143	---
976	M	10/19/47	167	167								
978	F	10/20/50	154	115								
979	F	6/5/55	117	46			129	57			141	---
980	F	10/3/52	148	94			151	106			153	---
981	M	8/8/54	128	55			136	61			147	81
988	F	7/ /54					143	81				
992	F	8/14/57									138	70
993	F	2/25/47	156	123								
995	F	3/19/57	114	47							131	
996	F	1/16/53	140	76			147	95			148	110
998	F	6/12/47	---	124								
1002	M	3/22/55					125	56			132	63
1004	M	5/31/58	109	42			119	50				
1006	M	8/12/58	111	42								
1008		/58	104	36							126	64
1009	M	1/22/59	104	41							121	---
1010	M	1/27/59	104	37			114	46			122	54
1011	F	3/9/59	102	37							119	
1013	M	/60	95	32			107	40				
1014	M	/62	115	42			127	50			134	58
1015	M	12/31/60	101	37							122	---
1017	M	/62					116	43			125	51
1018	M	3/17/60	97	34			110	41			121	48
1019	F	1/18/60					111	44			120	54
1020	F	7/15/59	---	31							123	---
1021	F	5/11/59	102	36							124	---
1022	F	11/20/59	105	35			115	45			123	53
1024	M	11/13/59	105	42								
1025	F	6/13/59	99	32			110	40			118	---
1026	F	5/28/60	104	34			116	40			126	53
1027	M	12/7/60	94	30							114	---
1028	M	2/22/61	---	31			105	34			117	---
1029	F	3/8/61	98	35			109	42			119	51
1030	M	3/26/61	96	32								
1031	F	1/5/61	102	35			113	42			124	54
1032	M	4/7/60	98	32			106	37			115	---
1033	M	/50	155	126			157	147				
1034	F	/62	119	44			130	54				
1035	F	/49	148	112			148	126			148	---
1036	M	/50	141	76			156	99				
1037	M	9/18/61	---	28			103	35			112	42
1038	M	10/31/61	93	33			104	40			112	50
1039	M	12/18/61	---	26			100	31			110	37
1040	M	9/24/61	95	33								
1044	F	9/17/62	---	24			---	30			125	36
1046	M	10/9/62					90	28				
1049	M	/61	105	40			116	49				

Pediatric Anthropometric Data (Height and Weight) on Rongelap Unexposed Children
1965 through 1969 (cont'd)

No.	Sex	Birth Date	1965 Ht cm	1965 Wt lb	1966 Ht cm	1966 Wt lb	1967 Ht cm	1967 Wt lb	1968 Ht cm	1968 Wt lb	1969 Ht cm	1969 Wt lb
1052	M	/53	140	82			155	121				
1053	M	/56	106	39							126	57
1054	M	11/29/63	---	22							100	---
1056	M	3/4/64	---	17			90	30			105	39
1057	F	8/4/63	75	23			90	32				
1058	M	1/16/64	---	18			88	24			98	29
1059	M	5/ /64	---	17			82	22				
1060	F	7/ /64	---	16								
1062	M	8/ /64	---	18			84	24			---	33
1063	M	10/ /64	---	16			---	28			97	37
1064	M	7/ /64									92	27
1065	F	9/ /63					94	34			107	---
1066	F	4/ /63					93	29			103	32
1067	M	10/ /61					99	32			109	37
1068	M	4/ /63					88	30				
1069	F	3/ /65					---	24			97	30
1070	F	7/ /65					---	23			112	31
1072	M	10/ /65					---	20			86	26
1074	M	6/ /66					---	19				
1075	F	6/ /66					---	19				
1076	M	6/ /66					---	16			81	24
1077	M	1/ 67					---	11				
1078	F	2/ /67									---	22
1079	M	/ /66					82	26				
1080	F	/ /63					100	36			109	---
1082	M	10/ /66									---	27
1084	F	3/1/67									---	26
1088	M	2/2/68									---	21
1089	M	3/16/68									---	21
1091	M	8/14/68									---	19
1092	M	10/4/67									---	25
1093	M	8/2/67									77	22
1094	F	10/8/67									---	19
1095	F	3/ /68									75	20

Pediatric Anthropometric Data (Height and Weight) on Children Born to Exposed Parents

1965 through 1969

No.	Sex	Birth Date	1965 Ht cm	1965 Wt lb	1966 Ht cm	1966 Wt lb	1967 Ht cm	1967 Wt lb	1968 Ht cm	1968 Wt lb	1969 Ht cm	1969 Wt lb
87	F	10/17/54	124	54			136	66			148	---
88	M	9/8/55	124	59			134	73			145	98
89	M	12/28/55	120	50			129	60			138	74
90	M	11/29/55	129	61			138	72			146	86
91	M	1/3/55	129	60			138	71			153	99
92	F	3/16/56	121	51			---	62			142	---
93	M	2/17/57	122	51			131	61			139	74
94	F	10/ /56	112	45			120	51			134	---
95	F	2/5/56					133	60			149	---
96	M	2/12/58	113	48			121	58			---	71
97	M	10/31/57					128	56			138	---
98	M	3/5/58	105	40								
100	F	4/26/56	122	52			133	70			145	---
101	F	4/24/58	110	43			122	51			132	---
102	M	3/16/58					126	51			135	---
103	F	5/28/58	111	40			122	47			131	59
104	M	10/2/58	109	38			119	46			127	---
105	F	10/9/58	112	44			122	53			132	---
106	F	3/11/59	107	38			119	50			128	64
108	M	1/7/60	109	38			118	47			127	55
109	M	1/7/60	107	44			119	---			128	---
110	M	12/5/59	104	38			115	64			124	57
111	M	5/24/59	102	31			112	40			121	49
112	F	6/8/59	101	34			112	43			122	52
113	M	2/27/61	98	33			110	40			120	
115	M	8/16/60	95	37			107	42			116	---
116	M	5/15/60	102	40			114	42			123	---
117	F	3/28/61					111	39				
118	M	11/25/60	98	33			108	40			117	46
119	F	7/19/60	97	36			108	41			118	---
120	F	6/27/60	100	37			113	48			123	62
121	F	6/7/60					98	33				
122	F	4/12/60	99	34			111	40			121	50
123	F	11/26/61	88	29			103	36				
124	F	10/23/61	92	30			104	36				
125	F	6/ /61	95	32			109	40			119	50
126	M	9/26/61	---	29			104	37			116	45
127	F	5/17/62	---	27			97	34			110	42
128	F	1/30/63	---	23			95	31			109	---
130	M	4/19/63	---	19			92	29			100	---
131	M	10/28/63	---	21							104	---
132	M	/ /63	---	21			92	31			105	39
134	F	5/ /63	---	25							109	41
135	F	9/10/62	---	14			80	22				
136	F	3/ /63					95	31			106	---
137	F	8/ /64	---	14							96	27
138	F	9/ /64	---	14			81	21			91	27
139	F	12/ /64	---	12								
141	M	3/ /65					---	24			94	32
142	M	3/ /63					96	30			108	---

Pediatric Anthropometric Data (Height and Weight) on Children Born to Exposed Parents

1965 through 1969 (cont'd)

No.	Sex	Birth Date	1965 Ht cm	1965 Wt lb	1966 Ht cm	1966 Wt lb	1967 Ht cm	1967 Wt lb	1968 Ht cm	1968 Wt lb	1969 Ht cm	1969 Wt lb
143	F	12/14/64	---	9			81	23				
144	F	4/22/63					---	31				
145	F	3/30/64	71	21							97	35
149	F	3/30/65					---	19				
150	M	1/12/66					---	20				
152	F	6/ /65									---	32
153	F	6/31/66									---	19
154	F	9/15/66					---	16				
155	M	11/22/66									83	26
156	F	2/20/67					---	10			83	25
157	M	4/23/65					---	20				
163	M	2/ /67									77	24
165	F	5/ /67									---	26
166	M	7/ /68									---	16
168	M	7/22/66									---	27
172	M	11/20/68									---	16
173	F	3/9/17									---	26

Appendix 2

RESULTS OF CULTURE FAIR INTELLIGENCE TEST
(SCALE 2 FORM A) 1969

EBEYE GROUP					RONGELAP GROUP				
Subject (Initial)	Age (Yrs.	Mos.)	Sex	Raw Score	Subject (Study No.)	Age (Yrs.	Mos.)	Sex	Raw Score
M.L.	16	1	M	20	2	16	5	M	12
J.B.	15	9	M	22	6	16	5	M	22
C.C.	15	11	M	29	15	22	0	F	11
R.P.	16	11	M	24	42	18	0	F	16
J.O.	16	4	M	31	47	23	6	M	20
K.B.	17	8	M	27	48	20	10	F	10
T.K.	15	6	M	23	53	23	3	F	14
F.C.	15	5	M	21	61	23	0	F	9
H.M.	16	0	M	25	65	16	3	F	8
R.P.	13	10	F	29	88	13	6	M	8
M.R.L.	16	9	F	16	93	12	1	M	12
K.B.	15	9	M	25	811	15	1	F	15
L.L.	14	3	M	34	813	15	2	M	9
S.A.	17	2	M	17	814	16	11	M	18
R.L.	15	8	F	29	816	19	5	F	16
R.R.	17	1	F	26	821	21	7	F	15
M.H.	15	11	F	22	921	15	5	M	11
I.A.	17	2	F	21	959	20	7	F	15
E.A.	13	10	F	27	960	17	3	M	13
					981	14	7	M	13
					966	14		F	10
					1523	23		M	21
					1529	23		M	10
					823	25	7	M	15

For the 14-year age group (13 yrs. 9 mos. to adult) a total raw score of 25 stands at the 20th percentile and a total raw score of 30 stands at the 44th percentile.

Appendix 3

Individual Lens Scores (Fleck counts)

Males			Females	
I.D. #	Score	Age	I.D. #	Score
		Non-exposed		
813	11	15	811	0
921	5	15	812	0
931	0	15	1534	3
981	0	15	980	8
		15	805	4
		16	996	28
814	3	17		
1533	0	17		
		18	1035	2
		18	926	3
		18	960	2
		19	816	0
		20	959	0
		21	821	3
		21	891	75
		22	993	33
1523	4	23		
1529	20	23		
823	9	25		
		27	1525	19
		27	825	56
827	4	29	896	1
	-	30	829	2
830	14	30	938	34
		31	832	25
		31	1505	5
		33	1050	23
		33	1528	28
		34	934	0
		34	1043	4

Appendix 3 (continued)

Individual Lens Scores (Fleck count)

Males			Females	
I.D. #	Score	Age	I.D. #	Score
		34	932	35
		34	914	47
834	3	35	1001	13
		35	835	45
882	13	36	841	2
836	0	36	865	48
833	1	36		
1005	0	36		
966	19	37		
920	22	37		
881	5	37		
840	55	39	1520	12
1526	51	40	843	90
		41	867	24
1527	17	43		
864	16	43		
944	160	44	922	1
		44	945	265
842	48	45		-
868	73	46	846	11
880	2	48	982	0
1517	60	49		
849	0	50	844	10
		55	970	3
1007	17	58		
		60	956	21
		60	851	53
948	5	61	957	42
878	29	69		
915	5	72		
		76	859	80

Appendix 3 (continued)

Individual Lens Scores (Fleck count)

Males			Females	
I.D. #	Score	Age	I.D. #	Score
		Exposed to 175 r		
5	0	15	86	0
		16	33	12
3	35	16	65	0
2	5	17		
		18	42	13
		18	21	0
		21	72	2
36	4	22	15	35
47	18	23	61	5
76	9	26		
35	2	28	24	66
		30	39	68
		31	74	5
		32	22	19
73	12	33	12	67
37	24	35		
		36	18	35
9	5	37		
10	0	39		
		40	14	5
27	35	41		
		43	71	14
40	19	44		
		45	64	42
		45	66	33
7	17	51	63	18
		52	78	80
4	24	53		
79	9	54		
68	0	59		
11	0	65		
82	5	65		
		74	58	0

Appendix 3 (continued)

Individual Lens Scores (Fleck count)

Males			Females	
I.D. #	Score	Age	I.D. #	Score
		Exposed to 14 r		
2245	4	15		
		17	2226	4
		18	2171	1
		19	2159	20
		21	2209	80
2144	11	22		
2235	7	23	2128	0
2156	15	24		
2250	25	26		
2165	8	27		
2234	0	28		
		31	2249	11
		39	2117	60
2252	35	54		
		Exposed to 70 r		
84	20	15		
6	14	15		
		16	8	1
		21	48	11
		23	53	2
		23	81	63
		30	70	12
		47	45	148
16	13	54		
41	23	59		
		69	1	10

Appendix 4

Thyroid Data on Exposed Marshallese Populations
Rongelap (175 rad)

Patient Number	Age at Exposure	Sex	Thyroid Status	Age Abnormality Noted	PBI, T4* 1965	1966	1967	1968	1969	Cholesterol 1965	1966	1967	1968 - 1969	Remarks
3	1	M	Myxedema	12	1.5 0.8	3.2 1.0	3.6	1.2	2.0				187	Marked growth retardation TSH >100 1967
5	1	M	Myxedema	12	2.1 0.8	2.6 1.8	1.1	1.0	1.0			195	155	Marked growth retardation A PBI in 1963 was 2.5 µg%
33	1	F	Adenomatous nodules Subtotal thyroid-ectomy 1966	13	7.0	5.9 3.8		6.3	5.7				103	
54	1	M	Adenomatous and Hurthle cell nodules Subtotal thyroid-ectomy 1966	13	8.3	5.0 4.3	4.8	4.6				89	98	
65	1	F	Adenomatous nodules Subtotal thyroid-ectomy 1966	13	7.0	4.1 2.7	2.3		8.7 3.1			120		Considerably below average statural ranking; TSH 20 1967
2	2	M	Adenomatous nodules Subtotal thyroid-ectomy 1965	12	7.0 4.2	5.2 2.6		4.8 2.6	10.7 3.5				174	Below average statural ranking
17	3	F	Adenomatous nodules Total thyroidectomy 1964	12	2.3	1.8 <0.5	1.4		5.9			171	123	TSH >100 with T-4 of 1.4 in 1967
19	3	M	Adenomatous nodules Subtotal thyroid-ectomy 1968	14-15	3.9 3.3				6.2					Considerably below average statural ranking
21	3	F	Adenomatous nodules Total thyroidectomy and parathyroidec-tomy 1964	12	0.7	1.3 <0.5	1.6	0.8	5.2			120	168	Below average statural ranking. Increased TSH level in 1967 along with low T-4 from not taking thyroid medication properly

*T-4 underlined

| Surgery

Patient Number	Age at Exposure	Sex	Thyroid Status	Age Abnormality Noted	PBI, T4* 1965	1966	1967	1968	1969	Cholesterol 1965	1966	1967	1968	1969	Remarks
32	3	M	Negative			4.6 4.0	3.0	4.0	6.2			74	130		
42	3	F	Adenomatous nodules Subtotal thyroid-ectomy 1966	15		5.7 4.6		6.0	14.1 7.3				196		Considerably below average statural ranking
23	4	M	Adenomatous nodules Subtotal thyroid-ectomy 1968	16			2.3	4.5	1.4				188		
69	4	F	Adenomatous nodules Subtotal thyroid-ectomy 1964	14	7.1	5.7 1.7									
72	6	F	Carcinoma follic-ular-papillary; com-plete thyroidectomy 1969	17	5.3		3.0	3.7	5.9			128	156		Thyroid had adenomatous nodules also
15	7	F	Adenomatous nodules Subtotal thyroid-ectomy 1969	21	6.4		3.4	6.6	9.5 3.9			137	194		
20	7	M	Adenomatous nodules Subtotal thyroid-ectomy 1965	18	6.6 4.2				4.9						Below average statural ranking
36	7	M	Adenomatous nodules Adenoma, subtotal thyroidectomy 1969	19	4.1	4.2 4.3		2.6	2.6			122			A PBI in 1963 was 2.7 µg%
47	8	M	Negative					5.3	4.5				174		
61	8	F	Adenomatous nodules Subtotal thyroid-ectomy 1966	19	6.4	7.9 4.7			11.6 3.8						

*T-4 underlined

| Surgery

Patient Number	Age at Exposure	Sex	Thyroid Status	Age Abnormality Noted	PBI, T4* 1965	1966	1967	1968	1969	Cholesterol 1965	1966	1967	1968	1969	Remarks
76	11	M	Negative (?)		4.4	5.6			3.1						Diffuse hypertrophy of thyroid 1969 T-4 3.1
75	12	F	Negative		4.7				8.1 6.7						
24	13	F	Negative					12.4					216		
35	13	M	Negative		4.6				3.0						
67	14	F	Negative		6.6				6.8						
39	15	F	Negative		11.9				6.2				177		
49	15	F	Negative						6.6						
74	16	F	Negative						3.0						
22	17	F	Negative					8.9	6.7 6.9				161		
12	18	F	Negative					11.3	10.7				250		
73	18	M	Negative		8.4				11.6 7.2						
37	20	M	Negative		5.3										
18	21	F	Carcinoma - follicular; complete thyroidectomy 1969	34	11.7				3.9						
9	22	M	Negative												
10	24	M	Negative		8.3										
14	25	F	Negative		7.1										

*T-4 underlined

| Surgery

Patient Number	Age at Exposure	Sex	Thyroid Status	Age Abnormality Noted	PBI, T4* 1965	1966	1967	1968	1969	Cholesterol 1965	1966	1967	1968	1969	Remarks
27	26	M	Negative		7.1	4.1									
77	26	M	Negative												
71	28	F	Negative												
40	29	M	Nodule	40	10.3			5.5	2.6? 8.0				176		Nodule reduced after 1965, but again suspicious 1969
64	30	F	Carcinoma-follic-ular-papillary; complete thyroid-ectomy 1965	41	8.7 |	2.0	2.0	1.8	10.3			235	160		Remains euthyroid on thyroxine, no metastasis
66	30	F	Negative		8.0										
7	36	M	Negative												
63	36	F	Negative												
78	37	F	Negative												
4	38	M	Negative												
79	39	M	Negative												
68	44	M	Negative		7.1 4.6	5.8	4.3					121			
34	45	F	Negative		9.1 4.6	6.5	3.6					210			
80	46	M	Negative												
11	50	M	Negative		8.4		3.14					112			
82	50	M	Negative												
60	56	F	Negative												
58	59	F	Negative	|Surgery	9.2										

*T-4 underlined

Rongelap 69 rad (Ailingnae)

Patient Number	Age at Exposure	Sex	Thyroid Status	Age Abnormality Noted	PBI, T4* 1965	1966	1967	1968	1969	Cholesterol 1965	1966	1967	1968	1969	Remarks
6	1	M	Negative		8.5	5.0 3.6		2.2	3.8						Note slightly less T-4 recently
8	1	F	Negative		5.7	5.4 4.0		5.4	4.1						
44	4	M	Negative			4.7									
48	6	F	Negative			4.8		5.9	7.3 4.3						Slight hypertrophy isthmus ? 1969
53	8	F	Negative		8.5			5.2							
81	8	F	Negative					2.7							
70	15	F	Negative					4.3							Removal of neurofibroma neck 1968
51	25	F	Negative		9.7										
45	32	F	Negative		8.4										
50	34	M	Negative		10.1										
16	39	M	Negative												
41	44	M	Negative		6.7										
**59	44	F	Adenomatous goiter Subtotal thyroid-ectomy 1966		8.6 5.3	6.4 4.7 |	5.9	7.1							
1	54	F	Negative												

*T-4 underlined
**Died September 1968

\| Surgery

Appendix 5

HOSPITAL RECORDS OF THYROID CASES (1968-1969)

BROOKHAVEN NATIONAL LABORATORY
HOSPITAL of the MEDICAL RESEARCH CENTER
UPTON, NEW YORK

DISCHARGE SUMMARY

NAME: Rongelap 19 | UNIT NO.: 8-18-91 R

PAVILION 1 OPD

ADMITTED: AUGUST 4, 1968 DISCHARGED: AUGUST 30, 1968

This 17-year-old boy was admitted to this Hospital for evaluation of a nodular thyroid gland which did not respond satisfactorily to treatment in the islands.

HISTORY OF PRESENT ILLNESS: In 1966 the thyroid gland was found to be slightly enlarged with irregular soft nodularity with a more distinct 1 cm. nodule detected in the right lower lobe. He had been put on thyroid hormone therapy (Synthroid, 0.3 mg. / day) since September 1965 but had only taken the drug for one month. He took thyroid medication thereafter however and in 1967 it was thought that his thyroid nodularity had reduced somewhat. However in 1968 a new 1 cm. thyroid nodule was palpated in the upper left lobe, and a nodule was still palpated in the right lower lobe. Earlier PBI's were 6.4 µg% in 1958 and 4.1 µg% in 1959 with BEI of 2.7 µg%. His 1968 serum was lost. He has appeared to be euthyroid. His stature has remained about 2 years behind his peers. In view of his lack of response to thyroid hormone therapy it was decided that he should be fully evaluated here.

The patient was 5 years old at the time of the fallout. He received an estimated 175 rads of whole body gamma radiation, irradiation of the skin from the fallout deposited thereon and internal absorption of radioisotopes. His thyroid gland received about 700-1400 rads plus 175 rads of gamma radiation. During the first two days he experienced loss of appetite and nausea. About 2 weeks postexposure he developed "Beta burns" of the skin mainly on the scalp, axillary and anal region and feet. These healed rapidly. He also developed rather marked epilation with normal regrowth of hair occuring in several months. His peripheral blood leukocyte count was depressed but within a year had returned to near normal levels. There was no evidence of the clinical radiation syndrome associated with the hematologic depression. Following recovery from the early radiation effects he remained in good health with no serious illnesses or injuries. No evidence of thyroid disease has been apparent and the PBI and thoroxine (T-4) levels were 3.9 and 3.3 µg% in 1966. His serum TSH level was not elevated at that time. His statural growth has been somewhat below that of his peers.

PHYSICAL EXAMINATION: This 17-year-old Marshallese boy was of rather shorter stature than most Marshallese males of his age but he appeared well-nourished and was without complaints. The only notable physical findings were related to the thyroid gland. The left lobe of that gland was slightly enlarged, soft and slightly tender. In the lower pole of the right lobe there was a 1-2 cm. nodule of firmer consistency. No regional lymph adenopathy was noted. The patient appeared to be euthyroid.

LABORATORY & X-RAY DATA: Thyroid workup: Thyroxine 3.0 µg%; cholesterol 110 mg% (82% esters); BMR -1, -16; thyroid scan with ^{99m}Tc showed a cold nodule indenting the lower right pole of the thyroid. ^{131}I uptake and a repeat uptake study following TSH, 10 units daily for 3 days, showed a poor response to the pituitary hormone. The

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thyroxin level did not increase after the stimulation. His serum was negative for antithyroglobulin antibodies.

OTHER: Chest x-ray negative, EKG normal, other laboratory tests essentially negative except for a few WBC on repeated urinalysis (cause not determined) and the finding of whipworm ova in the feces.

HOSPITAL COURSE: The following thyroid experts were consulted: Dr. J.E. Rall, Dr. J. Robbins of NIH, and Dr. B. Colcock of Lahey Clinic, Boston. The consensus was that the diseased thyroid tissue should be surgically removed. The patient was transferred to the New England Baptist Hospital in Boston, Massachusetts on August 18, 1968. Dr. B.P. Colcock of Lahey Clinic carried out subtotal thyroidectomy removing multinodular thyroid tissue from both lobes. Dr. W.A. Meissner of the New England Deaconess Hospital examined these tissues and his pathological diagnosis was "adenomatous goiter". Recovery from surgery was uneventful and the patient was transferred back to this Hospital August 25. A small pouch filled with serous fluid at one end of the surgical wound was drained following his return and the subsequent hospital course was uneventful with satisfactory healing of the wound. He was placed on thyroid medication and discharged for return to the Marshall Islands on August 30, 1968.

FINAL DIAGNOSIS: Adenomatous goiter.

DISCHARGE MEDICATION: To continue thyroid hormone therapy for life. A note was sent to the Marshallese practitioner in charge of the case stressing the importance to continued thyroid medication. The patient was given an adequate supply of the hormone to last for at least one month.

Robert A. Conard

Robert A. Conard, M.D.

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Dict: 9-13-68
Typed: 9-16-68

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DISCHARGE SUMMARY

NAME	UNIT NO.
Rongelap 23	8-18-89 R
PAVILION 1	OPD

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ADMITTED: AUGUST 4, 1968 DISCHARGED: AUGUST 30, 1968

This 18-year-old Marshallese boy was admitted to this Hospital for evaluation of nodules of the thyroid gland resulting from fallout exposure in 1954.

HISTORY OF PRESENT ILLNESS: Though nodules of the thyroid gland were first noted at the time of the 1967 medical examinations development of the nodules could have occurred earlier since the boy had not been examined in 1966. Multiple, small, non-tender nodules were palpated in the gland with one larger nodule about 1.5 cm. in the right lobe, and a smaller one in the left lobe. He appeared to be euthyroid and normally developed. His serum thyroxin level in March was 4.5 µg% and serum cholesterol 188 mg% (in 1967 his thyroxin level was 2.3 µg%). L-thyroxine (Synthroid, 0.3 mg./day) was prescribed in 1967 but his adherence to the treatment regimen was spasmodic. Therefore during the 1968 examinations it was decided that since his thyroid nodules had not reduced, hospital evaluation was indicated.

The patient was 4 years of age at the time of exposure to fallout on Rongelap. He received a whole body dose estimated to be 175 rads, expesure to the skin (unknown dose) and a dose to the thyroid gland from absorbed radioiodines of about 700-1400 rads plus 175 rads of penetrating gamma radiation. Relative lymphopenia was noted at 3 days slight leukopenia at 6 weeks followed by rapid return to the normal range. He developed slight "Beta burns" on the neck, axillary and anal region which cleared up in a few weeks. He had moderate epilation of the temple with complete regrowth of hair by 6 months. No clinical evidence of irradiation syndrome was apparent at any time. Anthropometric measurements and skeletal age studies have revealed normal growth and development with no evidence of thyroid deficiency. A PBI level in 1958 was 9.6 µg%. Since the examination in March, 1968 he has been asymptomatic and in good health. He has been on thyroid medication intermittently.

PHYSICAL EXAMINATION: The patient is a husky 18-year-old Marshallese boy and appears to be in excellent health. Principal findings were related to the thyroid gland. The right lobe showed enlargement with a 3 cm. soft irregular nodule in the lower portion. The nodule was slightly tender to deep palpation. An enlarged lymph node was noted in the right submaxillary region but no other regional adneopathy was noted. The patient appeared to be euthyroid. Except for a slight ringworm infection over the shoulders the remainder of the physical examination was essentially negative.

LABORATORY & X-RAY DATA: Thyroid workup showed the following: the thyroxin level was 4.6 µg%, cholesterol 119 mg% (72% esters). Test for serum antithyroglobulin antibodies was negative: a thyroid scan (using ^{99m}Tc) showed a large cold area over most of the right lobe of the gland. Thyroid uptake of ^{131}I uptake was in the lower

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range of normal. Following TSH administration (10 units daily for 3 days) the thyroid uptake of ^{131}I had showed a good increase. However the serum thyroxin level did not show any increase over the pre TSH stimulation level. X-ray of the chest showed extrinsic pressure on the trachea, probably from thyroid enlargement. The basal metabolic rate was -8. The remainder of the laboratory findings were essentially negative.

HOSPITAL COURSE: His hospital course was uneventful. The following thyroid experts were consulted: Dr. J.E. Rall and Dr. J. Robbins of NIH, and Dr. B. Colcock of Lahey Clinic, Boston. The consensus was that surgical removal of diseased thyroid tissue was indicated. The patient was transferred to the New England Baptist Hospital in Boston on August 18, 1968 and under general anesthesia the thyroid gland was explored on August 20th. The gland was found to be grossly multinodular with many cystic areas and a subtotal thyroidectomy was performed. Microscopic examination by Dr. W.A. Meissner of the New England Deaconess Hospital resulted in a diagnosis of "adenomatous goiter". Recovery from surgery was uneventful and the patient was transferred back to this hospital on August 25th. The surgical wound healed nicely and the patient was placed on L-Thyroxin (Synthroid, 0.3 mg. daily). He was discharged from the hospital on August 30, 1968 to travel back to his home in the Marshall Islands.

FINAL DIAGNOSIS: Adenomatous goiter.

DISCHARGE MEDICATION: To continue thyroid medication for life. The patient was given ample hormone supply to last him for approximately a month. A note was sent to the Marshallese practitioner who will be in charge of the patient stressing the importance of continued thyroid medication.

Robert A. Conard

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DISCHARGE SUMMARY

NAME	UNIT NO.
Rongelap 54	8-18-90 R
PAVILION 1	OPD

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ADMITTED: AUGUST 4, 1968 DISCHARGED: AUGUST 30, 1968

This 15-year-old Marshallese boy was admitted to this Hospital for evaluation of a nodular thyroid gland resulting from exposure to radioactive fallout in 1954.

HISTORY OF PRESENT ILLNESS: In 1965 the patient's thyroid revealed a slight diffuse enlargement. In 1966 more definite enlargement with small nodules in the left lobe and isthmus were detected. He was placed on L-thyroxine treatment, but he has been very inconsistent in taking the drug. The 1967 and 1968 examinations revealed that the nodules had enlarged, the one in the left lobe being about 2 cm. in diameter. The nodules were firm but not hard and not tender. Though some exposed Rongelap children have shown some degree of hypothyroidism and growth retardation this boy has appeared euthyroid with normal growth and development. His serum thyroxin level (T-4) over the past three years has been 4.3, 4.8 and 4.6 µg% and his serum cholesterol 98 mg%.

The patient was 1 year of age at the time of exposure. He received an estimated 175 rads of whole body gamma irradiation, heavy irradiation of the skin and internal absorption of radioisotopes. The thyroid gland received about 700-1400 rad from radioiodines absorbed plus 175 rad of gamma radiation. He developed marked "Beta burns" of the skin. The lesions cleared however without complications within several weeks. He had slight transient epilation of the scalp and cyanotic changes in the fingernails also. During the first six weeks he developed a leukopenia and a platelet depression. However he never showed any clinical evidence of the radiation syndrome and his blood counts approached normal by one year. Yearly anthropometric measurements and skeletal age studies have revealed normal growth and development and he has always appeared to be euthroid (substantiated by normal PBI's and serum cholesterol levels). Since examined in March 1968 the patient has been asymptomatic and in good health. He has taken his thyroid medication intermittently.

PHYSICAL EXAMINATION: This 15-year-old Marshallese boy appeared to be well-nourished and of normal size for his age. Examination of the thyroid gland revealed that the left lobe was enlarged with multiple soft nodules palpable. A few small nodules were believed to be present also in the right lobe. A few small anterior cervical lymph nodes were palpable. The patient appeared to be euthyroid. There were no other no notable findings on physical examinations other than a mild tenia versicola infection of the face, arms and trunk.

LABORATORY & X-RAY DATA: Thyroid workup: serum thyroxin 4.9 µg%, cholesterol 145 mg%, (75% esters); BMR - 13, -10; serum antithyroglobulin antibodies negative. Thyroid scan using I.V. ^{99m}Tc showed large defects along the lateral aspects of the left lobe ^{131}I uptake was normal and a good response was obtained to TSH stimulation (10 units, I.M. daily for 3 days); thyroxin level increased from 4.9 to 6.3 µg%.

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Other: Chest x-ray and EKG negative. Other laboratory findings were negative except for a few WBC in the urine and the presence of the whipworm parasites in the feces.

HOSPITAL COURSE: A consultation was held with Drs. J.E. Rall and J. Robbins of NIH and Dr. B. Colcock of the Lahey Clinic. The consensus was that the diseased thyroid tissue should be surgically removed. The patient was transferred to the New England Baptist Hospital in Boston August 18. Dr. B. Colcock carried out a subtotal thyroidectomy removing thyroid tissue containing many nodules and cysts. Dr. W.A. Meissner of the New England Deaconess Hospital examined the tissues and his pathological diagnosis was "adenomatous goiter" and a nodule of oxyphilic cells. Recovery from surgery was uneventful and the patient was transferred back to this hospital on August 25. He was placed on thyroid hormone therapy. He remained asymptomatic with satisfactory healing of the surgical wound and was discharged to return to his home in the Marshall Islands on August 30, 1968.

FINAL DIAGNOSIS: Adenomatous goiter with nodule of oxyphilic cells.

DISCHARGE MEDICATION: To continue thyroid hormone therapy for life. A letter was sent to the Marshallese practitioner in charge of this patient stressing the importance of continued thyroid hormone treatment.

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DISCHARGE SUMMARY

NAME	UNIT NO.
Rongelap 70	8-18-8? R
PAVILION 1	OPD

ADMITTED: AUGUST 4, 1968 DISCHARGED: AUGUST 30, 1968

This 29-year-old woman was admitted to this Hospital for evaluation of a mass in the left region of the neck discovered during the 1968 annual medical survey at Rongelap Island.

HISTORY OF PRESENT ILLNESS: During the routine medical examination of the Rongelap people this patient was discovered to have a 1-2 cm. firm mass in the left side of the neck lateral to the thyroid gland near the insertion of the sternocleidomastoid muscle. The mass was non-tender, firm and did not move with the thyroid gland on swallowing. No lymph adenopathy in this region or elsewhere was noted. During the past two years the patient has complained of loss of appetite, weight loss and not feeling very well. (Her weight of 110 pounds on the past survey is 5-10 pounds below her usual weight. In 1954 her PBI was 5.7 µg% and in March of this year her serum thyroxine level (T-4) was 4.3 µg% and serum cholesterol 230 mg%. In view of the prevalence of thyroid abnormalities in the exposed Marshallese people and the suspicious nature of this patient's lesion it was considered wise to hospitalize her for full evaluation.

The patient was 15 years of age in 1954 at the time of the fallout accident. At that time she was one of a group of 18 that was away on a nearby atoll (Alingnae). This group received a whole body gamma dose of 70 rads about half the exposure of the other Rongelap people. Before the accident she was apparently healthy with a history of only occasional headaches. Following the accident only a few transient effects of exposure were noted: (a) very slight fallout lesions on the back of her neck at three weeks post exposure with some cyanotic changes in her fingernail beds, both of which cleared up within a few weeks; (b) and slight transient leukopenia . It was estimated that the dose to her thyroid gland was about 55 rads from radioiodines absorbed plus 70 rads of gamma radiation.

Following these early findings the patient remained generally in good health. No serious illnesses or injuries were noted. Growth and development was normal except for slight immaturity of sexual development (sparse pubic and axillary hair and small breasts). Menarche was at 14 years of age followed by normal menstrual periods. She has had four pregnancies, her first (1958) resulted in a stillbirth following a breech delivery due to "contracted pelvis": the second (1963) resulted in a normal delivery and baby. She has since had another stillbirth and a miscarriage. From the time of the first examinations she has remained somewhat anemic (not uncommon in Marshallese females) with hematocrit readings ranging from 28-33.

Since the survey examination of March 1968 she has noted occasional pain on swallowing, eating meals and drinking fluids. The pain is

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not steady and only occurs at intervals for a week or two. It has increased in frequency lately. She has not noted any recent weight loss and her appetite has been fairly good except that when she had the pain on swallowing she does not eat well. Bowel habits have been normal with normal stools. She has had nocturia (2-3 times a night) but no dysuria. Up until two weeks ago she had been taking her thyroid medication regularly.

PHYSICAL EXAMINATION: This 29-year-old Marshallese woman appeared well-nourished and asymptomatic. The thyroid appeared to be of normal size with no apparent nodularity. However, lateral to the thyroid beneath the left sternocleidomastoid muscle near its insertion was a fairly hard mass of about 2-3 cm. in diameter. The mass was slightly tender to deep pressure. The mass had about doubled in size since the last examination about 5 months previously. No regional lymph adenopathy was noted. Lymph nodes elsewhere were not notable. The breasts were normal. Examination of the chest was negative except for a soft systolic murmur noted over the aortic area. The abdominal examination was negative. Pelvic examination was negative. Thus there were no notable findings in this patient except referable to the neck region that would suggest a primary lesion.

LABORATORY & X-RAY DATA: The thyroid workup was negative in all respects with normal serum thyroxin and cholesterol levels. Serum antithyroglobulin antibody titer was negative. The BMR was +4, 0; thyroid scans showed a normal gland and thyroid uptake before and after TSH stimulation were normal. EKG was normal, chest x-ray showed the heart to be top normal in size. The lung fields were clear. Barium enema and G.I. series were normal except for a soft tissue mass in the left upper quadrant of the abdomen which was thought possibly to be splenic in origin. A skeletal survey revealed no evidence of metastatic disease. A liver and spleen scan following the injection of ^{99m}Tc-sulfur colloid revealed the spleen to be normal in size but a questionable defect in the liver was noted. A BAP smear was negative for malignant cells. Except for slight anemic tendency and the presence of whipworm parasites in the feces the remainder of the laboratory findings were generally negative.

HOSPITAL COURSE: A consultation was held with Drs. J.E. Rall and J. Robbins of NIH and Dr. B. Colcock of Lahey Clinic. The consensus was that the mass in the neck was quite likely to be of a malignant nature and surgical exploration was indicated. The patient was taken to the New England Baptist Hospital, Boston on August 18, 1968. On August 19th, Dr. B.P. Colcock surgically removed an oval well encapsulated mass weighing 6.8 Grams and measuring 2.5 x 2 cm. in diameter. Dr. W.A. Meissner of the New England Deaconess Hospital examined the tissues microscopically and reported the tumor to be a neurofibroma. Also present was a negative myelinated nerve and a lymph node showing chronic inflammation. Recovery from the surgery was uneventful and the patient was transferred back to this hospital on August 25. She was asymptomatic except for a slight soreness of the neck region. Her wound healed nicely except for a slight area of drainage at one corner. She was discharged from the Hospital on August 30th fit to travel back to her home in the

Marshall Islands.

FINAL DIAGNOSIS: Neurofibroma.

DISCHARGE MEDICATIONS: No medication necessary.

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DISCHARGE SUMMARY

NAME (Rongelap 15) UNIT NO. 08-22-57R

PAVILION 1 OPD

ADMITTED: Aug. 26, 1969 DISCHARGED: Sept. 22, 1969

This 22-year-old Marshallese lady was admitted to the Hospital for evaluation of a nodular thyroid gland believed to result from radioactive fallout in 1954.

HISTORY OF PRESENT ILLNESS: The patient was 7 years old at the time of exposure to fallout and she received an estimated 175 rads of whole body gamma radiation, beta radiation of the skin, and internal absorption of radioisotopes. The dose to the thyroid gland from radioiodines is estimated to have been between 500-1000 rads in addition to the 175 rads of gamma radiation. She developed transient mild beta burns of the scalp and slight epilation. Her leukocytes and platelets fell to about half normal levels within several weeks,but she had no signs or symptoms related to this depression. By one year she had largely recovered from these acute effects and her subsequent medical history is generally negative up to the present developments.

In 1967 several small nodules were palpated in the left upper lobe of the thyroid. In addition in 1968 a 1 cm. nodule was palpated in the right lobe. This past March,1969, examination of the thyroid revealed three distinct nodules, the largest one 2 cm. in the right-lower lobe and the other smaller in size in the left lobe. She has remained completely euthyroid and has shown no evidence of any retardation of growth and development. Her PBI and cholesterol levels have always been in the normal range. She had been taking thyroid-hormone medication fairly regularly since September 1965.

PHYSICAL EXAMINATION: The patient was alert, and appeared to be completely euthyroid with no major complaints. The findings on physical examination were largely confined to the thyroid gland. The three nodules were noted as described above except that perhaps the nodule in the left lobe of the gland had enlarged slightly. No lymphadenopathy was detectable. The only other finding on physical examination that might be noted was slight leukorrhea.

LABORATORY AND X-RAY FINDINGS: Thyroid studies revealed the following: PBI 8.1 µg.%, total iodine 8.2 µg.%, iodoprotein 2.6 µg.%, and T-4 5.3 µg.%; BMR -15; cholesterol 153 mg.%, esters 119 mg.%; thyroid scan showed a functioning nodule in the right lobe; uptake of radioiodine was normal but response to TSH stimulation was only fair;a test for thyroid antibodies was negative. The hemogram and other blood chemistry findings were normal. The chest x-ray was normal. Vaginal discharge was positive for monilia.

PREOPERATIVE HOSPITAL COURSE: The patient was asymptomatic

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and remained in good health. Her vaginal discharge improved on treatment with Mycostatin vaginal suppositories. On September 7, she was transferred to the Cleveland Metropolitan General Hospital.

SURGERY AT THE CLEVELAND METROPOLITAN GENERAL HOSPITAL: On September 8, the thyroid was explored by Dr. B. M. Dobbins. The thyroid was about normal in size but both lobes contained many fine nodules. The lower right lobe containing the larger nodule and a portion of the lower left lobe which was nodular were removed. Frozen sections indicated that the lesions were benign and extensive resection was not carried out.

POST SURGICAL HOSPITAL COURSE: The patient recovered satisfactorily from the surgical procedure. Since her return to Brookhaven, she has been a-symptomatic and her wound has healed nicely. A repeat thyroid scan showed that the functioning nodule noted prior to surgery had been removed. The patient was discharged on September 22 to return to the Marshall Islands.

DIAGNOSIS: 1. Adenomatous nodules of both lobes of the thyroid gland, some nodules showed prominent papillary component.

DISCHARGE MEDICATION: The patient is to continue taking thyroid-hormone medication under the supervision of the Marshallese health aide on her Island.

Robert A. Conard M. D.

RC:LR

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DISCHARGE SUMMARY

NAME (Rongelap 18) UNIT NO. 08-22-58R

PAVILION 1 OPD

ADMITTED: Aug. 26, 1969 DISCHARGED: Sept. 22, 1969

This 36-year-old Marshallese woman was admitted to this Hospital for evaluation of a nodular-thyroid gland.

The patient was 21-years of age at the time of exposure to fallout. She was in the more heavily exposed group, receiving an estimated 175 rads of whole body gamma radiation, beta radiation to the skin, and internal absorption of radioisotopes. Her thyroid gland received an estimated 160 rads from radioiodines absorbed, plus 175 rads from whole body exposure. She had signs of mild hematopoietic depression shortly after exposure but no signs of infection or bleeding and by one year her blood elements had recovered to normal levels. During the early period she also experienced mild transient beta burns to the skin.

HISTORY OF PRESENT ILLNESS: Her thyroid abnormality was detected for the first time this past March when a multi-lobulated mass in the left lower pole of the thyroid was noted. Up until this time she had always appeared to be euthyroid. A serum sample taken at that time of examination showed a T-4 level of 3.9 µg.%.

This is the third adult case in the more heavily exposed group that has developed thyroid pathology. The patient claims she had been taking her thyroid-hormone therapy regularly. Two of her sons, who were less than 10 years of age at exposure, have had excisions of benign nodules during the past two years.

PHYSICAL EXAMINATION: This mother of nine children appeared to be in excellent health. She appeared to be euthyroid. The only positive findings of significance in her physical examination were referable to the thyroid gland. Since the examination in March a discrete mass in the right upper pole was noted for the first time. The mass was hard and spherical. Less distinct nodulation was noted in the lower left lobe. No lymphadenopathy was noted.

LABORATORY AND X-RAY DATA: The thyroid studies revealed the following: PBI 9.2 µg.%, total iodine 9.2 µg.%, iodoprotein 2.3 µg.%, and T-4 6.2 µg.%; thyroid autoantibodies less than 1-16; thyroid scan showed a non-functioning nodule in the right upper lobe; uptake of radioiodine showed normal function of the gland; iodine uptake response to TSH stimulation, however, was poor. The ^{99m}Tc studies showed good trapping function; cholesterol 172 mg.%, esters 120 mg.%. Her BMR was +16. Positive stool examinations from ascaries and trichuris was the only other positive laboratory finding of significance. The high sedimentation rate and reversal of the A/G ratio is common to the Marshallese people. X-ray of the chest was negative.

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DISCHARGE SUMMARY

NAME (Rongelap 18) | UNIT NO. 08-22-58R

PAVILION 1 OPD

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PRE-SURGICAL HOSPITAL COURSE: Her hospital course during the preliminary examinations here at Brookhaven was uneventful.

SURGERY AT THE CLEVELAND METROPOLITAN GENERAL HOSPITAL, CLEVELAND OHIO: Dr. B. Dobbins performed thyroid surgery on September 10, 1969. A hard, white mass measuring about 8 mm. in diameter was found in the right upper lobe. A frozen section indicated malignancy and a complete thyroidectomy was done. In addition to the nodule just described the removed thyroid also showed a small focus of white-hard-tissue near the isthmus, and there was generalized granularity of the remainder of the gland. The patient was given 12.5 μc. of radioiodine preoperatively, and the malignant nodule was found to be nonfunctioning based on lack of radioiodine count. Lymph node involvement was not noted.

POSTOPERATIVE COURSE: The patient recovered satisfactorily from her operation. A slightly positive Chvostek sign was noted for several days. The patient was returned to this Hospital on September 14. During the week here before her discharge it was found that her serum calcium level was 9.2 mg.%. A thyroid scan following 350 μc. 131^{I} showed that the thyroidectomy had been complete except for one small questionable area 2.4 cm. to right and 3 cm. above the sternal notch. Her wound healed nicely, and she was discharged on September 22 fit for travel back to the Marshall Islands.

DIAGNOSIS:

1. Follicular carcinoma of the thyroid. Adenomatous nodules in remainder of gland; no lymph node metastasis.

DISCHARGE MEDICATION: The patient was advised that it was mandatory that she continue taking her thyroid and hormone medication always. The Marshallese practitioner, Dr. Riklon, who accompanied the patient here will personally supervise the continuation of this therapy. The patient will be examined again on the forthcoming medical survey in March 1970.

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DISCHARGE SUMMARY

NAME | UNIT NO.

Rongelap 36 | 08-22-60R

PAVILION 1 | OPD

ADMITTED: Aug. 26, 1969 DISCHARGED: Sept. 22, 1969

This 22-year-old Marshallese boy was admitted to this Hospital for evaluation of nodularity of the thyroid gland following exposure to radioactive fallout 15 years ago (1954).

HISTORY OF PRESENT ILLNESS: The patient was 8 years old at the time he was exposed to fallout and received an estimated 175 rads of whole body gamma radiation, beta radiation of the skin, and internal absorption of radioisotopes. It is estimated that his thyroid gland received approximately 500-1000 rads from radioiodines plus the gamma dose of radiation. He experienced early effects of radiation exposure including beta burns of the skin and transient leukopenia and platelet depression. He never showed any clinical evidence of the radiation exposure and by one year he had recovered from the acute findings. His subsequent medical history has been largely negative up to the present thyroid findings. He has had no retardation of growth and development and has always appeared to be euthyroid.

The first indication of thyroid abnormality was noted in 1966 when the gland was found to be slightly enlarged with a soft, 1 cm. diameter prominence in the lower right lobe with several small nodules noted on the left side also. His PBI and cholesterol levels were normal at that time. During the next two years continued enlargement of the nodules of the thyroid were noted and the T-4 level had reduced 2.6 µg.%. Examination this past March (1969) revealed that the mass in the lower right lobe was about 2-3 cm. in diameter with a cluster of smaller nodules in the left side believed to be attached to the trachea. The T-4 level was again 2.6 µg.%. However, he appeared to be euthyroid. The patient had two brothers who had benign nodules removed a year ago, and his mother had a malignant lesion removed several years ago from the thyroid.

PHYSICAL EXAMINATION: The patient was alert, healthy, and appeared to be euthyroid. Except for mild fungus infection of the skin the main findings were related to the thyroid gland. A soft prominence, 2-3 cm. in diameter, was noted in the right lower pole of the thyroid. Several lesser nodularities appeared to be present on the left side. No associated lymphadenopathy was detected.

LABORATORY AND X-RAY FINDINGS: Thyroid studies revealed: PBI 4.3 µg.%, total iodines 4.6 µg.%, iodoproteins 1.6 µg.%, and T-4 3.2 µg.%; BMR +3%; cholesterol 142, esters 107 mg.%. Thyroid scan showed a multinodular thyroid with rather marked enlargement of the right lower lobe which contained mainly nonfunctioning tissue. Iodine uptake was normal but response to TSH stimulation was poor. Hemogram and blood chemistry findings were generally negative. X-ray of the chest was normal. Thyroid autoantibodies showed less then 1-16 dilution reaction.

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DISCHARGE SUMMARY

NAME | UNIT NO. 08-22-60R

PAVILION 1 OPD

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PREOPERATIVE HOSPITAL COURSE: The patient was completely asymptomatic with good appetite and appeared to be completely euthyroid. On September 7 he was transferred to the Cleveland Metropolitan Hospital.

SURGERY CLEVELAND METROPOLITAN HOSPITAL: On September 8, the thyroid was explored by Dr. B. M. Dobbins. There was a discrete mass in the lower pole on the right measuring 2.5 cm. in diameter. Beneath this nodule there was a second mass measuring about 1.5 cm. in diameter. The left lobe contained a cluster of nodules. Both lobes were subtotally resected to remove the nodular areas with a pad of normal tissue around them. It was believed that the tissue left behind was "reasonably normal." It was noted that there were many fine vessels over the surface of the thyroid very much like one sees in a gland that has been therapeutically irradiated with radioiodine.

POST SURGICAL HOSPITAL COURSE: The patient's recovery from surgery was rapid and uneventful. He was transferred back to this Hospital on September 10. He remained completely asymptomatic and his wound healed nicely without complications. The repeat thyroid scan showed post operative absence of the inferior portions of the right and left lobes of the thyroid gland. The patient was discharged on September 22 to travel back to the Marshall Islands.

DIAGNOSIS: 1. Adenomatous nodules of both lobes of the thyroid gland. Degenerating follicular adenoma.

DISCHARGE MEDICATION: It was impressed upon the patient that it was imperative that he continue taking his thyroid-hormone medication from now on. He will be under the supervision of the local Marshallese practitioner in the Islands. The patient will be seen again on the forthcoming survey in March 1970.

Robert A. Conard

Robert A. Conard M. D.

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DISCHARGE SUMMARY

NAME | UNIT NO. 08-22-56R

(Rongelap 72)

PAVILION 1 OPD

ADMITTED: Aug. 26, 1969 DISCHARGED: Sept. 22, 1969

This 21-year-old Marshallese girl was admitted to this Hospital for evaluation of nodularity of the thyroid gland believed to result from radioactive fallout exposure in 1954.

HISTORY OF PRESENT ILLNESS: She received an estimated 175 rads of whole body gamma radiation, radiation of the skin, and internal absorption of radioisotopes. The thyroid received in the range of 500-1000 rads from radioiodines absorbed. She had acute symptoms of radiation effects including transient anorexia and nausea followed by hematopoietic depression the development of wide-spread beta burns over the scalp and neck regions. She recovered from these effects during the first year of post exposure. During the years following her exposure she has remained in generally good health and has had two normal deliveries of healthy children, the last occurring in July. She has always appeared to be euthyroid with normal PBI and cholesterol values. In 1965 a small 3 mm. nodule was discovered in the right lower lobe of the thyroid. In the following two years, however, the nodule was not noted. During the survey this past March 1969 she complained of a "lump in the throat" and a slightly irregular 1-2 cm. mass was noted in the lower left lobe. She had been very irregular about taking her thyroid hormone medication, since she has been on distant islands a large part of the time.

PHYSICAL EXAMINATION: The physical examination revealed an alert, apparently euthyroid Marshallese female of about her stated age. The examination was essentially negative except for findings related to the thyroid gland. A slightly irregular mass was noted in the lower part of the left lobe of the thyroid. It was fairly far posterior and seemed to be slightly adherent to the trachea. Another B-B sized mass was noted anteriorly near the junction of the isthmus.

LABORATORY AND X-RAY FINDINGS: Thyroid studies revealed: PBI 5.3 µg.%, total iodine 5.5 µg.%, iodoprotein 0.8 µg.%, and T-4 4.2 µg.%; BMR +3.5; cholesterol 187 mg.% and esters 136 mg.%. Thyroid autoantibodies showed less than 1-16 dilution reaction; scan showed a multinodular gland with slight enlargement of the left lobe; thyroid iodine uptake was normal but the response following TSH stimulation was poor. X-ray of the chest was negative. The only other laboratory finding of significance was the presence of ascaries and trichuris in the stool specimens.

SURGERY AT THE CLEVELAND METROPOLITAN GENERAL HOSPITAL: The patient was transferred to the Cleveland Metropolitan Hospital on September 7. On September 9 Dr. Brown Dobbins carried out thyroid surgery. The gland contained several nodular areas some of which on frozen section appeared to be adenomatous lesions but others on frozen section appeared to be

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malignant including involvement of two closely adjacent lymph nodes and several distant lymph nodes. In view of the metastizing nature of this malignancy on the left side, radical dissection was carried out removing all lymph nodes on that side of the anterior neck. No enlargement of lymph nodes was seen on the right side. A complete thyroidectomy was done carefully preserving the parathyroid glands.

POSTOPERATIVE HOSPITAL COURSE: The patient recovered satisfactorily from the operation but with some degree of edema of the subcutaneous tissues in the left side of the neck and lower jaw and a positive Chvostek for several days. She was returned to this Hospital on September 10. On arrival here there was slight drainage of the lower part of the incision of the neck which gradually reduced with daily care. The edema of the left side of the neck along with slight tenderness gradually reduced though at the time of her discharge from the Hospital on September 22 there was still some edema and discomfort on the left side of the neck. The wound had healed nicely except for a small granulating area in the lower part of the incision. Her serum-calcium level rose from 7.1 mg.% at the time of her return to 7.9 mg.% just before her discharge. The positive Chvostek noted earlier had disappeared at the time of departure. A repeat scan and radioiodine uptake study (following 350 mCi ^{131}I) showed almost complete removal of the entire thyroid gland. However, scintophotos revealed a slight remnant of thyroid tissue in what had been the lower-right pole region. At the time of surgery the patient had been given a blood transfusion. However, it was noted on her return that she was slightly anemic. Therefore, she was placed on Ferrous Sulfate treatment.

DIAGNOSIS:

1. Mixed follicular-papillary carcinoma of the thyroid with localized metastasis to cervical lymph nodes. Multiple macrofollicular adenomatous nodules were also present.

DISCHARGE MEDICATION: The mandatory continued treatment with thyroid hormone was impressed on the patient. Dr. Riklon, the Marshallese practitioner who accompanied the patient here will supervise her continued medication and on her return to Kwajalein will have the patient seen by the Head of the Kwajalein Hospital for a checkup. He has been requested also to check her serum calcium from time to time to be sure that it returnes to normal.

Robert A. Conard

Robert A. Conard, M. D.

RC:LR

BROOKHAVEN NATIONAL LABORATORY
HOSPITAL of the MEDICAL RESEARCH CENTER
UPTON, NEW YORK

DISCHARGE SUMMARY

NAME	UNIT NO.
Utirik 2229	08-22-59R
PAVILION 1	OPD

ADMITTED: Aug. 26, 1969 DISCHARGED: Sept. 22, 1969

This 34-year-old Marshallese lady from Utrik Island was admitted to this Hospital for evaluation of a nodular lesion of the thyroid gland suspected of being malignant.

HISTORY OF PRESENT ILLNESS: The patient was exposed to a small amount of fallout on Utrik Island during the time of the accident in 1954. It was estimated that her thyroid gland received only about 13 rads from radioiodines absorbed plus 14 rads from whole-body gamma radiation. There were no detectable effects related to radiation exposure. The patient has only been seen several times during the past 15 years, and her medical history during this period reveals very little of significance. When she was examinaed this past March, she was found to have a single, firm, smooth, freely movable mass of the right lobe of the thyroid. No cervical lymphadenopathy was noted. She apparently had been completely euthyroid though there is no record of any PBI or cholesterol tests. During the past several months she has noted some pain in the lower front of the neck region when she coughes and a slight hoarseness in her voice.

PHYSICAL EXAMINATION: The patient is somewhat obese and appeared slightly older than her stated age. Aside from thyroid findings, the physical examination was generally negative except for slight errosion of the cervix. The thyroid gland was found to be visibly enlarged and a firm to hard nodular enlargement of the right lower lobe, several cm. in diameter was palpated. The mass was movable and nontender. Th e trachea was thought to be slightly shifted to the left. No lymphadenopathy was noted.

LABORATORY AND X-RAY FINDINGS: Thyroid findings revealed: PBI 5.8 µg.%, total iodines 5.8 µg.%, iodoproteins 1.4 µg.%, and T-4 3.4 µg.%; BMR -25; cholesterol 188 mg.%, esters 148 mg.%; thyroid autoantibodies showed less than 1-16 dilution reaction; thyroid scan showeda nonfunctioning nodule in the lower right pole; the gland gave good ^{99m}Tc response after TSH stimulation but no increase in iodine uptake response. The chest x-ray revealed slight shift in the trachea to the left, presumably resulting from thyroid pressure. Stool examinations revealed trichuris. Urinalyses showed moderate number of RBC and WBC.

PREOPERATIVE HOSPITAL COURSE: Soon after hospitalization, the patient had a bout of upper respiratory infection with slight fever and cough which cleared up within several days. In view of the positive urinary findings Gantrisin treatments were started. Later urine samples were noted to be free of RBC and WBC. On September 7 the patient was transferred to the Cleveland Metropolitan General Hospital.

SURGERY AT THE METROPOLITAN GENERAL HOSPITAL: On September 8, the thyroid was explored by Dr. Brown B. Dobbins. A total right lobectomy was performed removing an encapsulated nodular mass about 3-4 cm. in diameter whose cut surface was white with fine granular

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character. Two additional pea-sized nodules adjacent to this tumor were also included in the lobe that was removed. In addition an exploration of the retrothyroidal lymph node area as well as the anterior mediastinum was done but no lymph node involvement was noted. The entire isthmus was removed. The left lobe was left intact.

POST SURGICAL HOSPITAL COURSE: The patient's convalescence was uneventful, and she was transferred back to this hospital on September 10. Her subsequent course showed uncomplicated healing of her wound. A repeat scan of the thyroid showed absence of the right lobe. She was started on thyroid hormone therapy and appeared to be in excellent condition at the time of her discharge on September 22.

DIAGNOSIS:

1. Follicular carcinoma with capsular invasion and minimal blood vessel invasion.

DISCHARGE MEDICATION: The patient was informed that she would have to take thyroid medication continously from now on. She was given a supply of Synthyroid tablets and was told to notify us when she ran low on her supply. She will be checked again on our forthcoming medical survey in March.

Robert A. Conard

Robert A. Conard, M. D.

RAC:LR

Appendix 6

Marshall Island Radiochemical Urine Analysis 1967-1969

Rongelap Island

Subject #	Age	Sex	Year	Vol.(ml)	mgCa/liter	pCi ^{90}Sr/liter	nCi ^{137}Cs/liter
1	69	F	1968	1070	15	1.5	1.0
2	17	M	1968	360	10	4.4	8.6
6	15	M	1967	700	19	2.6	3.2
			1968	650	5	1.3	1.1
8	16	F	1967	180	29	6.3	2.0
9	37	M	1967	1600	75	1.3	1.0
			1968	1400	121	2.6	1.0
11	65	M	1967	790	125	2.7	0.7
			1968	1800	13	1.0	1.2
14	40	F	1968	1720	46	5.2	2.6
			1969	1460	200	15.0	11.0
15	22	F	1967	650	55	9.0	2.5
			1969	820	20	4.4	3.2
16	54	M	1967	810	221	4.0	1.5
17	18	F	1967	340	29	6.3	7.7
			1968	525	13	4.2	7.8
23	19	M	1967	400	26	0.4	0.2
27	41	M	1967	1160	58	2.6	2.2
34	60	F	1967	480	152	5.0	3.3
			1969	1080	98	4.0	3.4
40	44	M	1968	730	124	4.4	1.8
			1969	1400	170	3.8	1.9
41	59	M	1967	1600	25	0.8	0.3
			1969	960	94	3.7	2.7
47	23	M	1969	1150	88	2.3	1.5

Subject #	Age	Sex	Year	Vol. (ml)	mgCa/liter	pCi ^{90}Sr/liter	nCi ^{137}Cs/liter
48	21	F	1967	460	118	15.0	4.7
			1969	500	47	8.6	5.8
49	30	F	1969	300	120	5.0	4.6
53	23	F	1967	820	125	6.3	1.1
			1968	1100	117	6.4	3.8
			1969	840	130	7.3	4.0
64	45	F	1968	1680	38	0.8	0.5
66	45	F	1969	1460	69	2.3	2.0
73	33	M	1967	780	202	5.4	5.1
			1969	1720	120	2.8	1.0
80	60	M	1968	1220	89	1.7	0.9
			1969	1460	170	1.9	0.9
805	15	F	1967	160	36	5.8	4.9
			1968	620	31	6.4	4.1
814	17	M	1968	1620	49	5.2	3.4
816	19	F	1969	1160	63	3.1	2.5
830	30	M	1969	880	260	1.9	0.5
833	36	M	1967	1100	162	4.5	2.5
840	39	M	1968	1180	149	12.0	1.1
			1969	1130	230	14.0	2.5
845	39	M	1967	980	210	3.8	2.0
851	60	F	1969	620	180	3.9	4.0
856	69	M	1968	1745	56	4.2	3.7
864	43	M	1968	1000	213	5.2	3.3
865	36	F	1968	780	20	2.4	2.3
882	36	M	1967	1400	46	0.9	0.5
			1969	1000	94	1.0	0.7
900	12	F	1967	710	23	6.4	3.7
914	34	M	1967	660	106	7.7	3.0
			1969	940	80	5.1	3.5
915	72	M	1969	220	120	3.7	5.9

Subject #	Age	Sex	Year	Vol.(ml)	mgCa/liter	pCi ^{90}Sr/liter	nCi ^{137}Cs/liter
928	56	F	1967	550	81	3.6	2.4
			1968	980	26	1.5	0.6
932	34	F	1967	720	32	1.8	2.0
945	44	F	1968	90	46	1.8	2.2
			1969	560	74	2.1	1.8
956	60	F	1968	1760	45	3.1	1.8
959	20	F	1968	1020	256	15.0	4.9
966	37	M	1968	180	108	4.4	3.2
			1969	520	170	3.1	4.4
1001	35	F	1969	280	110	7.8	6.8
1049	9	M	1967	800	153	5.6	2.0
1050	33	F	1969	330	320	2.5	1.0
1502	30	F	1967	630	101	7.6	4.4
1529	23	M	1969	1720	32	0.8	1.1
				Kili Island (Bikini People)			
1			1969	480	140	0.35	0.16
2			1969	800	33	0.32	0.16
3			1969	1080	50	0.18	0.07
4			1969	400	82	0.38	0.50
5			1969	220	140	0.82	0.45
6			1969	800	93	0.32	0.45
7			1969	620	100	0.29	0.27
8			1969	380	61	0.34	0.10
9			1969	340	120	0.53	0.76
10			1969	440	64	0.20	0.39
11			1969	260	110	0.15	0.28
12			1969	340	46	0.38	0.36
13			1969	100	140	1.70	0.56
14			1969	600	100	0.73	0.23

APPENDIX 7

INDIVIDUAL HEMATOLOGICAL FINDINGS 1967

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein(g)
				Rongelap Exposed Males, Age 13-15							
2	222	7.48	3.85	3.22	0.07	0.26	0.70	58	412	12.2	7.2
3	307	10.80	4.32	4.00	0.11	1.94	4.30	36	343	9.5	6.5
5	335	8.05	5.51	2.13	0.08	0.24	0.80	37	358	10.7	7.5
54	180	5.89	2.77	2.00	0	1.12	0	37	370	10.1	6.5
85*	216	7.83	4.74	2.58	0.16	0.35	0	39	438	10.7	7.4
Mean	252	8.01	4.23	2.79	0.08	0.78	1.16	41.4	384	10.6	7.0
				Allingnae Exposed Males, Age 13-15							
6	205	6.85	2.06	4.32	0	0.41	0.70	36	384	9.5	6.8
				Rongelap Exposed Females, Age 13-15							
33	279	5.82	3.31	2.27	0.06	0.12	0	41	437	12.2	7.8
65	215	8.17	3.43	2.70	0	2.00	0.40	38	396	10.4	7.1
86*	211	14.70	10.58	2.50	0.15	1.47	0	39	414	10.1	7.5
Mean	235	9.56	5.77	2.49	0.07	1.20	0.13	39.3	416	10.9	7.5
				Allingnae Exposed Females, Age 13-15							
8	206	6.02	3.07	2.35	0.06	0.54	0	36	402	9.5	7.5
				Rongelap Exposed Males, Age >15-40							
9	170	7.28	3.57	3.06	0	0.58	0.70	44	414	10.4	7.0
10	264	6.22	---	---	---	---	---	47	497	14.4	8.5
19	207	7.57	3.94	3.07	0.04	0.45	0.80	45	520	12.5	7.5
20	255	5.54	3.30	1.72	0.11	0.36	0.60	50	513	14.8	7.5

*Exposed in Utero

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein(g)
23	226	7.39	2.81	3.14	0	1.44	0	44	479	11.6	7.6
27	185	8.08	4.48	3.07	0.08	0.44	0	41	410	12.5	7.3
32	178	10.00	3.10	4.40	0.10	2.40	0	42	399	10.0	7.0
35	310	-4.76	---	---	---	---	---	53	510	15.2	7.4
36	212	6.73	3.50	2.69	0.13	0.27	1.30	45	512	14.0	8.4
37	219	6.25	2.38	2.81	0.03	1.00	0	45	437	13.2	7.6
47	198	8.12	5.52	2.15	0	0.45	0	47	452	14.4	7.0
73	232	5.90	3.10	1.71	0.12	0.32	0	41	485	12.5	7.0
77	223	5.31	3.24	1.96	0	0.11	0	47	470	11.9	7.8
Mean	222	6.86	3.54	2.71	0.05	0.71	0.31	45.5	469	12.9	7.5
				Ailingmae Exposed Males, Age>15-40							
44	205	11.30	6.95	3.84	0.23	0.28	0	43	496	10.7	7.5
				Rongelap Exposed Females, Age>15-40							
12	254	5.41	2.49	2.84	0.03	0.05	0	41	442	12.2	7.4
14	172	6.50	4.10	2.15	0.07	0.20	0	36	378	11.0	7.4
15	282	5.72	3.40	2.12	0	0.20	0	37	386	10.4	7.2
17	208	6.11	3.15	2.51	0	0.46	0	33	414	8.6	8.5
18	266	11.30	8.53	1.86	0.23	0.68	0	39	426	11.0	7.4
21	255	6.80	2.53	2.97	0	0.62	0.60	39	399	10.1	7.3
22	218	6.32	3.95	1.99	0	0.38	0	37	372	11.3	7.0
24	236	5.34	2.40	2.16	0.13	0.61	0.30	40	427	11.0	7.5
39	314	8.25	4.33	3.26	0.08	0.50	0.80	41	410	11.6	7.5
42	195	6.57	2.63	3.35	0.07	0.53	0	36	344	8.9	7.4
49	321	6.63	2.88	3.12	0	0.66	0.30	36	395	10.7	7.0
61	340	8.91	5.57	2.90	0.08	0.36	0	38	402	10.1	8.0
72	245	5.02	1.56	3.16	0.05	0.25	0	36	360	11.3	8.0
74	286	12.90	7.61	3.35	0.26	1.68	0	46	472	12.2	9.0
75	344	11.60	4.93	3.19	0	3.36	0	45	429	11.9	8.5
Mean	262	7.56	4.00	2.73	0.07	0.70	0.13	38.7	404	10.8	7.7

Pt. #	Plat (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein(g)
			Ailingnae Exposed Females, Age >15-40								
48	268	6.30	2.02	4.00	0.03	0.09	0.90	35	418	8.3	8.3
53	240	7.95	3.70	3.10	0.08	0.99	0.80	41	440	11.9	8.5
70	330	3.96	1.94	1.80	0.06	0.16	0	24	431	5.3	6.6
81	149	5.80	2.58	2.81	0.03	0.38	0	36	416	9.5	7.3
Mean	247	6.00	2.56	2.93	0.05	0.41	0.85	34.0	426	8.7	7.7
			Rongelap Exposed Males, Age >40								
4	204	7.06	1.73	4.73	0.07	0.28	1.80	45	427	12.8	7.7
7	151	5.77	2.39	3.00	0.12	0.20	0.60	37	388	11.0	7.4
11	218	4.70	2.33	1.79	0.09	0.49	0	38	358	11.0	7.0
40	351	8.34	3.88	3.92	0	0.54	0	44	440	11.9	6.5
68	225	4.69	2.49	1.88	0.09	0.23	0	44	414	11.3	6.7
79	109	4.86	2.41	2.24	0.10	0.12	0	48	473	11.3	7.1
80	288	5.74	2.55	2.84	0.08	0.20	0.60	45	457	11.3	7.4
82	237	8.08	5.29	2.22	0.16	0.40	0	41	431	9.2	6.9
Mean	223	6.16	2.88	2.83	0.09	0.31	0.38	42.8	424	11.2	7.1
			Ailingnae Exposed Males, Age >40								
16	221	4.65	2.77	1.88	0	0	0	45	560	11.6	6.6
41	194	4.55	1.98	1.96	0.05	0.21	0	42	438	12.8	7.1
Mean	207	4.66	2.38	1.92	0.03	0.11	0	43.5	499	12.2	6.8

Pt. E	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein(g)
Rongelap Exposed Females Age>40											
34	186	6.59	1.68	3.62	0.13	0.49	0	58	349	10.1	7.0
58	213	4.70	2.07	2.54	0	0.09	0	40	358	11.0	7.2
60	217	10.00	3.45	5.05	0.05	1.25	2.00	38	352	10.4	7.7
63	217	5.00	1.98	2.75	0	0.27	0	41	437	10.4	6.9
64	301	7.69	5.69	1.46	0.08	0.38	0.80	38	409	11.3	8.0
66	207	5.73	2.09	3.44	0	0.20	0	40	393	11.6	7.5
71	217	8.37	4.31	3.18	0.08	0.80	0	43	411	11.9	7.3
78	290	5.44	2.04	3.05	0.03	0.33	0	43	379	11.6	7.9
Mean	231	6.69	2.91	3.14	0.05	0.48	0.35	42.6	386	11.0	7.4
Ailingnae Exposed Females, Age>40											
1	137	6.30	3.37	2.65	0.13	0.16	0	40	395	12.4	7.3
45	275	4.48	2.55	1.72	0.04	0.18	0	38	366	11.0	7.5
59	208	8.82	2.69	4.67	0.05	1.32	0.90	41	445	11.6	8.0
Mean	206.6	6.53	2.87	3.01	0.07	0.55	0.30	40.3	402	11.7	7.6
Male Children of Exposed Parent(s) Age<13											
88	207	6.78	---	---	---	---	---	58	418	11.6	7.1
89	228	5.06	2.38	2.23	0.05	0.35	0.50	38	409	11.6	6.5
90	282	5.20	2.24	2.65	0.05	0.16	1.00	36	386	8.3	6.5
91	292	9.62	4.81	3.85	0	1.01	0	38	354	10.7	7.8
93	308	7.47	3.25	3.77	0.15	0.30	0	37	407	11.3	7.3
96	445	8.01	3.64	3.48	0.08	0.88	0	36	399	10.1	7.2
97	218	13.70	8.29	3.15	0.27	1.99	0	38	401	10.4	7.1
104	261	15.40	9.32	4.16	0.15	1.62	1.50	37	414	10.4	7.5
110	214	11.20	6.10	3.53	0	1.46	0	39	500	10.1	7.4
111	417	7.20	3.71	3.10	0.07	0.32	0	55	418	8.9	6.6
113	300	10.00	6.80	2.25	0.05	0.90	0	40	492	11.0	
115	339	14.90	7.60	5.36	0	1.94	0	39	423	11.0	
116	206	8.48	3.82	4.07	0.08	0.68	0	38	407	11.0	7.4
118	249	11.40	4.67	4.73	0.06	1.94	0	39	452	10.7	

Pt.#	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein(g)
131	372	9.20	5.34	3.04	0	0.83	0	39	448	11.0	
132	410	7.31	4.13	2.81	0.07	0.29	0	31	399	9.5	
141	450	11.80	6.55	4.37	0.30	0.59	0	29	444	7.3	
142	357	12.50	4.63	5.31	0.19	2.38	0	33	411	9.5	
148	359	8.31	2.58	5.32	0.08	0.25	0.80	29	459	6.3	
150	377	14.00	3.78	7.84	0.14	2.24	0	33	465	8.0	
155	318	7.97	1.43	5.66	0.04	0.48	0	38	293	7.1	
157	534	10.60	2.07	7.42	0.53	0.37	2.10	37	411	9.2	
158	360	7.81	1.25	5.55	0.16	0.86	0	28	306	9.2	
Mean	326	9.74	4.47	4.26	0.11	0.99	0.27	37.6	414	9.7	7.1
				Female Children of Exposed Parent(s), Age<13							
92	226	8.99	4.50	3.33	0.18	0.90	0.90	39	402	9.8	8.0
94	279	10.70	5.83	3.85	0.11	0.91	0	39	452	11.0	7.4
100	227	7.31	4.39	2.85	0	0.07	0	34	324	8.3	7.1
101	310	11.40	5.07	5.42	0.11	0.80	0	38	407	12.2	8.0
103	325	7.05	2.89	3.45	0.14	0.56	0	37	425	10.4	6.0
105	382	8.88	3.69	3.91	0.13	1.15	0	42	425	11.0	
106	115	4.72	1.35	2.78	0.09	0.54	0	58	344	8.0	7.3
108	255	14.60	7.52	3.80	0.15	3.14	0	39	407	11.3	7.8
112	299	8.48	4.45	3.14	0	0.89	0	39	370	8.9	7.8
117	448	9.39	4.55	4.04	0.19	0.52	0.90	33	380	10.1	
119	332	13.30	5.25	5.72	0.13	2.06	1.30	40	376	12.8	
120	209	7.65	2.94	3.02	0	1.68	0.80	38	435	9.8	7.5
122	431	8.48	5.60	2.08	0.08	0.55	1.70	34	366	12.2	6.4
123	414	11.60	3.77	5.63	0.23	1.97	0	37	402	10.1	
124	323	6.43	3.79	2.48	0.16	0	0	37	418	9.8	
125	296	9.85	5.91	3.84	0.10	0	0	37	414	11.0	7.1
127	240	9.31	6.66	2.33	0.28	0.05	0	33	378	10.7	
128	370	9.65	5.36	3.72	0.10	0.48	0	43	462	12.5	
134	538	11.90	2.74	8.39	0	0.77	0	36	410	10.4	
135	562	15.00	8.93	4.05	0.23	1.80	0	37	400	11.6	
136	414	18.00	13.50	3.06	0.09	1.08	4.50	36	414	9.5	
137	247	7.72	4.59	2.70	0.08	0.27	0.80	34	470	9.8	
138	510	7.58	2.77	4.24	0.08	0.49	0	42	448	7.5	
139	519	12.10	3.63	6.29	0.12	2.06	0	32	441	7.5	
140	366	14.90	11.18	2.98	0	0.60	1.50		484		
143	414	9.39	4.74	4.51	0.09	0.05	0	29	503	7.1	
144	383	5.75	2.30	2.30	0.17	0.46	0	33	434	9.8	
145	435	15.0	3.55	7.85	0	3.70	0	36	395	8.9	
149	528	9.71	3.40	5.29	0.15	1.75	0	36	370	8.6	
152	498	8.64	3.37	4.49	0.09	0.60	0.90	34	509	11.0	
153	474	9.09	5.95	2.77	0.14	0.23	0	28	342	7.5	
154	377	10.60	2.70	7.10	0.32	0.48	0	30	390	8.6	
156	304	6.19	3.53	1.92	0.06	0.62	0.60	26	273	9.8	
Mean	361	9.98	4.86	4.04	0.12	0.95	0.42	36.4	408	9.9	

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein (g)
Rongelap Unexposed Males, Age 13-15											
813	250	6.12	2.23	3.64	0.06	0.18	0	39	410	11.0	6.7
921	215	8.10	3.32	3.32	0.16	1.30	0	34	401	11.0	7.1
931	286	8.44	3.50	4.22	0.08	0.51	1.30	41	460	11.3	7.3
981	520	7.64	3.44	3.29	0.08	0.76	0.40	40	429	10.0	7.5
1036	322	5.19	2.83	2.10	0.16	0.10	0	44	450	10.4	7.4
1052	212	6.59	4.55	1.78	0.13	0.13	0	41	407	11.0	7.3
Mean	301	7.01	3.31	3.06	0.10	0.50	0.28	39.8	426	10.8	7.2
Rongelap Unexposed Females, Age 13-15											
805	229	5.06	1.97	2.43	0.15	0.51	0	39	463	9.5	6.1
811	351	4.20	2.06	2.02	0.04	0.08	0	39	407	11.0	7.1
812	257	7.92	4.16	2.69	0.08	0.91	0.80	47	418	10.7	7.4
879	294	7.76	2.48	3.30	0.04	1.90	0.40	42	396	10.7	
911	208	7.54	4.83	1.89	0.08	0.75	0	33	376	10.4	7.2
937	287	8.31	4.20	3.57	0.08	0.54	0	35	412	10.1	7.5
955	288	6.27	3.07	2.82	0	0.38	0	38	402	11.9	7.7
980	313	13.80	8.76	3.66	0	1.38	0	42	427	12.2	7.6
988	230	6.50	2.89	2.70	0.03	0.81	0.70	39	393	11.0	7.0
996	234	8.66	4.59	2.94	0	1.13	0	35	346	9.5	7.7
Mean	269	7.60	3.90	2.80	0.05	0.84	0.19	38.9	404	10.7	7.3

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein(g)
Rongelap Unexposed Males, Age>15-40											
815	225	6.70	2.58	3.42	0.03	0.67	0	44	453	11.6	7.7
823	301	7.14	4.93	1.79	0	0.36	0.70	42	411	10.1	6.9
827	236	9.05	4.48	4.25	0	0.41	0	48	453	12.8	7.7
830	207	6.08	3.04	2.28	0.03	0.67	0	47	433	14.0	6.9
833	269	5.09	2.42	2.32	0.10	0.25	0	47	494	11.3	6.8
840	231	10.00	4.10	3.90	0.10	1.80	1.00	34	513	13.6	7.8
845	272	6.03	3.59	2.17	0.15	0.12	0	49	505	12.2	7.5
863	260	7.34	3.12	3.78	0	0.44	0	49	490	13.6	7.3
881	270	6.48	3.18	3.08	0.03	0.13	0.60	44	437	10.7	7.4
882	212	4.85	1.36	2.96	0.05	0.49	0	41	457	11.0	6.5
885	229	11.70	6.55	4.10	0	1.05	0	45	435	13.2	8.0
919	210	5.41	2.73	2.27	0.05	0.35	0	44	468	11.6	8.3
939	270	8.52	3.92	3.83	0	0.68	0.90	53	506	13.6	7.3
943	326	9.89	5.44	3.96	0.10	0.40	0	45	507	15.2	8.3
958	321	7.89	---	---	---	---	---	42	420	11.0	7.5
966	257	4.79	2.66	1.92	0.05	0.17	0	44	435	10.7	7.0
971	321	5.28	1.93	2.85	0.11	0.40	0	45	434	12.8	8.0
1033	226	7.10	2.84	3.44	0.11	0.71	0	43	465	13.2	7.6
1501	188	5.30	3.26	1.99	0	0	0.50	46	467	12.8	7.3
1519	336	6.20	3.07	2.79	0.12	0.16	0.60	48	468	12.8	6.8
1523	368	6.72	2.76	3.70	0.07	0.20	0	51	560	11.9	8.0
1524	227	6.71	3.86	2.48	0	0.37	0	50	450	10.7	7.5
1526	205	9.95	6.17	3.03	0.05	0.70	0	52	542	14.8	7.5
Mean	259	7.14	3.55	3.01	0.05	0.48	0.20	45.8	470	12.4	7.5

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein (g)
Rongelap Unexposed Females, Age>15-40											
829	223	6.25	2.81	2.50	0.06	0.81	0.60	34	331	10.4	8.6
835	487	9.35	4.63	4.02	0.28	0.42	0	40	468	11.6	7.5
841	253	6.21	4.16	1.61	0.25	0.19	0	35	364	9.8	6.7
865	323	9.15	7.14	1.78	0.14	0.09	0	36	365	9.8	7.0
867	316	8.92	5.31	3.12	0	0.49	0	40	426	11.3	7.5
888	239	9.98	6.19	2.30	0	0.50	0	40	390	10.4	7.9
891	237	8.03	5.66	1.85	0.08	0.44	0	46	433	13.2	
896	252	6.53	3.59	2.71	0.10	0.13	0	39	392	8.6	7.6
909	219	6.16	2.68	2.71	0	0.71	0	30	329	7.3	5.6
914	217	5.96	2.50	3.01	0.06	0.30	0.90	37	405	11.3	7.0
925	318	7.73	4.99	2.24	0.12	0.31	0	37	401	10.7	7.6
926	225	10.80	5.02	4.75	0.11	0.81	1.10	41	473	12.8	
932	300	7.14	3.36	2.93	0	0.82	0.40	39	420	8.9	7.8
934	246	9.65	6.18	3.09	0.29	0.10	0	40	426	10.7	7.5
938	210	6.88	4.68	1.82	0.03	0.34	0	35	376	8.9	
946	256	7.59	4.33	2.96	0	0.38	0	45	448	12.8	
950	315	10.00	3.20	5.55	0	1.20	1.00	41	433	11.9	7.9
951	342	7.72	4.01	3.51	0	0.27	0	37	496	11.6	7.4
959	227	6.44	3.16	2.74	0.16	0.39	0	41	414	11.6	7.5
960	298	11.40	6.73	3.65	0.23	0.80	1.10	40	450	10.4	8.5
993	246	8.93	5.22	3.26	0.09	0.36	0	38	414	11.3	6.9
1001	108	8.89	5.60	2.04	0.09	0.18	0.90	42	440	11.6	6.8
1035	325	7.74	2.21	5.19	0.08	0.19	0.80	42	414	11.0	8.3
1043	267	5.62	3.09	2.08	0.06	0.39	0	40	414	10.7	7.4
1050	275	5.42	3.14	2.11	0.05	0.11	0	37	376	11.0	7.5
1502	166	7.54	4.37	2.98	0.04	0.15	0	39	445	10.1	8.0
1520	233	6.81	3.44	3.17	0.07	0.14	0	41	396	11.3	7.6
1525	243	6.27	3.76	2.19	0.13	0.19	0	39	390	10.4	7.0
Mean	263	7.83	4.33	2.92	0.09	0.40	0.19	39.0	412	10.8	7.5

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein (g)
Rongelap Unexposed Males, Age >40											
850	228	4.73	2.67	1.66	0.05	0.35	0	43	425	11.3	7.7
853	246	7.04	4.51	2.34	0.14	0	0	45	402	12.5	7.8
855	205	6.19	3.84	1.80	0.06	0.50	0	37	358	10.7	7.6
856	239	4.75	2.04	2.14	0.05	0.52	0	41	400	10.7	7.4
860	234	6.52	3.55	2.09	0.20	0.68	0	34	289	8.0	7.2
864	241	6.42	2.18	3.92	0.06	0.26	0	47	475	11.9	7.3
868	127	6.39	2.65	3.07	0.13	0.48	0.60	45	453	11.6	7.5
878	275	7.19	5.07	1.76	0	0.36	0	41	420	10.7	8.0
884	225	7.16	5.94	1.15	0	0.07	0	40	391	11.3	6.6
897	227	7.12	3.74	2.78	0.07	0.53	0	41	379	11.6	7.9
915	341	4.26	1.77	2.17	0.04	0.28	0	43	402	11.6	7.3
935	212	5.42	3.96	1.84	0.05	0.11	0	44	389	11.9	6.2
944	211	6.29	3.27	2.52	0.06	0.38	0.60	48	473	14.0	7.7
947	259	9.84	7.33	1.87	0	0.64	0	40	401	10.4	7.5
961	268	7.67	4.03	2.65	0.08	0.84	0.80	44	456	13.2	7.9
969	267	9.72	6.61	2.96	0.05	0.10	0	48	465	11.0	7.7
975	210	4.34	2.60	1.69	0	0.04	0	44	423	11.6	6.5
1041	250	7.66	4.90	2.41	0.08	0.23	0	43	410	11.0	8.2
1515	252	6.81	4.12	2.32	0	0.31	0.70	41	368	11.0	8.0
1517	458	9.50	4.28	4.85	0.29	0.10	0	43	438	11.0	7.6
Mean	249	6.75	3.95	2.40	0.07	0.34	0.14	42.6	411	11.4	7.5
Unexposed Females, Age >40											
844	182	9.45	7.56	1.61	0.09	0.19	0	42	416	10.7	8.1
852	262	8.92	4.86	2.77	0.22	1.07	0	41	407	10.1	7.7
858	210	6.72	4.70	1.41	0.07	0.53	0	38	393	10.5	7.6
859	346	6.73	3.10	3.30	0.24	0.10	0	38	366	9.5	7.1
889	215	6.72	4.33	2.15	0	0.23	0	39	414	11.6	8.0
898	219	8.93	4.82	2.59	0.18	1.34	0	39	365	10.4	7.8
908	232	5.56	2.84	2.50	0.06	0.11	0	40	346	9.2	7.0
922	250	8.37	6.07	1.46	0	0.67	0.80	35	388	9.2	7.5
928	276	5.41	3.19	1.84	0	0.38	0	37	360	11.9	8.8
929	220	6.71	3.20	2.75	0.13	0.54	0	34	392	10.1	8.0
941	256	7.04	3.34	2.60	0.07	0.95	0.70	38	368	10.7	7.3
942	210	8.26	4.46	2.89	0	0.91	0	43	426	11.9	8.7
945	289	6.10	3.93	1.59	0	0.58	0	46	463	12.5	8.0
956	293	5.97	2.51	3.28	0	0.18	0	38	360	10.1	7.5
1042	264	4.63	2.64	1.92	0.07	0	0	40	381	10.4	7.6
Mean	248	7.03	4.11	2.31	0.08	0.52	0.10	39.2	390	10.6	7.8

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (&)	RBC (X10-4)	Hgb. (g)	Serum Protein(g)
			Male Children of Unexposed Parents, Age 13								
802	210	4.96	2.23	2.38	0.10	0.20	0.50	39	442	11.0	6.4
807	284	9.48	5.31	3.03	0.28	0.76	0.90	38	412	9.2	6.5
809	260	8.81	3.44	4.27	0	1.10	0	39	402	10.4	8.5
870	316	8.57	3.21	3.30	0.09	1.97	0	40	409	11.9	
904	237	10.90	7.25	2.62	0	1.04	0	39	414	11.0	7.6
924	267	12.80	5.50	4.61	0	2.69	0	36	444	11.6	7.1
1002	321	7.57	3.11	3.67	0.08	0.72	0	37	450	10.7	7.3
1004	473	14.20	9.09	4.33	0.07	0.57	0.70	36	346	9.5	
1010	202	6.52	2.77	3.39	0.06	0.23	0.60	35	381	10.1	7.0
1013	450	10.50	6.51	3.20	0.05	0.63	1.10	33	389	9.5	
1014	346	7.67	3.07	4.07	0.23	0.31	0	35	392	10.1	
1017	456	6.94	4.51	2.01	0.14	0.28	0	36	440	10.4	6.5
1028	547	8.54	5.38	2.56	0	0.00	0	34	445	10.1	7.4
1032	217	17.60	---	---	---	---	---	42	449	10.7	7.4
1037	274	5.86	4.13	1.35	0.18	0.18	0.30	34	345	8.9	
1038	426	13.80	4.00	4.76	0.21	4.69	1.40	34	393	8.3	
1039	387	9.56	6.55	2.29	0.19	0.53	0	37	316	11.0	
1046	265	7.39	2.14	4.58	0	0.67	0	32	423	9.5	
1047	485	7.74	4.06	2.79	0.39	0.50	0	37	402	11.6	
1049	348	8.48	3.73	4.41	0.08	0.25	0	36	416	10.1	7.8
1056	255	8.01	5.21	2.40	0.08	0.24	0	31	453	10.4	
1058	634	9.96	5.88	3.88	0	0.20	0	36	457	9.2	
1059	390	13.20	---	---	---	---	---	36	400	10.4	
1062	364	5.90	3.39	2.42	0	0.09	0	37	431	7.3	
1063	349	13.80	4.28	8.42	0.14	0.97	0	34	399	8.0	
1064	344	7.14	3.75	3.25	0.11	0.04	0	34	409	8.6	
1067	360	9.30	6.14	2.88	0.09	0.19	0	37	344	10.4	
1068	513	12.40	6.14	4.59	0.25	1.43	0	32	412	9.5	
1072	446	7.87	4.09	3.62	0	0.08	0.80	32	493	8.3	
1074	341	10.50	4.26	5.67	0.21	0.26	0	32	388	8.0	
1076	424	8.07	2.78	4.76	0.12	0.32	0.80	30	395	9.5	
1077	475	6.38	1.85	4.43	0.03	0.06	0	24	251	6.3	
1079	284	8.01	2.60	5.05	0.16	0.12	0.80	34	420	8.3	
1503	497	5.97	3.52	2.24	0.09	0.12	0	36	409	9.5	7.0
1504	358	7.61	3.69	3.73	0.08	0.11	0	36	416	9.8	
Mean	362	9.14	4.21	3.65	0.10	0.67	0.23	35.3	406	9.7	7.3

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neut. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Hgb. (g)	Serum Protein (g)
			Female Children of Unexposed Parents, Age < 13								
808	409	6.65	3.99	2.00	0	0.67	0	41	442	9.2	8.4
866	431	7.02	3.51	3.19	0.04	0.28	0	40	449	11.0	7.6
900	234	5.06	2.07	2.43	0.20	0.35	0	37	410	8.3	7.1
901	287	7.43	2.34	4.46	0.37	0.19	0.70	36	407	8.0	7.1
903	232	6.04	3.32	2.45	0	0.27	0	36	396	10.7	7.5
906	227	6.01	2.19	3.34	0	0.48	0	38	414	8.9	6.7
923	230	7.59	4.44	2.92	0.15	0.08	0	39	388	9.2	7.3
954	232	7.62	4.50	2.71	0.04	0.40	0	39	433	10.1	7.5
979	240	5.19	1.63	1.71	0.10	1.63	1.00	43	484	13.2	7.5
1019	202	9.14	4.43	3.70	0.09	0.91	0	33	354	10.7	6.9
1022	271	6.31	2.43	2.90	0.06	0.85	0.60	36	370	9.8	6.5
1025	323	9.42	5.46	3.34	0.24	0.28	0.90	40	411	11.6	7.5
1026	370	8.58	2.79	4.03	0	1.59	1.70	35	365	8.9	8.0
1029	349	6.13	3.43	2.33	0.12	0.25	0	36	402	8.0	
1031	386	8.04	5.43	2.25	0.08	0.28	0	35	389	10.1	
1034	334	8.21	4.31	3.57	0.25	0.08	0	38	440	10.7	
1057	593	11.10	3.83	3.77	0.06	1.39	0.60	35	355	10.6	
1065	418	10.50	4.88	4.46	0	1.05	1.10	36	405	10.4	
1066	389	6.70	2.91	3.38	0.07	0.34	0	37	393	9.5	
1069	381	8.59	3.95	4.21	0.09	0.34	0	32	472	7.7	
1070	347	6.46	2.55	3.20	0.26	0.45	0	36	410	10.7	
1075	504	16.30	---	---	---	---	---	35	431	9.2	
1078	633	6.62	1.52	4.30	0.07	0.73	0	24	313	7.1	
1080	376	11.60	6.21	4.29	0.35	0.87	0	30	368	9.2	
1516	235	7.78	3.58	3.58	0	0.62	0	38	384	10.0	7.0
1518	413	12.10	4.11	6.90	0.12	0.97	0	33	475	10.1	
1521	212	5.09	1.91	2.72	0.08	0.36	0	33	399	9.5	7.0
1522	465	9.29	1.63	6.87	0.14	0.65	0	34	450	8.9	
Mean	347	8.09	3.53	3.44	0.11	0.61	0.22	35.9	407	9.7	7.3

Individual Hematological Findings, 1968

Pt. #	Plat. (X10-3)	WBC (XI0-3)	Neutro. (X10-3)	Lymph (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein(g)
Rongelap Exposed Males, Age 14 - 40										
2		4.62	1.50	2.77	0.05	0.28	0.50	47		7.8
3		10.84	4.01	5.15	0.16	1.30	2.20	42		8.9
5		11.96	7.83	2.57	0	1.19	3.60	41		7.5
9		6.80	3.44	3.07	0.24	0.04	0.40	44		7.2
10		7.28	4.26	2.55	0	0.36	1.10	48		7.5
19		11.08	7.70	2.44	0	0.83	1.10	48		7.3
23		7.80	3.94	3.43	0.20	0.23	0	47		8.0
27		6.28	3.32	2.70	0.07	0.14	0	41		8.1
32		5.96	3.31	2.24	0	0.36	0.60	45		6.7
35		2.68	0.52	2.02	0	0.13	0	53		7.5
36		8.00	3.96	3.28	0.12	0.64	0	48		8.4
37		7.32	4.03	3.11	0.11	0.07	0	49		7.5
47										8.0
54		5.48	1.86	2.85	0.05	0.66	0.50	45		7.8
73		4.24	2.61	1.40	0	0.23	0	46		7.2
76		6.72	3.80	2.39	0.10	0.37	0.70	47		7.3
77		5.72	3.52	2.09	0	0.11	0	51		8.5
85*		10.24	4.10	5.02	0	1.13	0	40		7.4
Mean		7.24	3.75	2.89	0.12	0.47	0.63	46.0		7.7
Ailingnae Exposed Males, Age 14-40										
6		5.02	1.58	2.54	0.15	0.70	0	38		7.2
84*		6.60	1.82	3.86	0	0.92	0	37		7.6
Mean		5.81	1.70	3.20	0.08	0.81	0	37.5		7.4
Rongelap Exposed Females, Age 14-40										
12		6.40	4.03	2.18	0	0.19	0	39		7.2
14		4.52	2.83	1.60	0.05	0.09	0	39		7.9
15		4.88	2.07	2.61	0.05	0.15	0	39		7.5
17		7.40	4.40	2.41	0.04	0.56	0	33		8.3
18		8.52	5.92	2.00	0	0.43	1.70	36		6.5
21		7.60	3.81	0.76	0	0.91	1.10	38		7.9
22		5.08	1.17	2.92	0.08	0.91	0	41		7.8
24		5.52	3.53	1.16	0.05	0.28	0	37		7.5
33		8.04	3.86	3.26	0.04	0.80	0.80	43		8.4
39		7.20	3.71	2.59	0	0.90	0	42		8.0
42		8.48	5.98	2.20	0.04	0.25	0	31		7.3
49		9.16	5.22	3.57	0	0.23	1.40	35		7.7
61		6.60	3.40	2.97	0.17	0.07	0	45		8.3
67		7.38	4.98	1.59	0.08	0.74	0	41		7.5
72		7.88	4.73	2.76	0.08	0.32	0	38		8.5
74		10.48	6.92	2.10	0	1.47	0	39		7.7
75		9.24	5.41	2.59	0	1.20	0.50	40		6.7
86*								41		7.5
Mean		7.32	4.23	2.31	0.04	0.56	0.36	38.7		7.7

*Exposed in utero

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro (X10-3)	Lymph (X10-3)	Mono. (X10-3)	Eosin (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein(g)
Ailingnae Exposed Females, Age 14-40										
8		7.56	4.23	2.72	0	0.34	2.60	40		8.5
48		8.40	5.47	2.32	0.21	0.34	0.80	35		7.3
53		6.76	4.33	2.03	0.14	0.27	0	42		8.6
70		5.44	4.02	1.17	0.08	0.16	0	35		7.9
81		4.24	2.40	1.70	0.02	0.04	0.40	37		8.3
Mean		6.48	4.09	1.99	0.09	0.23	0.76	37.8		8.1
Rongelap Exposed Males, Age> 40										
4		7.44	4.09	2.90	0.07	0.37	0	49		8.3
11		6.40	3.86	2.09	0.03	0.39	0.60	42		7.4
40		7.16	2.51	2.11	0.04	2.43	0.70	41		6.6
68		5.33	3.92	1.15	0.11	0.11	0.50	40		6.9
79		4.70	2.66	1.65	0.09	0.31	0	43		7.2
80		8.26	3.84	3.55	0.17	0.66	0.40	43		7.8
82		8.22	5.30	2.30	0	0.62	0	42		7.7
Mean		6.79	3.74	2.25	0.07	0.70	0.31	42.9		7.4
Alingnae Exposed Males, Age>40										
16		4.68	2.11	2.11	0	0.47	0	48		7.9
41		7.82	4.22	3.17	0	0.39	0.40	45		7.3
Mean		6.25	3.17	2.64	0	0.43	0.20	46.5		7.6
Rongelap Exposed Females, Age>40										
34		4.84	1.77	2.81	0.05	0.22	0	38		7.7
58		5.84	2.45	3.33	0.06	0	0	37		7.5
60		6.92	2.25	3.88	0.03	0.62	1.40	46		8.0
63		7.10	2.63	3.98	0	0.50	0	41		7.0
64		5.38	2.80	2.26	0.05	0.27	0	36		7.6
71		7.28	4.26	2.33	0.07	0.55	0.70	45		8.0
78		7.65	4.13	2.83	0.15	0.54	0	40		8.0
Mean		6.43	2.90	3.06	0.06	0.39	0.30	40.4		7.7
Ailingnae Exposed Females, Age>40										
1		6.48	4.47	1.49	0.03	0.49	0	48		7.8
45		4.56	2.30	1.78	0.02	0.46	0	38		8.0
59		11.96	7.23	3.47	0.06	0	0	39		5.3
Mean		7.67	4.67	2.25	0.03	0.32	0	41.7		7.0

Individual Hematological Findings, 1969

Pt #	Plat. (X10-3)	WBC (X10-3)	Neutr. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. %	RBC (X10-4)	Serum Protein (g)
Rongelap Exposed Males, Age 15 to 40 years										
2	211	6.00	3.12	2.16	0.18	0.48	0.60	48	460	7.7
3	112	6.81	3.54	2.32	0.07	0.75	1.40	45	400	8.3
5	157	5.54	2.16	2.94	0.11	0.28	0.60	43	410	7.5
9	140	6.93	3.88	2.63	0.07	0.21	1.40	47	4.30	6.6
10	129	4.56	2.42	1.83	0109	0.23	0.0	51	380	8.4
23	-	6.40	2.37	3.52	0.19	0.26	0.60	-	-	7.7
32	-	7.16	3.87	2.72	0.21	0.36	0.0	56	-	-
35	140	5.40	2.27	2.86	0.05	0.22	0.0	56	510	7.1
36	160	8.81	4.14	4.14	0.18	0.35	0.0	49	440	8.5
37	114	6.01	3.07	2.64	0.12	0.12	0.60	46	410	7.5
54	-	6.28	3.14	2.51	0.0	0.44	1.90	43	-	-
73	132	4.96	2.28	2.18	0.05	0.40	0.50	52	480	7.1
76	105	6.03	3.26	2.41	0.06	0.30	0.0	49	350	7.5
85*	192	7.02	3.65	3.09	0.14	0.14	0.0	40	440	6.8
mean	145	6.28	3.08	2.71	0.11	0.32	0.50	47.3	428	7.6
Ailingnae Exposed Males, Age 15 to 40 years										
6	198	7.21	3.46	3.24	0.36	0.87	0.0	43	400	7.3
84*	220	4.34	1.30	2.74	0.04	0.26	0.0	37	340	7.2
mean	209	5.78	2.38	2.99	0.20	0.57	0.0	40.0	370	7.2

* Exposed in utero

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. %	RBC (X10-4)	Serum Protein (g)
Rongelap Exposed Females, Age 15 to 40 years										
12	178	3.63	1.89	1.45	0.11	0.18	0.0	40	360	7.1
14	189	7.69	3.85	2.92	0.31	0.54	0.8	35	320	7.2
15	244	5.45	2.45	2.40	0.27	0.27	0.50	38	440	7.2
17		6.12	2.51	2.75	0.12	0.73	0.0	36	-	-
18	132	5.09	2.75	1.83	0.15	0.31	0.60	39	280	7.4
21	180	5.18	2.17	2.64	0.05	0.21	1.00	41	360	7.9
22	107	3.84	1.23	2.34	0.08	0.19	0.0	40	280	7.7
24	120	4.72	1.56	2.74	0.19	0.19	0.50	43	310	8.4
33	255	7.17	3.01	3.66	0.21	0.14	1.40	40	380	8.0
39	226	8.46	3.30	4.23	0.08	0.68	1.70	43	340	8.5
42	189	8.54	3.93	4.01	0.09	0.43	0.90	35	290	6.7
47	134	9.23	3.51	4.80	0.37	0.46	0.90	50	430	7.8
61	271	8.15	3.68	3.83	0.16	0.49	0.0	43	490	7.8
65	227	6.29	2.39	2.90	0.06	0.88	0.60	40	420	6.9
67	-	6.24	3.12	2.75	0.06	0.31	0.0	34	-	-
72	291	6.90	2.35	4.21	0.14	0.14	0.70	34	300	7.5
74	214	7.83	3.37	4.07	0.16	0.24	0.0	49	420	9.4
75	-	5.72	3.15	2.12	0.11	0.29	0.60	-	-	-
86*	146	7.50	4.20	2.85	0.08	0.30	0.80	46	400	8.5
mean	194	6.52	2.86	3.08	0.15	0.37	0.60	40.3	364	7.8
Ailingnae Exposed Females, Age 15 to 40 years										
8	153	7.53	4.75	2.41	0.15	0.15	0.80	40	380	8.0
53	365	7.24	3.76	2.60	0.07	0.72	0.70	31	240	7.8
70	293	4.33	2.69	1.43	0.04	0.17	0.0	42	380	7.1
81	166	4.34	1.91	2.26	0.04	0.13	0.0	37	330	7.8
mean	244	5.86	3.28	2.18	0.08	0.29	0.38	37.5	333	7.7

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. %	RBC (X10-4)	Serum Protein (g)
	Rongelap Exposed Males, Age greater than 40 years									
4	126	7.99	3.68	3.68	0.16	0.32	1.60	48	640	8.4
7	199	4.92	2.61	2.02	0.10	0.20	0.0	42	380	7.9
11	180	4.31	2.16	1.90	0.09	0.17	0.0	34	260	7.2
16	179	4.17	2.00	1.96	0.08	0.12	0.0	49	560	6.7
27	158	8.21	3.28	4.18	0.16	0.49	0.0	43	320	7.4
40	235	7.88	3.31	4.10	0.16	0.32	0.0	41	400	6.3
68	236	6.19	3.59	2.10	0.06	0.31	1.20	46	430	7.1
77	243	12.89	6.45	5.29	0.64	0.52	0.0	47	520	8.8
79	97	5.46	2.35	2.78	0.22	0.11	0.0	51	460	7.4
80	179	6.41	2.24	3.59	0.13	0.32	1.30	47	430	6.9
82	155	5.08	2.33	2.34	0.10	0.25	0.50	43	390	7.1
mean	181	6.68	3.09	3.09	0.17	0.28	0.30	44.6	435	7.4
	Ailingnae Exposed Males, Age greater than 40 years									
41	138	4.59	2.30	1.98	0.14	0.18	0.0	43	440	6.6

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct (%)	RBC (X10-4)	Serum Protein (g)
Rongelap Exposed Females, Age greater than 40 years										
34	132	7.12	3.63	2.85	0.07	0.57	0.0	39	350	7.3
49	218	6.00	2.22	3.30	0.06	0.42	0.0	39	380	7.2
58	87	5.02	1.54	2.97	0.15	0.41	0.50	41	320	6.8
60	126	7.84	2.82	4.16	0.08	0.63	1.60	37	310	8.1
63	202	5.76	1.79	3.51	0.12	0.23	1.20	43	420	6.9
64	168	8.61	4.22	3.70	0.09	0.60	0.0	41	430	7.3
66	296	5.62	2.98	2.14	0.11	0.34	0.60	45	410	7.4
71	162	7.14	4.71	2.28	0.07	0.07	0.0	43	360	8.1
78	175	6.69	3.81	2.68	0.07	0.13	0.0	40	420	7.6
mean	174	6.64	3.08	3.07	0.09	0.38	0.90	40.9	378	7.4
Ailingnae Exposed Females, Age greater than 40 years										
1	177	7.79	4.37	2.49	0.16	0.62	1.60	41	460	7.9
45	165	5.88	3.41	2.12	0.18	0.12	0.60	35	340	8.0
48	176	4.83	2.95	1.45	0.15	0.29	0.0	40	380	6.7
mean	173	6.17	3.58	2.02	0.16	0.34	0.70	38.7	393	7.5

Pt #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Male Children of Exposed Parent(s), Age less than 15 years										
88	186	6.70	3.41	2.81	0.27	0.13	0.70	43	430	7.2
89	190	7.13	2.57	4.07	0.36	0.14	0.0	43	430	7.1
90	248	5.62	2.24	2.98	0.11	0.28	0.0	43	310	7.0
91	150	5.76	1.84	3.46	0.11	0.35	0.0	45	410	7.3
93	161	7.81	4.30	3.12	0.08	0.31	0.0	39	410	7.9
96	189	7.27	3.20	3.64	0.36	0.07	0.0	40	410	7.3
97	213	9.91	5.55	3.67	0.20	0.50	0.0	36	420	7.0
102	119	6.51	3.52	2.60	0.07	0.33	0.0	40	320	7.2
103	117	9.79	4.12	4.93	0.10	0.70	1.00	44	460	7.0
104	219	12.17	5.84	5.84	0.24	1.22	0.0	39	450	8.0
109	145	8.93	4.29	3.57	0.45	0.54	0.90	34	360	7.0
110	207	9.79	2.84	5.78	0.59	0.59	0.0	41	460	7.1
111	436	8.06	3.23	3.71	0.24	0.81	0.80	37	430	7.6
113	319	8.05	4.35	3.46	0.08	0.16	0.0	42	430	7.7
115	218	12.38	4.95	6.94	0.25	0.25	0.0	36	510	6.8
116	228	10.71	5.68	10.20	0.33	0.50	0.0	42	590	7.8
118	267	10.97	4.39	5.71	0.32	0.55	0.0	42	430	7.6
126	148	8.24	3.05	4.78	0.16	0.16	0.80	44	520	7.7
130		9.54	3.91	5.15	0.10	0.38	0.0	44	370	-
131		9.82	3.05	6.19	0.29	0.20	1.00	36	370	-
132	430	9.75	2.63	6.93	0.39	0.78	0.0	35	420	7.4
141		9.53	3.81	5.24	0.29	1.14	0.0	31	400	-
142	379	13.57	4.34	9.15	0.27	0.81	0.0	35	390	7.2
148		9.77	4.01	4.59	0.20	0.88	1.00	41	400	-

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Male Children of Exposed Parent(s), Age less than 15 years (cont'd)										
150		11.05	3.87	5.86	0.33	0.99	0.0	37	590	-
155		8.14	3.42	4.15	0.08	0.49	0.0	34	370	-
157		13.40	5.36	6.16	0.27	1.61	0.0	39	370	-
158		7.56	2.19	4.77	0.23	0.38	0.0	32	330	-
161		8.39	4.78	3.10	0.08	0.42	0.0	40	380	-
163		11.55	5.77	4.62	0.46	0.69	0.0	27	350	-
166		10.08						36		
169		9.70	3.59	5.24	0.29	0.49	1.00	30	260	-
171		6.14	2.89	2.89	0.06	0.31	0.0	47	340	-
172		5.55	2.11	3.20	0.11	0.11	0.0	30	320	-
mean	228	9.27	3.79	4.77	0.24	0.52	0.22	38.4	405	7.3

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Female Children of Exposed Parent(s), Age less than 15 years										
87	133	5.65	2.88	2.09	0.23	0.45	0.0	42	370	7.8
92	177	8.34	4.17	3.92	0.08	0.17	0.0	41	370	7.5
94	192	7.66	4.05	2.83	0.15	0.54	0.80	38	420	7.3
95	235	7.48	3.52	3.22	0.15	0.60	0.0	42	380	7.1
100	220	5.35	2.14	3.05	0.11	0.05	0.0	36	290	7.9
101	126	9.12	4.02	4.56	0.09	0.46	0.0	40	340	8.3
105	133	5.61	2.25	2.86	0.11	0.34	0.0	42	360	7.7
106	196	8.44	2.62	4.81	0.34	0.68	0.0	44	480	7.8
119		11.85	5.33	5.69	0.47	0.36	0.0	37	380	-
120	210	8.29	4.39	2.65	0.08	0.17	0.0	41	420	8.0
122	283	10.00	2.80	5.80	0.50	0.90	0.0	35	360	7.2
125	326	10.44	3.24	6.37	0.31	0.42	1.00	42	430	7.3
127	376	11.77	2.83	8.01	0.12	0.82	0.0	36	470	5.5
128		8.73	2.01	5.67	0.35	0.52	1.70	38	320	-
134		12.19	3.41	7.56	0.37	0.73	1.20	35	390	-
135	319	11.27	3.27	6.87	0.45	0.68	0.0	38	400	7.5
137	330	9.90	4.06	5.25	0.40	0.20	0.0	38	410	6.6
138		14.10	7.19	5.50	0.28	1.13	0.0	45	410	-
139		12.06	4.95	6.52	0.36	0.12	1.20	36	400	-
140		8.24	3.96	3.46	0.16	0.58	0.80	44	400	-
143		9.86	4.24	4.74	0.10	0.79	0.0	35	450	-
145		7.88	3.86	3.39	0.08	0.55	0.0	36	-	-
152		8.38	3.02	4.78	0.08	0.50	0.0	35	370	-
153		8.57	4.46	3.51	0.17	0.43	0.0	32	400	-

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Female Children of Exposed Parent(s), Age less than 15 years (cont'd)										
154		17.27	5.18	10.19	0.35	1.55	0.0	36	470	-
6		11.64	4.31	6.17	0.23	0.82	1.20	30	440	
160		13.92	6.96	5.15	0.28	1.39	1.40	37	410	
164		9.72	4.96	4.38	0.19	0.19	0.0	49	44	
165		16.61	5.98	8.97	0.33	1.33	0.0	32	420	
173		12.27	6.87	4.90	0.25	0.25	0.0	32	350	
176		9.22	2.86	5.17	0.37	0.74	0.90	32	310	
mean	233	10.06	4.06	5.13	0.24	0.60	0.33	37.9	391	7.40

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X-10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Males, Age 15 to 40 years										
813	200	6.66	2.80	3.40	0.13	0.33	0.0	39	410	6.8
814	195	6.31	2.08	3.72	0.25	0.25	0.0	50	450	7.5
815	-	5.44	2.07	3.16	0.11	0.11	0.0	42	-	7.9
818	-	5.84	2.86	2.63	0.06	0.29	0.0	50	-	7.9
820	-	4.88	3.12	1.51	0.05	0.20	0.0	48	-	7.6
823	212	6.54	3.67	2.49	0.13	0.26	0.0	43	390	6.7
827	176	10.85	6.08	3.91	0.22	0.65	0.0	48	440	7.5
830	176	6.35	2.67	2.86	0.25	0.51	0.60	48	400	7.1
833	152	4.00	1.32	2.44	0.20	0.04	0.0	49	520	7.9
834	153	7.84	3.13	4.39	0.16	0.16	0.0	48	350	7.4
836	158	6.08	2.43	3.16	0.18	0.30	0.0	50	410	8.5
840	225	6.98	2.65	3.63	0.14	0.56	0.0	47	500	7.7
863	-	7.68	3.23	3.84	0.15	0.38	0.80	51	-	7.3
881	167	7.74	3.10	4.18	0.15	0.23	0.80	46	460	8.0
882	201	6.50	2.21	3.84	0.13	0.33	0.0	46	450	6.9
920	104	5.91	3.55	1.89	0.06	0.42	0.0	51	440	8.0
921	131	6.09	3.71	1.71	0.18	0.49	0.0	43	410	7.1
931	232	6.32	3.98	1.08	0.32	0.95	0.0	44	390	7.1
939	-	5.76	3.00	2.36	0.06	0.35	0.0	55	-	8.1
966	166	5.02	1.91	2.91	0.10	0.10	0.0	49	440	7.1
981	228	7.39	2.51	4.43	0.22	0.15	0.70	44	410	7.6
1005	113	6.80	2.38	3.74	0.14	0.54	0.0	54	460	7.1
1523	223	5.81	1.86	3.49	0.12	0.35	0.0	48	520	7.2
1526	248	9.38	3.75	5.16	0.09	0.38	0.0	55	500	8.1
1529	160	6.86	3.02	3.29	0.14	0.41	0.0	49	530	7.4
1533	199	8.56	2.65	4.96	0.34	0.60	0.0	44	600	7.5
mean	182	6.71	2.91	3.24	0.17	0.36	0.11	47.9	451	7.5

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Females, Age 15 to 40 years										
805	253	6.00	1.98	3.30	0.18	0.54	0.0	42	400	6.9
811	409	9.65	4.83	3.38	0.39	0.97	1.00	27	300	6.9
812	240	4.98	2.04	2.74	0.10	0.10	0.0	38	360	8.0
816	219	7.58	2.96	3.79	0.15	0.68	0.0	43	410	6.8
821	222	6.22	3.30	2.30	0.25	0.31	0.60	39	420	6.6
829	170	3.21	2.02	0.80	0.03	0.32	0.30	41	370	8.6
832	109	4.55	2.28	1.91	0.09	0.27	0.0	39	310	8.1
835	291	7.28	3.42	2.84	0.15	0.87	0.0	23	270	7.1
841	188	6.86	3.29	3.16	0.07	0.27	0.70	44	450	7.8
843	116	4.35	2.31	1.78	0.09	0.17	0.0	34	300	6.9
865	206	7.24	3.48	2.97	0.29	0.51	0.0	27	320	6.8
891	222	4.67	2.34	1.96	0.09	0.28	0.0	42	390	8.0
896	208	7.41	3.93	3.04	0.07	0.30	0.70	41	420	7.7
914	298	6.18	3.09	2.29	0.12	0.67	0.60	3[illegible]	380	6.6
925	-	6.96	2.78	3.34	0.28	0.42	0.0	43	-	8.0
926	179	8.55	4.79	2.82	0.17	0.68	0.90	39	410	8.0
932	287	5.46	3.28	1.97	0.05	0.16	0.0	36	360	7.0
934	227	8.40	3.44	4.20	0.08	0.59	0.80	44	360	7.6
938	234	7.48	4.86	1.65	0.15	0.82	0.0	35	330	7.5
955	-	6.84	4.38	2.33	0.07	0.07	0.0	41	-	-
959	268	8.02	5.06	2.49	0.16	0.32	0.0	36	380	6.9
960	260	11.42	5.71	4.80	0.11	0.80	0.0	44	460	8.3

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Females, Age 15 to 40 years (cont'd)										
962	-	6.12	2.14	3.43	0.12	0.37	0.60	38	-	7.0
980	111	5.29	2.75	2.17	0.05	0.32	0.0	42	360	7.0
993	238	6.65	2.46	3.52	0.27	0.33	0.70	44	360	7.8
996	280	9.40	4.32	4.51	0.38	0.19	0.0	40	340	7.2
1001	165	4.77	1.91	2.29	0.14	0.43	0.0	41	430	7.1
1035		7.57	3.10	2.88	0.30	1.29	0.0	43	400	8.5
1043	160	5.75	2.30	3.11	0.12	0.17	0.60	39	340	8.0
1050	219	6.95	3.54	2.99	0.07	0.35	0.0	43	380	7.5
1505	250	7.89	3.32	3.47	0.30	0.79	0.0	37	360	7.1
1520	194	7.04	2.32	3.87	0.28	0.42	1.40	42	420	7.3
1525	232	6.78	2.51	3.80	0.14	0.34	0.0	41	430	7.4
1528	324	7.88	2.68	4.89	0.08	0.16	0.80	45	540	7.9
1534	273	9.39	-	-	-	-	-	43	450	7.6
mean	227	6.79	3.20	2.96	0.16	0.45	0.28	39.2	382	7.5

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Males, Age greater than 40 years										
842	165	9.15	-	-	-	-	-	53	380	
849	-	6.42	3.60	1.86	0.26	0.71	0.0	51	400	8.1
850	166	5.03	1.61	2.82	0.15	0.40	0.50	43	410	8.2
855	176	5.81	2.91	2.44	0.12	0.29	0.60	42	360	7.7
856	231	6.33	3.35	2.03	0.06	0.89	0.0	41	390	7.6
864	193	7.31	3.95	2.49	0.07	0.80	0.0	47	480	7.0
878	151	6.17	3.15	2.71	0.12	0.12	0.60	42	460	8.9
880	193	7.25	4.06	2.76	0.07	0.36	0.0	45	410	7.5
883	-	5.68	2.95	2.10	0.06	0.57	0.0	46	-	7.5
897	106	7.20	3.46	3.46	0.07	0.22	0.0	40	320	7.9
910	-	4.44	2.13	1.78	0.18	0.36	0.0	47	-	7.8
915	179	5.93	3.14	2.25	0.18	0.35	0.0	45	390	7.6
918	87	5.43	2.34	2.34	0.05	0.65	0.50	46	380	7.5
944	136	4.72	2.93	1.18	1.14	0.38	0.90	49	500	8.6
947	119	10.18	5.09	4.18	0.10	0.71	1.00	39	390	7.0
963	-	4.76	3.43	1.00	0.19	0.19	0.50	49	-	
1007	164	5.43	2.77	2.39	0.05	0.16	0.50	46	430	8.0
1515	169	4.79	1.39	2.97	0.10	0.29	0.50	44	400	7.4
1517	188	5.12	2.20	3.61	0.15	0.15	0.0	45	440	7.0
1527	156	6.72	3.02	3.36	0.07	0.27	0.0	51	480	8.0
mean	161	6.09	3.02	2.46	0.17	0.41	0.29	45.6	414	7.7

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X 10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Females, Age greater than 40 years										
844	141	8.16	3.84	3.84	0.24	0.24	0.0	44	440	6.6
846	164	5.61	2.75	2.41	0.11	0.28	0.60	42	370	8.4
851	224	5.60	2.69	2.24	0.11	0.56	0.0	38	380	7.2
858	176	6.06	1.88	3.64	0.12	0.26	0.60	38	360	8.0
859	112	4.65	2.23	1.77	0.05	0.56	0.50	44	300	8.1
867	169	8.77	5.00	2.98	0.09	0.70	0.0	42	360	7.5
868	113	5.95	3.45	1.55	0.12	0.77	0.60	48	450	7.8
898	93	4.87	2.39	1.95	0.10	0.39	0.50	41	320	8.0
908	186	7.74	4.26	3.10	0.08	0.31	0.0	43	380	7.3
922	178	5.35	2.73	2.03	0.05	0.43	1.10	41	390	8.7
928	-	4.68	1.92	2.39	0.09	0.23	0.50	45	-	8.8
941	101	7.52	4.44	2.48	0.15	0.38	0.80	40	350	7.9
945	166	5.55	3.61	1.39	0.11	0.44	0.0	47	460	7.6
948	136	6.28	2.83	3.01	0.13	0.25	0.60	50	410	7.8
956	261	7.62	2.51	4.72	0.15	0.15	0.80	42	370	7.8
957	85	7.25	3.34	3.48	0.07	0.36	0.0	34	320	8.0
970	155	7.21	-	-	-	-	-	37	280	7.4
982	122	6.14	3.44	1.84	0.12	0.61	1.20	41	410	7.9
mean	152	6.39	3.14	2.64	0.11	0.41	0.46	42.1	374	7.8

Pt. #	Plat. (X10-3)	WBC (X10-3)	Nuetro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Males, Age less than 15 years										
801	398	11.95	2.63	7.41	0.36	1.43	1.20	38	440	8.3
802	235	7.83	2.98	4.23	0.31	0.31	0.0	42	460	7.1
803	258	7.78	2.57	4.59	0.16	0.47	0.0	39	410	6.7
806	217	8.02	4.49	2.97	0.16	0.24	1.60	44	370	7.4
807	301	11.22	5.50	4.27	0.22	1.24	0.0	38	410	7.2
809	186	7.27	3.78	2.76	0.36	0.36	0.0	34	380	8.3
870	234	9.75	5.07	4.00	0.20	0.49	0.0	36	420	7.1
904	263	8.03	3.85	3.77	0.16	0.24	0.0	39	410	6.8
905	81	5.68	3.47	1.88	0.17	0.17	0.0	39	270	7.4
952	145	5.90	4.25	1.12	0.12	0.41	0.0	38	370	7.1
972	265	7.41	4.82	1.41	0.07	1.11	0.0	41	430	7.0
1002	178	8.20	4.02	4.02	0.0	0.80	0.80	39	440	7.4
1004										
1006										
1009	279	6.82	3.56	2.46	0.07	0.75	0.0	40	410	7.5
1010	227	9.61	3.94	5.29	0.19	0.19	0.0	38	390	6.8
1014	193	8.41	3.87	3.95	0.17	0.42	0.0	37	390	7.6
1015	174	13.23	7.94	3.31	0.13	1.85	0.0	38	380	7.9
1017	287	7.95	4.69	2.78	0.08	0.32	0.80	40	460	7.7
1018	405	9.35	3.74	5.42	0.09	1.03	0.0	37	440	6.6
1027	331	9.91	5.35	3.47	0.10	0.99	0.0	37	400	7.8
1028	137	8.04	4.18	3.06	0.08	0.72	0.0	37	400	7.5

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutr. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Males, Age less than 15 years (cont'd)										
1032	287	7.53	3.99	2.56	0.15	0.83	0.0	39	430	7.0
1037		6.39	3.00	3.13	0.13	0.13	0.0	34	380	-
1038	421	10.25	4.72	4.20	0.52	0.62	0.0	32	350	6.3
1039		9.88	3.95	4.94	0.40	0.59	0.0	35	380	-
1047		7.97	3.75	3.50	0.16	0.48	0.80	37	430	-
1053	217	6.59	4.09	1.71	0.13	0.66	0.0	42	450	7.2
1054		10.73	5.69	4.08	0.11	0.86	0.0	39	440	-
1056		6.72	3.16	2.89	0.27	0.40	0.0	37	370	-
1058		10.02	3.11	5.81	0.30	0.60	2.00	35	400	-
1062		7.09	2.41	4.40	0.14	0.14	0.0	34	300	-
1063		11.27	5.30	4.85	0.23	0.90	0.0	34	350	-
1064		10.74	6.72	4.08	0.0	0.54	0.0	35	450	-
1067		9.83	5.21	3.54	0.20	0.89	0.0	40	390	-
1068	-	18.61	-	-	-	-	-	35	340	-
1072		11.02	4.63	5.73	0.22	0.44	0.0	35	420	-
1076		11.81	3.66	6.73	0.48	0.95	0.0	32	370	
1077		8.14	2.60	4.48	0.24	0.81	0.0	37	370	
1078		10.50	4.62	5.25	0.11	0.53	0.0	36	440	
1081		7.72	4.48	2.16	0.23	0.77	0.80	40	330	
1082		11.60	6.03	5.10	0.12	0.35	0.0	34	360	

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Males, Age less than 15 years (cont'd)										
1088		13.10	6.81	5.50	0.0	0.79	0.0	30	400	-
1089		7.36	4.79	2.43	0.07	0.07	0.0	35	390	-
1091		9.92	3.57	5.08	0.09	0.18	0.0	30	380	-
1092		8.88	4.51	3.82	0.09	0.45	0.0	31	400	-
1093		6.58	3.49	2.24	0.26	0.53	0.70	32	410	-
1099		4.80	2.74	1.87	0.10	0.10	0.0	34	280	-
1102		10.37	5.91	4.05	0.10	0.31	0.0			
mean	249	9.08	4.28	3.86	0.18	0.52	0.19	36.6	393	7.3

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Females, Age less than 15 years										
808	251	10.89	5.12	4.90	0.11	0.76	0.0	34	340	7.6
810	194	7.54	3.77	3.09	0.15	0.53	0.0	39	360	7.6
866	185	5.93	2.85	2.85	0.06	0.18	0.0	46	400	7.1
901	339	9.41	4.05	4.71	0.09	0.47	0.90	36	410	7.0
903	139	8.09	4.21	2.59	0.08	1.21	0.0	37	320	7.7
906	243	7.54	3.77	3.17	0.08	0.53	0.0	39	480	7.4
23	284	8.61	4.39	3.19	0.43	0.52	0.90	42	460	7.6
954	183	7.99	4.96	1.84	0.08	0.96	1.60	38	400	7.6
979	170	6.17	3.21	2.47	0.31	0.19	0.0	43	360	7.9
992	274	7.63	2.29	4.66	0.08	0.61	0.0	40	390	7.5
995	164	9.68	5.81	2.81	0.10	0.97	0.0	39	380	8.0
1008	235	10.17	4.78	4.58	0.31	0.41	1.00	42	410	8.2
1011	229	8.44	4.39	3.29	0.25	0.34	1.70	38	320	8.4
1019	215	7.33	2.57	4.03	0.37	0.37	0.0	37	370	7.0
1020	249	8.78	3.78	3.60	0.18	1.23	0.0	37	390	8.5
1021	276	8.79	5.01	3.08	0.35	0.35	0.0	37	390	7.5
1022	211	7.05	2.33	3.95	0.28	0.49	0.0	39	380	6.6
1025	-	16.09	6.76	3.11	0.55	0.67	0.0	40	420	-
1026	272	8.09	3.88	3.56	0.16	0.49	0.0	38	390	7.4
1029	260	8.85	2.48	5.75	0.18	0.44	0.0	43	440	7.6
1031	175	15.00	7.35	5.70	0.60	1.20	1.50	37	410	8.0
1044	-	10.52	4.21	4.84	0.42	0.84	2.10	32	350	-

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Nonexposed Females, Age less than 15 years (cont'd)										
1060		9.67	3.00	5.13	0.39	1.16	0.0	35	380	
1065		14.85	7.28	5.64	0.13	1.78	0.0	35	340	
1066		10.63	5.42	4.57	0.32	0.64	0.0	37	400	
1069		10.10	4.60	4.10	0.40	0.80	1.00	39	430	
1070		9.89	3.86	5.24	0.10	0.59	1.00	35	420	
1075		16.27	6.51	6.51	0.65	2.28	3.30	36	350	
1079		11.93	5.13	6.09	0.12	0.60	0.0	36	410	
1080		16.77	8.72	6.04	0.34	1.68	0.0	33	370	
1084		11.49	5.75	4.25	0.23	1.15	1.10	37	450	
1087		17.46	8.21	8.56	0.35	0.35	0.0	36	410	
1094		11.08	5.76	4.43	0.33	0.55	0.0	34	430	
1095		10.56	4.44	5.18	0.11	0.74	1.10	37	440	
1096		8.38	3.02	4.69	0.25	0.42	0.0	31	410	
1509	266	7.38	2.88	3.69	0.30	0.44	0.70	36	400	7.4
1518		15.91	5.09	9.23	0.32	1.27	0.0	35	270	
1521	306	6.60	2.18	3.57	0.26	0.53	0.70	36	410	7.2
mean	232	10.07	4.57	4.44	0.26	0.76	0.49	37.4	392	7.6

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Utirik Males, Age 15 to 40 years										
2115		9.31	4.56	4.28	0.19	0.28	0.0	46	500	7.3
2123		5.87	3.29	2.17	0.23	0.18	0.0	46	380	7.6
2124		9.58	5.08	3.64	0.19	0.67	0.0	51	500	8.1
2144		7.16	2.86	3.87	0.29	0.14	0.0	50	-	7.5
2150		7.61	3.96	2.67	0.08	0.84	0.80	52	510	7.4
2153		6.11	2.93	2.69	0.12	0.37	0.0	41	-	8.3
2155		7.51	2.93	4.21	0.08	0.30	0.0	44	-	8.0
2156		4.37	2.28	1.79	0.09	0.22	0.0	50	-	7.0
2165		7.62	2.97	4.35	0.15	0.15	0.0	43	-	8.4
2167		7.62	4.19	2.97	0.08	0.38	0.0	46	-	7.7
2168		6.95	3.41	2.92	0.07	0.56	0.0	49	-	7.8
2174		11.19	6.76	3.92	0.22	0.78	1.10	47	470	7.7
2178		7.83	2.34	4.78	0.31	0.39	0.0	42	-	7.7
2201		6.53	-	-	-	-	-	45	-	8.0
2232		6.29	3.27	2.63	0.06	0.31	0.0	49	420	7.4
2234		6.89	2.76	3.51	0.07	0.55	0.0	52	-	7.4
2235		7.61	3.20	3.88	0.23	0.30	0.0	44	-	7.6
2236		9.79	4.78	4.58	0.20	0.20	0.0	49	-	7.6
2240		8.72	5.23	3.14	0.09	0.17	0.90	43	-	8.0
2242		6.25	3.25	2.50	0.13	0.38	0.0	38	-	6.8
2245		6.04	3.02	2.42	0.30	0.30	0.0	40	-	7.6
2250		7.01	3.43	2.66	0.14	0.70	0.70	44	-	7.0
2257		6.68	3.81	2.47	0.07	0.33	0.0	51	-	7.6
Mean		7.41	3.65	3.27	0.15	0.39	0.16	46.2	463	7.6

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X 10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Utirik Females, Age 15 to 40 years										
2107		13.60	7.34	5.31	0.13	0.82	0.0	43	-	8.5
2111		10.69	5.99	4.38	0.11	0.21	0.0	41	-	8.2
2113		7.99	4.15	3.12	0.16	0.48	0.80	43	460	7.5
2117		7.52	3.23	3.84	0.08	0.38	0.0	47	-	7.9
2128		6.62	3.24	2.98	0.07	0.33	0.0	35	-	8.6
2130		8.40	4.37	3.11	0.17	0.67	0.80	33	-	7.0
2132		7.52	3.61	3.38	0.08	0.38	0.80	42	-	8.5
2139		6.73	2.90	3.03	0.13	0.61	0.70	38	-	7.4
2149		7.71	3.39	3.93	0.15	0.23	0.0	39	-	7.8
2159		8.24	4.70	2.56	0.16	0.82	0.0	39	-	7.2
2160		8.37	5.44	2.26	0.17	0.50	0.0	40	-	7.7
2171		5.93	2.97	2.49	0.12	0.29	0.60	42	420	7.0
2172		6.53	3.39	2.74	0.07	0.39	0.0	42	-	8.1
2197		7.00	3.29	3.50	0.07	0.14	0.0	35	-	7.2
2217		7.36	3.61	3.24	0.15	0.37	0.0	33	-	7.2
2218		9.64	4.43	4.72	0.19	0.29	0.0	42	-	8.1
2226		6.18	3.09	2.85	0.06	0.19	0.0	40	-	7.5
2227		7.20	3.82	2.88	0.14	0.36	0.0	38	-	7.7
2230		5.72	2.57	2.52	0.11	0.46	0.60	43	-	7.6
2246		6.21	3.23	2.17	0.06	0.62	1.20	41	-	7.8
2247		8.77	-	-	-	-	-	36	-	7.9
2248		4.73	2.37	1.99	0.05	0.28	0.50	38	-	8.2
2249		5.47	2.63	2.46	0.11	0.27	0.0	41	-	7.4
2251		7.76	3.26	4.04	0.08	0.31	0.80	33	-	8.1
2254		5.02	2.66	1.91	0.10	0.71	0.0	29	-	7.1
2255		11.92	5.84	5.13	0.12	0.72	1.20	42	-	7.8
2256		6.68	3.61	2.34	0.13	0.53	0.70	46	-	8.5
mean		7.61	3.81	3.19	0.11	0.44	0.33	39.3	440	7.8

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Utitik Males, Age greater than 40 years										
2103		6.18	3.15	2.78	0.06	0.19	0.0	41		7.1
2105		8.56	4.37	3.43	0.16	0.51	0.90	52	470	8.5
2110		6.42	3.66	2.44	0.13	0.19	0.0	44	-	7.6
2114		7.22	4.12	2.53	0.07	0.51	0.0	37	-	8.0
2120		5.72	2.69	2.80	0.11	0.11	0.0	44	-	8.8
2125		5.96	3.40	1.97	0.12	0.36	1.20	53	-	8.6
2145		6.66	2.13	4.07	0.27	0.20	0.0	47	360	7.6
2148		5.82	2.45	3.09	0.17	0.12	0.0	45	-	7.9
2157		9.35	4.86	4.02	0.19	0.28	0.0	42	-	7.4
2206		5.20	2.97	1.87	0.05	0.31	0.0	43	-	7.2
2211		10.14	5.28	4.16	0.20	0.30	2.00	35	-	8.6
2252		5.06	2.38	2.28	0.10	0.30	0.0	51	-	8.9
2258		7.52	3.84	2.93	0.15	0.60	0.0	44	-	7.9
mean		6.91	3.48	2.95	0.14	0.28	0.32	44.4	415	8.0

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Utirik Females, Age greater than 40 years										
2109		6.68	2.87	3.41	0.20	0.20	0.0	42	-	7.8
2140		8.30	4.32	3.65	0.08	0.25	0.0	40	-	8.2
2146		7.42	3.04	3.94	0.15	0.30	0.0	43	-	8.0
2158		6.24	3.00	2.94	0.13	0.13	0.60	38	380	7.5
2162		6.50	3.97	1.95	0.07	0.52	0.0	42	380	7.0
2189		9.18	5.05	3.40	0.18	0.46	0.90	43	-	7.9
2193		8.64	3.37	4.58	0.17	0.43	0.90	39	-	8.1
2194		9.28	4.83	3.39	0.28	0.19	0.0	44	-	8.6
2196		8.79	4.31	3.78	0.18	0.44	0.90	41	-	7.7
2200		5.42	3.36	1.68	0.11	0.27	0.0	42	-	8.2
2208		9.65	5.41	3.57	0.19	0.48	0.0	40	-	8.0
2212		8.45	4.22	3.30	0.17	0.68	0.80	37	-	7.6
2215		9.20	5.89	2.85	0.18	0.18	0.90	40	-	7.7
2216		7.88	3.39	3.47	0.24	0.63	1.60	44	-	8.3
2220		7.28	4.00	2.91	0.07	0.29	0.0	37	-	7.6
2221		8.11	3.57	3.00	0.16	1.30	0.80	31	-	7.6
2224		9.32	4.66	3.73	0.09	0.75	0.90	40	-	7.0
2244		4.74	2.61	1.94	0.05	0.14	0.0	40	-	7.6
mean		7.84	3.99	3.19	0.15	0.42	0.46	40.2	380	7.8

Pt. #	Plat. (X10-3)	WBC (X10-3)	Neutro. (X10-3)	Lymph. (X10-3)	Mono. (X10-3)	Eosin. (X10-3)	Baso. (X10-2)	Hct. (%)	RBC (X10-4)	Serum Protein (g)
Male Children of Exposed Utirik Parent(s), Age less than 15 years										
2261		4.09	2.00	1.84	0.82	0.16	0.0	36		7.5
2268		7.12	4.20	2.35	0.14	0.36	0.70	44		8.0
2276		9.55	5.06	4.20	0.10	0.19	0.0	44		7.2
mean		6.92	3.75	2.79	0.35	0.24	0.23	41		7.6
Female Children of Exposed Utirik Parent(s), Age less than 15 years										
2270		8.14	5.21	2.28	0.08	0.57	0.0	-	-	7.5
2273		6.60	5.53	2.53	0.20	0.33	0.60	44		9.1
mean		7.37	4.37	2.40	0.14	0.45	0.30	44		8.3

Appendix 8

Mean Blood Counts at Various Times After Exposure

(a) Exposed Rongelap (175 rad group)

Postexposure Day	WBC ($\times10^{-3}$)		Neutrophils ($\times10^{-3}$)		Lymphocytes ($\times10^{-3}$)		Platelets ($\times10^{-4}$)				Hematocrit %			RBC ($\times10^{-6}$)		
	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female All Ages	Total Group	Male <15	Male >15	Female All Ages	Male <15	Male >15	Female All Ages
3	9.0	8.2	6.4	4.7	1.8	2.2	----	----	----	----	----	----	----	----	----	----
7	4.9	6.2	---	---	---	---	----	----	----	----	----	----	----	----	----	----
10	6.6	7.1	3.5	4.5	2.6	2.1	28.2	22.7	24.9	24.8	----	----	----	----	----	----
12	5.9	6.3	3.5	3.9	2.1	1.7	----	----	----	----	----	----	----	----	----	----
15	5.9	6.5	3.2	4.1	2.4	1.9	27.1	21.3	21.7	22.5	----	----	----	----	----	----
18	6.7	7.2	3.4	4.7	2.4	2.1	21.8	19.1	21.8	21.0	----	----	----	----	----	----
22	7.0	7.4	4.3	5.0	2.6	2.1	16.8	14.6	15.2	15.3	37.5	43.9	39.0	----	----	----
26	5.7	6.1	3.0	3.9	2.3	1.8	13.2	12.9	10.9	11.9	36.3	41.6	37.5	----	----	----
30	7.6	7.8	4.0	5.3	3.2	2.1	14.1	12.3	11.8	12.3	37.9	42.2	37.1	----	----	----
33	6.5	6.2	3.1	3.8	3.2	2.0	17.9	16.6	15.1	16.0	37.4	42.2	36.8	----	----	----
39	5.7	5.5	3.0	3.3	2.6	2.0	29.5	22.0	22.4	22.8	37.8	42.4	37.4	----	----	----
43	5.2	5.2	2.0	2.6	2.9	2.3	26.8	20.9	23.2	23.2	37.3	41.8	37.6	----	----	----
47	5.9	5.8	2.6	3.3	3.1	2.4	24.6	20.6	23.9	23.1	39.0	43.4	38.3	----	----	----
51	6.7	5.6	2.6	3.5	3.4	2.1	22.1	17.5	21.2	20.3	----	----	----	----	----	----
56	7.0	6.0	3.5	3.5	3.7	2.4	----	----	----	----	----	----	----	----	----	----
63	7.7	6.0	3.9	3.6	3.7	2.3	23.1	18.2	20.2	20.1	----	----	----	----	----	----
70	7.6	6.5	3.8	4.0	3.3	2.2	----	----	----	----	----	----	----	----	----	----
74	---	---	---	---	---	---	26.2	21.7	24.7	24.1	----	----	----	----	----	----
6-mo survey	8.5	6.6	4.6	4.2	3.6	2.2	24.4	20.3	23.2	22.6	38.0	41.7	38.2	----	----	----
1-yr survey	10.1	8.1	4.7	4.8	4.6	2.8	26.6	19.5	27.6	24.9	37.5	41.1	36.9	----	----	----
2-yr survey	11.8	8.6	5.9	4.8	4.7	3.1	30.0	21.4	25.5	24.7	38.7	41.2	38.1	----	----	----
3-yr survey	8.6	6.9	4.1	3.7	3.7	2.7	32.0	22.1	28.1	----	35.6	38.7	35.4	----	----	----
4-yr survey	8.9	7.5	3.3	3.4	4.6	3.6	32.5	27.1	30.8	----	35.6	41.0	35.8	----	----	----
5-yr survey	13.5	9.5	6.9	4.8	6.0	4.0	32.3	24.4	27.6	----	----	----	----	4.45	4.71	4.21
6-yr survey	----	6.5	---	3.5	---	3.7	----	----	----	----	----	----	----	----	----	----
7-yr survey	----	7.4	---	3.9	---	2.9	----	24.6[a]	27.3	----	37.6	41.7	37.0	4.54	4.45	4.11
8-yr survey	----	6.9	---	3.6	---	2.6	----	32.8[b]	32.1	---	38.5	43.0	39.3	4.68	4.67	4.44
9-yr survey	----	7.4	---	3.7	---	3.0	----	23.1[c]	28.4	----	39.1	43.7	38.4	4.29	4.38	4.12
10-yr survey	----	8.2	---	3.8	---	3.5	----	32.8	37.2	----	40.4	43.5	39.3	----	----	----
11-yr survey	----	7.4	---	3.7	---	3.0	----	26.3	28.5	----	39.9	44.0	37.7	4.65	4.60	3.94
12-yr survey	----	6.8	---	3.6	---	2.5	----	----	----	----	38.8	42.7	38.3	----	----	----
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

*Counted electronically with Coulter counter Model F.

(b) Ailingnae Group (69 rad)

Postexposure day	WBC ($\times 10^{-3}$)		Neutrophils ($\times 10^{-3}$)		Lymphocytes ($\times 10^{-3}$)		Platelets ($\times 10^{-4}$)				Hematocrit %			RBC ($\times 10^{-6}$)		
	< 5	> 5	< 5	> 5	< 5	> 5	Male < 10	Male > 10	Female all ages	Total Group	Male < 15	Male > 15	Female all ages	Male < 15	Male > 15	Female all ages
3	6.0	7.0	3.0	5.0	2.8	2.2	----	----	----	----	----	----	----	----	----	----
7	5.5	6.8	---	---	---	---	----	----	----	----	----	----	----	----	----	----
10	6.3	7.3	4.2	4.2	1.9	2.2	22.5	22.6	20.9	21.5	----	----	----	----	----	----
12	6.3	7.6	1.8	4.7	3.1	2.2	----	----	----	----	----	----	----	----	----	----
15	7.1	7.0	2.3	4.5	4.2	2.2	29.0	20.2	24.6	23.9	----	----	----	----	----	----
18	6.8	7.8	2.9	5.0	3.5	2.4	27.5	21.7	24.9	24.3	----	----	----	----	----	----
22	8.9	8.7	5.3	5.4	2.7	2.9	23.5	17.0	22.9	21.3	37.5	43.7	39.2	----	----	----
26	8.4	7.0	4.8	4.4	3.2	2.2	20.0	13.8	17.4	16.7	36.5	43.2	36.8	----	----	----
30	9.6	8.6	5.3	6.2	3.7	2.0	19.5	12.8	18.2	16.8	36.0	44.6	36.7	----	----	----
33	7.7	7.8	3.3	5.2	3.5	2.2	24.0	15.8	22.7	17.6	35.5	43.8	37.3	----	----	----
39	7.5	6.2	2.9	4.2	4.7	1.9	26.5	20.8	27.0	25.2	35.0	45.6	37.4	----	----	----
43	6.9	6.5	2.7	3.6	3.9	2.7	28.0	19.6	25.3	24.0	36.0	45.2	36.8	----	----	----
47	7.3	6.7	3.5	3.8	3.4	2.7	27.0	20.0	26.1	24.5	----	46.5	40.2	----	----	----
51	8.4	6.3	3.8	3.6	4.0	2.2	32.0	18.2	25.0	23.9	----	----	----	----	----	----
54	4.6	6.3	2.8	3.5	3.2	2.5	37.0	19.8	23.8	24.2	----	----	----	----	----	----
6 mo survey	7.7	6.5	4.8	3.9	2.7	2.2	25.2	19.2	23.9	22.7	37.5	40.1	37.3	----	----	----
1-yr survey	11.1	7.8	4.2	4.7	6.5	5.6	38.7	21.4	28.3	27.5	33.0	44.6	36.2	----	----	----
2-yr survey	11.0	9.1	4.9	5.1	4.8	3.2	51.2	17.4	26.4	26.7	35.7	44.4	37.5	----	----	----
3-yr survey	12.1	7.0	5.5	3.9	5.6	2.6	40.8	22.4	31.2	----	37.5	40.6	35.6	----	----	----
4-yr survey	11.5	7.5	2.8	3.7	7.0	3.3	33.2	24.7	33.6	----	36.1	43.1	35.7	----	----	----
5-yr survey	----	9.7	---	5.1	---	3.7	40.9	26.3	26.8	----	----	----	----	4.46	5.15	4.31
6-yr survey	----	7.3	---	3.6	---	3.0	----	----	----	----	----	----	----	----	----	----
7-yr survey	----	7.7	---	4.1	---	3.1	----	25.6[a]	28.1	----	36.0	44.2	37.0	4.56	5.11	4.19
8-yr survey	----	6.5	---	3.4	---	2.6	----	33.4[b]	32.7	----	37.0	42.5	37.8	4.51	5.12	4.35
9-yr survey	----	7.1	---	4.0	---	2.4	----	23.5[c]	23.6	----	36.0	44.0	38.3	3.77	4.69	4.10
10-yr survey	----	7.5	---	3.6	---	3.1	----	32.4	41.5	----	37.0	43.0	38.3	----	----	----
11-yr survey	----	7.1	---	3.8	---	2.7	----	33.5	34.7	----	37.5	46.0	37.6	4.33	5.09	4.11
12-yr survey	----	6.2	---	3.3	---	2.3	----	----	----	----	38.5	44.2	37.8	----	----	----
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

(c) 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[a]	31.4	----	37.2	44.4	37.0	4.52	4.68	4.12
Rong. cont 8 yr	----	7.7	---	4.2	---	2.9	----	34.8[b]	34.5	----	38.3	44.1	39.0	4.60	4.90	4.47
Rong. cont 9 yr	----	7.7	---	3.9	---	3.1	----	29.1[c]	32.5	---	39.4	43.8	38.3	4.33	4.50	4.13
Rong. cont 10 yr	----	9.1	---	4.8	---	3.5	----	35.4	37.9	----	37.4	44.1	38.3	----	----	----
Rong. cont 11 yr	----	7.3	---	3.9	---	2.8	----	28.1	28.3	----	39.6	44.4	37.6	4.65	4.71	4.14
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

[a] includes all males >7.

[b] Includes all males >8.

[c] Includes all males >9.

(d) Utirik Group (14 rad)

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
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	----	----	----

*Includes all males >9

(e) Children

A - Parent or Parents Exposed*

Age	WBC ($\times 10^{-3}$)	Neutrophils ($\times 10^{-3}$)	Lymphocytes (10^{-3})	Platelets ($\times 10^{-4}$) Male	Platelets ($\times 10^{-4}$) Female	Hematocrit % Male	Hematocrit % Female	RBC ($\times 10^{-6}$) Male	RBC ($\times 10^{-6}$) Female
<13(1967)	9.9	4.7	4.1	32.6	36.3	37.6	36.4	4.14	4.08
<15(1969)	9.8	3.9	4.9	22.4	23.8	38.4	37.9	4.05	3.91
B - Parents Not Exposed									
<13(1967)	8.7	3.9	3.6	36.2	34.7	35.3	35.9	4.06	4.07
<15(1969)	9.5	4.4	4.0	24.9	23.2	36.6	37.4	3.93	3.92

*Rongelap and Ailingnae Group only.