The Medical Research Center
Brookhaven National Laboratory
Upton, L. I., New York

Medical Effects of Exposure of Human Beings to Fallout Radiation from a Thermonuclear Explosion

Eugene P. Cronkite, Victor P. Bond, Robert A. Conard
Medical Department, Brookhaven National Laboratory, Upton, New York, USA

Key Words. Thermonuclear explosion • Fallout • Human radiation exposure • Short-term and long-term radiation effects

Abstract. On March 1, 1954, after detonation of a thermonuclear device on Bikini atoll, an unexpected wind shift resulted in the deposition of radioactive fallout on inhabited atolls. The fallout radiation caused fleeting systemic effects, dose-dependent depression of hematopoiesis and skin burns primarily due to the beta ray component of the fission radionuclides. Within a few weeks, hematopoietic recovery was substantial but slight depression of blood counts was maintained for several years. One case of fatal acute myeloblastic leukemia developed in a boy receiving 1.9 Gy as an infant. Cretinism developed in two boys exposed as infants with estimated thyroidal dose in excess of 50 Gy. Chemical hypothyroidism was detected in several persons. Thyroid adenomas and cancer commenced appearance ten years after exposure and became a major long-term medical problem. There have been no late effects attributable to the beta burns 40 years after exposure. Internal contamination from ingestion and inhalation of radionuclides is detectable. The doses are comparable to background levels in the U.S. There is no detectible decrease in longevity of the exposed Marshallese compared to an unexposed Marshallese population.

Introduction

The Marshall Islands are located in the eastern part of Micronesia a few degrees north of the equator. The atolls consist of a ring of low-lying coral islands surrounding a lagoon. The islands are formed on the rims of extinct volcanic craters. The coral sand consists primarily of calcium carbonate and lesser amounts of other minerals. From 1946 until the termination of atmospheric testing in 1958, the Marshall Islands were used for testing of nuclear devices. Testing was done on Bikini atoll and Enewetak atoll. In addition, the United States developed the Pacific Missile Range at Kwajalein, thus the area was under the control of the Department of Defense and the Atomic Energy Commission. Until Operation Castle in 1954, Marshallese populations were removed from the atolls near the test site during testing. During Operation Greenhouse 1951, fallout was detected but not considered a serious health problem. The Marshallese people were not evacuated from inhabited atolls for Operation Castle.

On March 1, 1954, at 6:30 a.m., a nuclear device was detonated on Bikini. An unexpected change in the wind structure distributed fallout over inhabited areas exposing Marshallese, American military personnel and Japanese fisherman on board the *Lucky Dragon*. Details of the accident and its effect on the Marshallese were published by *Cronkite et al.* [1, 2]. The data are summarized herein.

The intense heat of the detonation incinerated the coral producing calcium oxide in which fission products were deposited.

The Marshallese lived in buildings appropriate for the tropics with thatched roof of palm fronds and open sides for ventilation. Fallout material collected on the palm fronds, the ground, trees and on individuals that were in the open.

From the wind velocity, arrival time and decay patterns, isodose lines were constructed in the downwind region. The fallout occurred in a cigar shaped pattern. In the westerly direction the 8.00 Gy isodose curve extended about 140 miles, 5.00 Gy about 160 miles, 3.00 Gy about 190 miles and 2.00 Gy about 220 miles. The north-south distance for 2 Gy was roughly

Correspondence: Dr. Eugene P. Cronkite, Medical Department, Brookhaven National Laboratory, P.O. Box 5000, Upton, New York 11973-5000 USA.

Received November 14, 1994; accepted for publication November 14, 1994. 1066-5099/95/$5.00/0

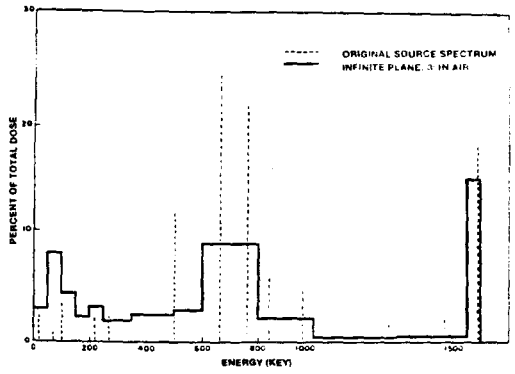

Fig. 1. Histogram of degraded energy spectrum of gamma radiation from a 4-day-old fallout field produced by Compton scattering at level of infinite plane 3 feet in air above uniformly distributed field.

40 miles with shorter distances for the higher isodose curves. A few miles north or south of the 2.0 Gy line would have placed people into the lethal range. These doses are estimates of what would have accumulated over a 48-hour period without shielding. The dose from which survival is unlikely is in the vicinity of 8.0 Gy, below which lethality may occur at doses as low as about 2.0 Gy. It is fortunate that persons on Ailinginae, Rongelap and Rongerik were just outside the 2.0 Gy isodose line.

Fallout was observed on Rongelap atoll and described as snowflakes. On Ailinginae and Rongerik atolls it was described as mist. On Utirik it was invisible.

The spectrum of gamma radiation from the fallout material showed peaks at 100, 700 and 1500 keV (Fig. 1). The two π geometry of exposure from the fallout field resulted in a very uniform depth dose with escalation of the absorbed dose in the first 1-3 cm at the surface of the body from the beta and low energy gamma components of the fission products, the surface dose being about eight times the 3 cm dose (Fig. 2). The absorbed dose rose continually to the time of evacuation about 51 h after the detonation on Rongelap to about 1.8 Gy (Fig. 3).

There were three groups of Marshallese and one group of American military personnel exposed (Table I). The absorbed dose in the Marshallese on Rongelap was revised to an average of 1.90 Gy [3]. The Marshallese on Ailinginae received about 0.80 Gy and on Utirik about 0.14 Gy. The American military personnel received about 0.78

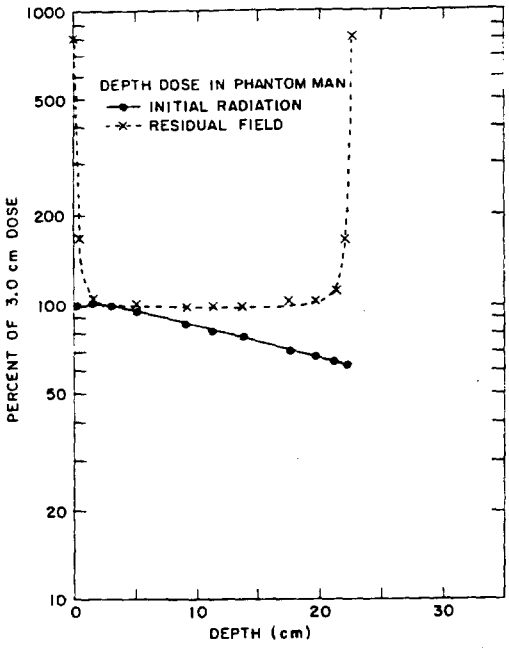

Fig. 2. Comparison of depth dose curves in phantom man of initial atomic bomb gamma radiation and gamma radiation from a planar field of fission products deposited on soil after an experimental nuclear detonation. Doses are expressed as percent of 3 cm dose.

Gy. They had better protection due to their metal housing and since they showered and put on

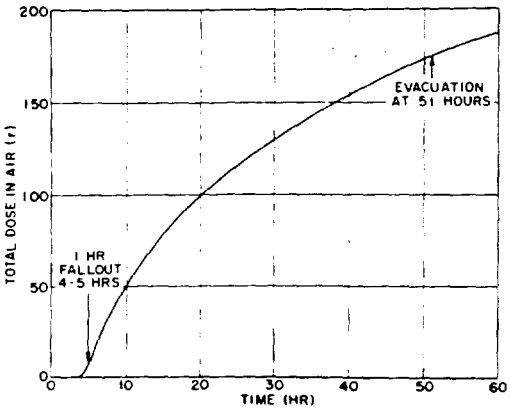

Fig. 3. Total cumulative dose in air from beginning of fallout at about 4-5 hours after detonation to evacuation at 51 hours post-detonation on Rongelap.

Table I. Expo

Rongelap
Ailinginae
Rongerik U.S
Utirik
Marshallese (
American Co

clothes, the b
less. Upon a
one would n
the magnitud
term change
torical or pre
Marshallese
nel. Accor
Marshallese
set up as sh
Figure
leukocytes
time after ex
shown by
counted. Th
cyte count
count. Duri
was no sigr
count. Com
there was
42 days aft
steady incre
that of the

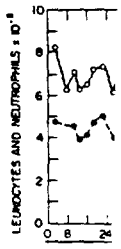

Fig. 4. To
Marshalle
population

Table I. Exposure data

	Total Exposed	Arrival Time Fallout Hours	Evacuation Hours	Dose Rate	Total Gamma Dose in Air (Gy)
Rongelap	67	4-6	50-51	375 mr/hr at 7 days	1.90
Ailinginae	18	4-6	58	100 mr/hr at 9 days	1.10
Rongerik U.S. Personnel	28	6.8	28.5-34	280 mr/hr at 9 days	0.78
Utirik	167	22	55-78	40 mr/hr at 8 days	0.11
Marshallese Control	117				
American Control	105				

clothes, the beta dose to the skin was substantially less. Upon arrival on March 8, it was clear that one would need comparison groups to determine the magnitude and significance of short- and long-term changes in blood counts. There were no historical or pre-exposure blood counts on any of the Marshallese or on the American military personnel. Accordingly, age-matched nonexposed Marshallese and American military personnel were set up as shown in Table I.

Figure 4 shows the changes in average total leukocytes and neutrophil counts as a function of time after exposure. The comparison population is shown by stars at the times when they were counted. The open circles show the total leukocyte count and the solid circles the neutrophil count. During the first two to three weeks, there was no significant diminution in the granulocyte count. Commencing about 30 days after exposure there was a slow diminution to a nadir at about 42 days after exposure. There was then a slow, steady increase in the granulocyte count to roughly that of the comparison population by two years after the exposure. Figure 5 shows that the average lymphocyte count was roughly 2000 per mm^3 and remained at this level for six months. About two to three years after exposure, the lymphocyte count had returned to that of the comparison population.

Figure 6 shows the serial changes in platelet counts. From day 8 to approximately day 30 there was a steady diminution in the average platelet counts to about 100,000 per mm^3. Thereafter there was an increase to near that of the comparison group by 40-50 days. Although the platelet counts returned to roughly 200,000 in males and females the counts remained consistently below that of the comparison population for seven years.

Another way of looking at the hematologic effects is to plot the cumulative percent of the blood counts as a function of time and blood counts. This is shown in Figures 7 and 8 for neutrophils and platelets. Group I Rongelap and Group II Ailinginae data show that the cumulative neutrophil counts at time of maximum depression are significantly shifted to the left

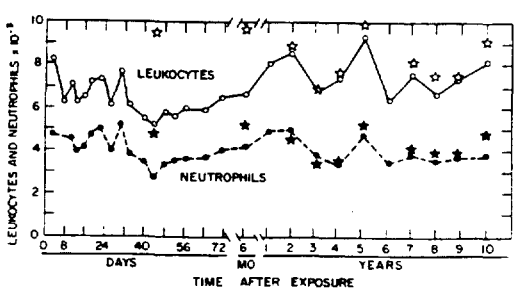

Fig. 4. Total leukocyte and neutrophil counts in Marshallese exposed on Rongelap. The comparison population is represented by stars.

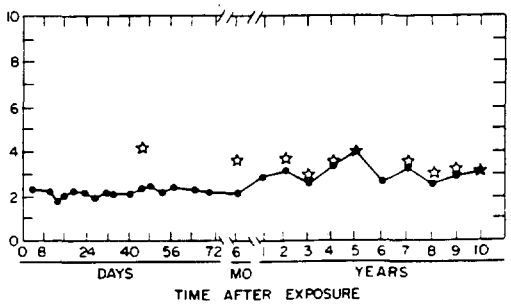

Fig. 5. Absolute lymphocyte count in Marshallese exposed on Rongelap compared to the comparison nonexposed population of Marshallese.

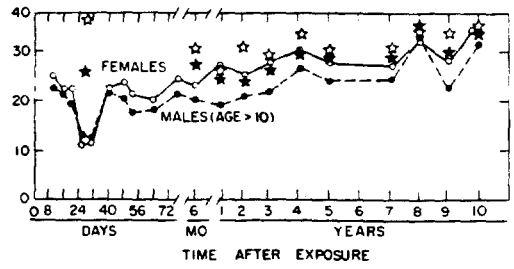

Fig. 6. Average platelet counts on Rongelapese for males and females for ten years after exposure to fallout radiation.

of the control population (Fig. 7). There is a slight difference between Rongelap (Group I) and Ailinginae (Group II).

Figure 8 shows a similar analysis for platelet counts of Rongelap, Ailinginae, Utirik and a control population at the time of maximum depression. The leftward shift of the cumulative platelet counts increases with the estimated dose of radiation. The cumulative platelet counts for Utirik detect a slight depression in counts from 0.11 Gy exposure.

The slowness in recovery of platelet levels in blood is demonstrated by the cumulative distribution of platelet counts seven years after exposure (Fig. 9). There is still a small but distinct shift to the left, suggesting that the genetic mechanism for regulating the platelet count is still perturbed.

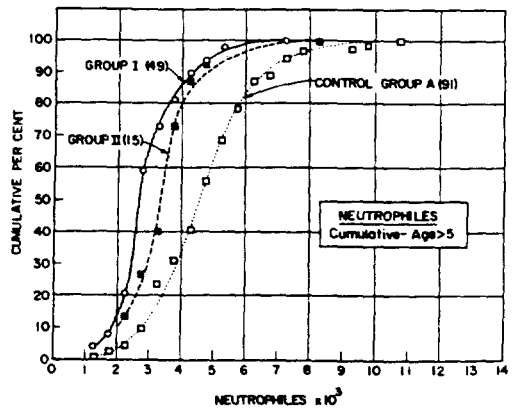

Fig. 7. Cumulative neutrophil counts for Rongelap and Ailinginae at the time of maximum depression compared to comparison population.

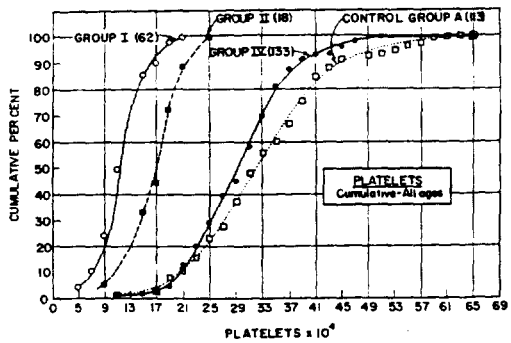

Fig. 8. Cumulative platelet counts for the Rongelap, Ailinginae, Utirik and comparison population at time of maximum depression for the exposed groups.

One case of acute myeloblastic leukemia developed in a boy age 19 who died in 1972. This was probably due to exposure to 1.9 Gy on Rongelap at one year of age. One case of leukemia (age 63) occurred in the comparison group [4].

Symptoms, Skin Lesions and Epilation

As mentioned earlier, the fallout on Rongelap was snow-like and extensive. It was mist-like on Ailinginae and Rongerik. It was invisible on Utirik. During the first two days,

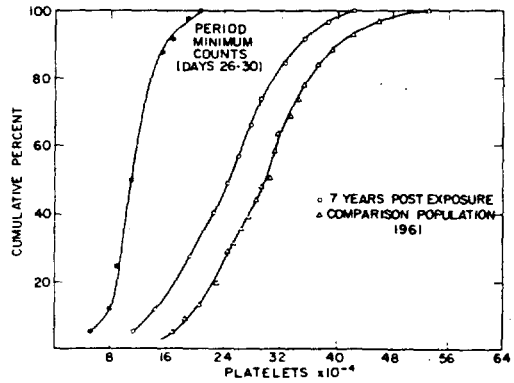

Fig. 9. Cumulative distribution of platelet counts at time of maximum depression and seven years after exposure of the Rongelap people and the comparison population.

about one-fourt
enced itching an
burning of the e
vomiting and
Rongelapese. T
nel on Rongerik
a lesser degree.
ger, took shelt
bathed and char
ing the develo
ninth exposure
conducted exte
there was no ev
temporal sequ
lesions and ep
About two wee
dosed group fr
hyperpigmenta
the early stag
neous lesions.
were experien
severe pain w
lesions were
stages the cut
ized by hyper
raised placque
later tended t
developed a
mented stage
lowed by dr
preceded from
leaving a pink
desquamation
istic appearar
fringed with

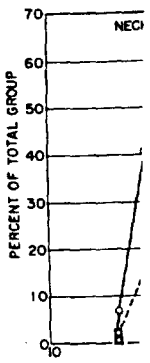

Fig. 10. Tin
epilation for

about one-fourth of the Marshallese experienced itching and burning of the skin and some burning of the eyes with lachrymation. Nausea, vomiting and diarrhea occurred in some Rongelapese. The American military personnel on Rongerik experienced these symptoms to a lesser degree. They were aware of the danger, took shelter in the aluminum buildings, bathed and changed clothes thus greatly reducing the development of skin lesions. On the ninth exposure day, when the medical team conducted extensive physical examinations, there was no evidence of injury to the skin. The temporal sequence in development of skin lesions and epilations is shown in Figure 10. About two weeks after exposure in the higher-dosed group from Rongelap, loss of hair and hyperpigmentation of the skin appeared. During the early stages of development of the cutaneous lesions, itching, burning and slight pain were experienced. With deeper lesions more severe pain was present. The most painful lesions were those on the feet. In the early stages the cutaneous lesions were characterized by hyperpigmented macules, papules or raised placques. They were initially small and later tended to coalesce and the larger lesions developed a dry, leathery texture. The pigmented stage of the superficial lesions was followed by dry, scaly desquamation which preceded from the center of the lesion outward, leaving a pink to white thin epithelium. As the desquamation proceeded outward, a characteristic appearance of a central depigmented area fringed with an irregular hyperpigmented zone was seen. Repigmentation began in the central area and spread outward over a period of many weeks and ultimately, the skin returned to a relatively normal appearance.

A few more serious lesions were seen on the scalp, neck and feet and in one case on the ear. They were characterized by transepidermal necrosis with wet desquamation resulting in crusting ulcerative lesions. Blisters were only observed on the feet which developed rather large bullae. A few of the lesions on the feet became infected, requiring local antibiotics. All lesions healed within about two weeks. Six months after exposure, the skin had returned nearly to normal and by one year pigmentation changes were slight. In addition to the lesions of the skin, a bluish-brown pigmentation of the fingernails became evident on the 23rd post-exposure day. As the nails grew out, the pigmentation moved distally and was not evident a year after exposure.

Epilation was correlated with ulceration of the scalp in most instances and thus was primarily due to beta and low energy gamma irradiation. Regrowth of hair in all individuals commenced during the third month after exposure and at six months there was regrowth of hair normal in color, texture and abundance except in the man that had the severe burn of the ear.

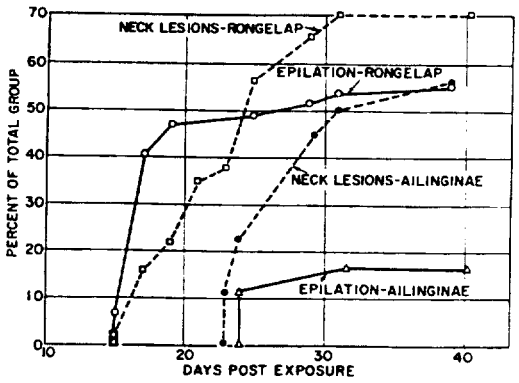

Fig. 10. Time of appearance of skin lesions and epilation for people from Rongelap and Ailinginae.

Therapy of Cutaneous Lesions

In general, treatment was nonspecific. The superficial lesions were treated with calamine lotion with 1% phenol which relieved the itching and burning. In a few instances, pontocaine ointment was used to control the symptoms. During the period of desquamation and oozing, the lesions were washed daily with soap and water and aureomycin ointment was applied. The bullae, if painful, were aspirated with sterile technique followed by a pressure dressing. One case of ulceration was treated with parenteral penicillin for two days. With this one exception systemic antibiotics were not required and were not used.

Thyroid Disease

Hypothyroidism—Cretinism

Two infants exposed on Rongelap became cretins. Their thyroidal estimated dose may have been as high as 200 Gy but more likely was 50

Table II. Thyroid tumors (consensus diagnosis) through 1987

	Adenomatous Nodules	Adenomas	Papillary Cancer	Follicular Cancer	Occult Cancer
Rongelap (67)	17	2	3	—	1
Ailinginae (19)	4	—	—	—	1
Utirik (167)	11	4	4	1	5
Comparison (227)	4	1	2	—	2

Gy. When diagnosed and treated with thyroxin, normal growth ensued [4].

With the development of thyroid tumors, the exposed people were placed on thyroxin to suppress TSH production. Later they were taken off suppressive therapy to measure thyroid function. Twelve individuals were biochemically hypothyroid and of course were replaced on thyroid hormone therapy to suppress development of thyroid neoplasms after the study was complete [4].

Thyroid Tumors

Table II lists the types of thyroid tumors detected and removed, and the pathologic diagnosis made by consensus of six endocrine pathologists. Of the 53 thyroid tumors diagnosed, 15 were cancer [3, 4].

Adenomatous nodules began appearing about 10 years after exposure in the individuals who had thyroidal doses greater than 3000 rem (Fig. 11). With time the number of cases diminished with none being observed in the 34th year after exposure.

There was a correlation between age at exposure and the appearance of thyroid nodules and cancer. Those less than ten years of age at exposure had the highest incidence in all exposure groups [3].

Figure 12 illustrates the correlation between absorbed dose of radiation in the thyroid and percent of population in each dose category for all groups that developed benign nodules, hypothyroidism and thyroid cancer. The fraction developing benign nodules increased with dose up to an average dose of 40-50 Sv. The fraction with cancer peaks at 20-30 Sv. Hypothyroidism peaks at around 30-40 Sv.

Tables III-V show the estimated average and maximum thyroidal dose for the persons exposed on Rongelap, Ailinginae and Utirik atolls. In all cases the dose increases with decreasing age. On Rongelap for example, the average dose in adults was 10 Gy with a maximum of 40 Gy, whereas in the one-year-old, the average was 50 Gy with a maximum of 200 Gy.

The younger the person the smaller the thyroid, and with approximately the same burden of radioiodine, the dose will be proportionally greater.

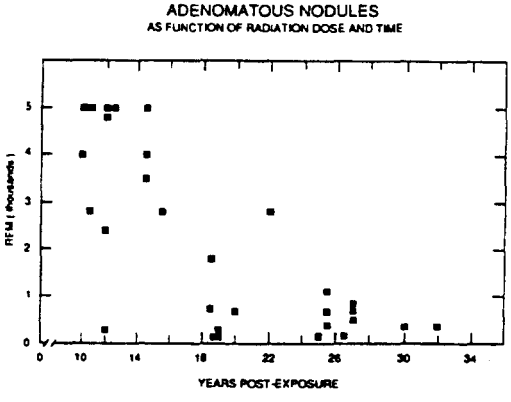

Fig. 11. Temporal appearance of adenomatous nodules of the thyroid as a function of thyroidal dose (divide dose in rem by 100 for Sv).

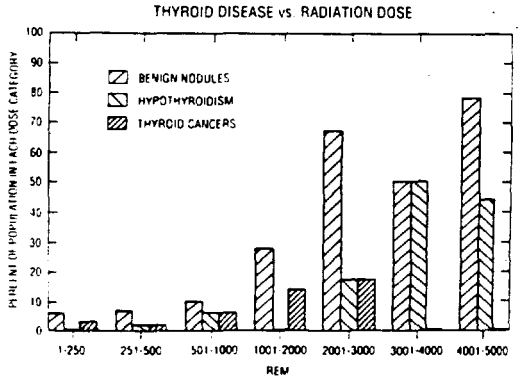

Fig. 12. Thyroid absorbed dose and the fraction of individuals with a given thyroidal dose that develop benign nodules, thyroid cancer or hypothyroidism (divide dose in rem by 100 for Sv).

Table III. Total thyroi

	Ronge
Age	
Adult Male	
Adult Female	
Fourteen-Year-Old	
Twelve-Year-Old	
Nine-Year-Old	
Six-Year-Old	
One-Year-Old	
Newborn	
In Utero, 3rd tri.	
In Utero, 2nd tri.	
Adult Male	
Adult Female	
Fourteen-Year-Old	
Twelve-Year-Old	
Nine-Year-Old	
Six-Year-Old	
One-Year-Old	
Newborn	
In Utero, 3rd tri.	
In Utero, 2nd tri.	

Table IV. Total thyroid

	Sifo
Age	
Adult Male	
Adult Female	
Fourteen-Year-Old	
Twelve-Year-Old	
Nine-Year-Old	
Six-Year-Old	
One-Year-Old	
Newborn	
In Utero, 3rd tri.	
In Utero, 2nd tri.	
Adult Male	
Adult Female	
Fourteen-Year-Old	
Twelve-Year-Old	
Nine-Year-Old	
Six-Year-Old	
One-Year-Old	
Newborn	
In Utero, 3rd tri.	
In Utero, 2nd tri.	

Table III. Total thyroid absorbed-dose estimate

Rongelap Island		
	Average Estimate, Gy	
Age	Internal	Total
Adult Male	10.00	12.00
Adult Female	11.00	13.00
Fourteen-Year-Old	14.00	16.00
Twelve-Year-Old	16.00	18.00
Nine-Year-Old	20.00	22.00
Six-Year-Old	24.00	26.00
One-Year-Old	50.00	52.00
Newborn	2.50	4.40
In Utero, 3rd tri.	6.80	8.70
In Utero, 2nd tri.	—	—
	Maximum Estimate, Gy	
Adult Male	40.00	42.00
Adult Female	44.00	46.00
Fourteen-Year-Old	56.00	58.00
Twelve-Year-Old	64.00	66.00
Nine-Year-Old	80.00	82.00
Six-Year-Old	96.00	98.00
One-Year-Old	200.00	200.00
Newborn	10.00	12.00
In Utero, 3rd tri.	27.00	29.00
In Utero, 2nd tri.	—	—

Table IV. Total thyroid absorbed-dose estimate

Sifo Island		
	Average Estimate, Gy	
Age	Internal	Total
Adult Male	2.80	4.00
Adult Female	2.90	4.10
Fourteen-Year-Old	4.10	5.30
Twelve-Year-Old	4.50	5.70
Nine-Year-Old	5.40	6.60
Six-Year-Old	6.40	7.60
One-Year-Old	13.00	14.00
Newborn	—	—
In Utero, 3rd tri.	—	—
In Utero, 2nd tri.	4.90	6.10
	Maximum Estimate, Gy	
Adult Male	11.20	12.00
Adult Female	11.60	13.00
Fourteen-Year-Old	16.00	17.00
Twelve-Year-Old	18.00	19.00
Nine-Year-Old	22.00	23.00
Six-Year-Old	26.00	27.00
One-Year-Old	52.00	53.00
Newborn	—	—
In Utero, 3rd tri.	—	—
In Utero, 2nd tri.	20.00	21.00

Table V. Total thyroid absorbed-dose estimate

Utirik Island		
	Average Estimate, Gy	
Age	Internal	Total
Adult Male	1.50	1.60
Adult Female	1.60	1.70
Fourteen-Year-Old	2.20	2.30
Twelve-Year-Old	2.40	2.50
Nine-Year-Old	3.00	3.10
Six-Year-Old	3.40	3.50
One-Year-Old	6.70	6.80
Newborn	0.48	0.59
In Utero, 3rd tri.	0.98	1.10
In Utero, 2nd tri.	2.60	2.70
	Maximum Estimate, Gy	
Adult Male	6.00	6.10
Adult Female	6.40	6.50
Fourteen-Year-Old	8.80	8.90
Twelve-Year-Old	9.60	9.70
Nine-Year-Old	12.00	12.00
Six-Year-Old	14.00	14.00
One-Year-Old	27.00	27.00
Newborn	1.90	2.00
In Utero, 3rd tri.	3.90	4.00
In Utero, 2nd tri.	10.00	10.00

Thyroidal dose was based on the distribution of the iodine family of radioisotopes in fission products, their decay rates, arrival of fallout, time on the contaminated atolls, thyroid size as a function of age, the excretion of ^{131}I in 24 h urine samples at 17 days, the biological half-life and the measured ^{131}I in air samples. The major route of radioisotopes into the body was via ingestion. Inhalation was a minor route. Details of the dose reconstruction from all of the factors enumerated above are considered in detail by *Lessard et al.* [3].

Other Radioisotopes

Extensive studies were performed on the urine of the exposed persons, tissues of pigs and chickens and newly-laid eggs. Details of animal studies are published [1]. Briefly, autoradiographs of bones were positive and the intensity of radioactivity in eggs diminished with successive eggs.

The mean body burdens of the Rongelap group at 82 days was based on measurements

Table VI. Mean body burden of the Rongelap group

Radioisotope	μCi at 82 days	USNRDL μCi at 1 day	LASL μCi at 1 day
^{90}Sr	0.19	1.6	2.2
^{140}Ba	0.021	2.7	0.34
Rare earth group	0.03	1.9	—
^{103}Ru	—	—	0.013
^{45}Ca	0	0	0.016 (μgm)

of urine concentration and the one-day burden calculated on the basis of published data on ^{90}Sr excretion in a human being [5]. The activity at one and 82 days post-accident is presented in Table VI. Details of the measurements and calculations are presented [1].

The Rongelap people had been living on another atoll and in 1957, it was deemed safe to return them to Rongelap. In 1958, whole body counts detected ^{137}Cs [6]. The body burden leveled out at about 25,000 Bq. remaining at this level for about 2500 days, and thereafter diminished so that at 8000 days past rehabitation, it was down to 5000 Bq. and at 9800 days the level was about 2500 Bq. The changes in level of ^{137}Cs body burden are shown in Figure 13.

In 1985, the Rongelapese were removed from Rongelap and placed on Kwajalein atoll because the inhabitants believed radiation was causing all of their health problems [7].

The cumulative dose to the Rongelapese from 1957-1985 was about 5 rem, as compared to U.S. exposure from natural background 0.05 Sv in 25 years at sea level and 0.07 Sv on the Colorado plateau.

Today the radioisotopes that contribute to the dose to individuals who might reside on Rongelap are ^{90}Sr, ^{137}Cs, ^{239}Pu, ^{240}Pu and ^{241}Am. ^{137}Cs would contribute 90% of the dose; ^{90}Sr, 2-5%; and transuranic nuclides less than 5%. The integrated annual dose would be in the vicinity of 0.001 Sv [7].

Longevity

Figure 14 shows the survival curves for the people exposed on Rongelap, Ailinginae, Utirik and Rongelap unexposed people. There is no significant difference. Actually of those exposed on Rongelap and Ailinginae, there was a slightly higher fraction alive in 1986 [4].

Conclusions

1 Unique fallout because of nature of coral atoll - $CaCO_3 \rightarrow CaO \rightarrow Ca(OH)_2$.

2 Nausea, vomiting and diarrhea induced in about 25% of the exposed persons by whole body exposure to 1.9 Gy.

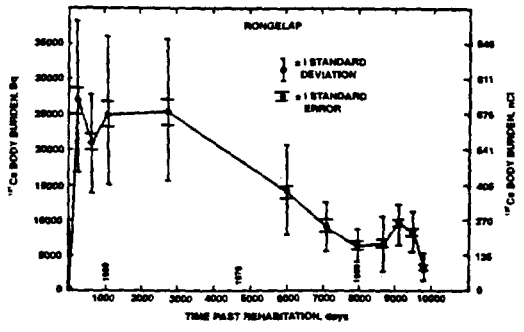

Fig. 13. Body burden of ^{137}Cs in the Rongelapese after their return to Rongelap in 1957 and for the ensuing 28 years.

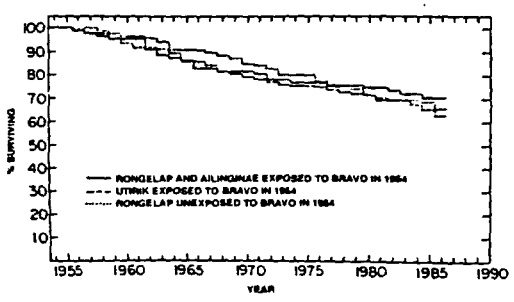

Fig. 14. Survival curves for Marshallese exposed on Rongelap, Ailinginae, Utirik and Rongelap unexposed people.

3 Hematologic effects, distinct mild pancytopenia - an increment of a few 0.1's of Gy - would have placed people of Rongelap in the lethal range.

4 Residual hematologic effect detectible for many years, particularly lymphocytes and platelets.

5 Skin lesions prevalent - mild to ulcerative - due to fallout on skin. No late effects observed as of 1993.

6 Thyroid disease - hypothyroidism and thyroid tumors - are the major long-term effects.

7 One case of acute myelocytic leukemia, probably due to the exposure to 1.9 Gy gamma radiation.

8 The Marshallese population is the most intensively and carefully studied population exposed to whole body ionizing radiation. They have been examined every 6-12 months for 40 years.

Acknowledgments

This research was supported by the U.S. Department of Energy under Contract DE-AC02-76CH00016.

References

1 Cronkite EP, Bond VP, Dunham CL. Some effects of ionizing radiation on human beings. A report on the Marshallese and American accidentally exposed to radiation from fallout and a discussion of radiation injury in the human being. U.S. Atomic Energy Commission. 1956. TID 5358 Superintendent of Documents U.S. Government Printing Office. Washington, D.C.

2 Cronkite EP, Bond VP, Conard RA et al. Response of human beings accidentally exposed to fallout. JAMA 1955:150:430-434.

3 Lessard E, Miltenberger R, Conard R et al. Thyroid absorbed dose for people at Rongelap, Utirik and Sifo on Mar 1, 1954. Brookhaven Nat'l. Lab. BNL 51882 UC-48 Biology and Medicine TIC 4500 available from National Technical Information Service U.S. Dep't Commerce 5285 Port Royal Road, Springfield, VA 22162 U.S.A.

4 Adams WH, Heotis PM, Scott WA. Medical status of Marshallese accidentally exposed to 1954, Bravo fallout radiation Jan 85 through Dec 1987. Brookhaven Nat'l Laboratory Report BNL 52192 UC-408. DOE/OSTL-4500-Interim 3. Brookhaven National Laboratory, Upton, NY 11973.

5 Cowan FP, Farrabee LB, Love RA. Health physics and medical aspects of a 90Sr inhalation incident. American J Roent Rad Therapy and Nuc Med 1952:67:805.

6 Conard RA. Fallout—The experiences of a Medical Team in the case of Marshallese population accidentally exposed to fallout radiation. Brookhaven Nat'l. Lab. report 46444, Upton, NY 11973. Available from National Technical Information Service, U.S. Dep't. Commerce 5285, Port Royal Road, Springfield, VA 22161.

7 Radiological Assessments for Resettlement of Rongelap in the Republic of the Marshall Islands. National Academy of Sciences Press, 2101 Constitution Ave, N.W. Box 285, Washington, D.C. 20418.