Radiation effects in the Marshall Islands

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ABSTRACT

On March 1, 1954, the detonation of a thermonuclear device on Bikini in the Marshall Islands resulted in the accidental deposition of fallout on several inhabited atolls and on a Japanese fishing vessel. The accident was unusual in that the explosion, occurring near ground level, resulted in a heavy, particulate fallout on Rongelap and Ailingnae, about 160km away. Utirik, further to the east, experienced a lesser, invisible fallout. Early radiation effects were observed in many of the 64 inhabitants of Rongelap and the 18 on nearby Sifo Island. During the second and third decades after the accident, most of the Rongelap children and many adults developed thyroid nodules, some of which proved to be malignant. In addition, thyroid atrophy accounted for severe growth retardation in 2 boys. The Utirik people (167 exposed) did not show early radiation effects, but thyroid nodules and thyroid cancer began to appear late in the second decade after exposure. The cancers in both groups were of the papillary type. There have been no deaths from thyroid cancer but one fatal case of acute myelogeneous leukemia occured in a Rongelap boy. The radiation exposure resulted from both internal and external sources. Calculation of risk coefficients for thyroid nodules and cancer, adjusted for their occurrence in a comparison population, gave a mean nodule risk (all ages) of 8.3 per 10^6 person. rad. year. and a mean cancer risk of 1.5 per 10^6 person. rad. year. The latter value is similar to that resulting from purely external exposure (eg medical x-rays) and is compatible with an equal risk from external x-rays and internal radiation from short half-life isotopes of iodine. The Marshall Islands experience clearly identifies the risk to the thyroid gland from radioactive iodine fallout in man; however, it gives no information about the risk that can be attributed to iodine-131, the predominant iodine isotope in long-range fallout and in medical use.

INTRODUCTION

An unusual radiation accident occurred on March 1, 1954, in the Marshall Islands of the Micronesian archipelago. A thermonuclear device detonated on a tower at the testing site on Bikini produced a 3-fold greater than anticipated yield, about 15 megatonnes, and an unexpected wind shear condition resulted in heavy fallout outside the test area. About 160km eastward, and 4 - 6 h after the explosion, the radioactive cloud deposited particulate, ash-like material on 64 inhabitants of Rongelap, on 18 Rongelapese on Sifo Island of the nearby Ailingnae Atoll and on 23 fishermen on a Japanese vessel, Fukuryu (Lucky Dragon) Maru 5. The fallout persisted for about 5 - 10 h (1). Slightly to the east, 28 American servicemen on Rongerik were exposed, and after 22h the cloud reached Utirik where 167 people were affected by a much decreased, invisible fallout. About 2 days later, the Marshallese were evacuated to Kwajalein where they were decontaminated. The Utirik people were able to return home soon thereafter, but it was 3 years before the radiation on Rongelap decayed to a safe level. The American servicemen were taken to Hawaii for examinations and then returned to duty. The fishermen on the Fukuryu, their exposure unbeknown to the authorities, sailed for 14 days before arriving at their home port of Yaizu and were hospitalized in Tokyo for about 2 months.

From the time of this unfortunate incident to the present, the exposed Marshallese have been under close medical observation and a number of reports on their health status have been published (2-5). Since 1956, this has been the responsibility of the Brookhaven National Laboratory (BNL) and was under the direction of Dr. Robert A. Conard until his retirement in 1980. An age- and sex-matched comparison group of unexposed Marshallese has also been monitored to obtain information on the baseline incidence of disease, in order to identify unexpected consequences of radiation exposure in the Rongelap and Utirik populations. Although this unique experience has revealed much about radiation-induced disease, it has also become clear that it may not be a precise model for other radiation accidents.

One difficulty has been a persistent uncertainty about the exact radiation exposure received by the populace. As is so often the case when an accident is not anticipated, the observers were unprepared to make the immediate measurements that are required for complete accounting. For example, the major datum from which the thyroid burden was derived is the analysis of pooled urine iodine-131 obtained 17 days after the exposure.

A thorough reevaluation of the thyroid-absorbed dose was recently completed by Lessard et al (1) of the Safety and Environmental Protection Division of BNL. It was extrapolated from the urine results and from several measured and derived quantities relating to fallout arrival and observation times, size and nuclide composition of the fallout, exposure-rate measurements, and diet and living patterns. Although the recalculated cumulated external doses of γ-rays were very close to the initial estimates (Table 1), the recalculated doses from the internal radionuclide burden were much higher. The latter were derived from the estimated thyroid content of 5 iodine radioisotopes and 2 tellurium

TABLE I

CUMULATED WHOLE-BODY DOSE ACCORDING TO LOCATION[a)]

Location	Cumulated whole-body dose (rad)[b)]	
	1955 Sondhaus	1984 Lessard
Rongelap	175	190
Ailingnae	69	110
Utirik	14	11
Rongerik	78	81

a) Source: Lessard et al (1)
b) 1 rad = 0.01 Gy.

TABLE II

TOTAL INTAKE OF RADIOACTIVITY FROM IODINE AND TELLURIUM ISOTOPES ACCORDING TO LOCATION[a)]

Radionuclide	$t_{1/2}$	Total intake of radioactivity from iodine and tellurium isotopes[b)] (μCi)		
		Rongelap	Ailingnae	Utirik
Iodine-135	6.7	1900-3500	670-1200	79-140
Iodine-134	0.8	670-1200	430-780	-
Iodine-133	20.8	1200-2100	310-570	160-320
Iodine 132	2.3	310-560	67-120	60-110
Iodine-131	193.0	53-96	11-20	13-23
Tellurium-132	78.0	300-550	72-130	57-110
Tellurium-131m	29.0	44-81	13-24	8-17

a) Source: Lessard et al (1).
b) The intake varied with age and sex; the lower value is for 1-year olds and the higher is for adult males.

radioisotopes (Table 2). The resulting total thyroid doses are given in Table 3. Lessard et al (1) give values for 9 different age groups, and for adult males and females, on the 3 islands inhabited by the exposed Marshallese. For the sake of simplicity, we have recorded only a selection of these values in Tables 2 and 3.

It is important to note that the majority of the thyroid-absorbed dose was

TABLE III
TOTAL THYROID ABSORBED-DOSE ESTIMATE ACCORDING TO LOCATION AND AGE[a)]

Location	Age	Total thyroid absorbed-dose estimate (rad) Average dose[b)]			Maximum dose[c)]		
		Internal	External	Total	Internal	External	Total
Rongelap	Adult	1000	190	1200	4000	190	4200
	9 year	2000	190	2200	8000	190	8200
	1 year	5000	190	5200	20000	190	20000
	Newborn	250	190	440	1000	190	1200
	In utero[d)]	680	190	870	2700	190	2900
Ailingnae	Adult	280	110	400	1120	110	1200
	9 year	540	110	660	2200	110	2300
	1 year	1300	110	1400	5200	110	5300
	In utero[e)]	490	110	610	2000	110	2100
Utirik	Adult	150	11	160	600	11	610
	9 year	300	11	310	1200	11	1200
	1 year	670	11	680	2700	11	2700
	Newborn	48	11	59	190	11	200
	In utero[d)]	98	11	110	390	11	400
	In utero[e)]	260	11	270	1000	11	1000

a) Source: Lessard et al (1)
b) Based on the average iodine-131 activity in urine collected from people exposed on Rongelap.
c) Based on the range of cesium-137 body burdens during protracted exposure, the weight of stomach contents in cases of sudden death, and other criteria, a value of 4 for maximum dose/average dose attributed to ingestion was assumed.
d) 3rd trimester.
e) 2nd trimester.

from short-lived isotopes. The ratio of the dose from these radionuclides to the dose attributed to iodine-131 (for an adult male) was 7.7 on Rongelap, 10 on Ailingnae and 4.7 on Utirik (6). It is also significant that only a minor portion of the thyroid dose could be attributed to inhalation and a negligible amount to β-particle irradiation from skin deposits. Thus, the internal thyroid dose was almost entirely due to ingested radionuclides.

In addition to the radioactive isotopes of iodine, the fallout contained a very large number of other fission products, some neutron-induced isotopes, but little fissile material. Among the most important were strontium-89, barium-140, rare-earth isotopes (including cerium, lanthanum, terbium, yttrium), rubidium-103 and calcium-450.

Several long-lived isotopes (strontium-90, cesium-137, zinc-65, and cobalt-60) persist in the environment (7, 8), but the resulting low levels of radiation are not believed to have contributed to the late radiation effects in the exposed Marshallese people.

Early Radiation Effects and Recovery

The intensity of the fallout radiation on Rongelap, Sifo and Rongerik is evident from the early radiation effects. These have been extensively reported in the BNL reports and are summarized in the 20-year and 26-year reviews (2, 3). On Rongelap, about two-thirds of the people developed anorexia and nausea and one-tenth had vomiting and diarrhea. In children aged under 5 years, 85% had nausea and 38% had vomiting. Itching and burning of exposed skin was noted by one-fourth of the people. Although the gastrointestinal symptoms lasted only about 2 days, skin burns appeared after 12 - 14 days in about 90% of the Rongelap inhabitants, with ulcerations in 15%. Epilation occurred in 28% of the adults and in almost all children, in many of whom it was extensive.

Lymphopenia (about 50% of the comparison level) was evident when first examined 3 days after exposure. Thrombocytopenia was maximal (about 30% of comparison) by 4 week. Neutropenia was evident by 5 - 6 weeks and reached about 50% of the comparison level. No consistent erythropoietic depression was observed. No bleeding or infections developed even at the lowest observed levels of platelets (35000) or neutrophils ($\leq$1000), and no transfusions or antibiotic therapy were given.

In the ensuing weeks, bone marrow depression showed rapid recovery, and then gradual near-normalization occurred during the first year. The skin lesions also healed rapidly, with repigmentation and regrowth of hair, leaving residual hyperpigmentated plaques and nevi in some skin areas. Careful reexaminations revealed no cases of skin cancer until 1986 when one patient had a basal cell epithelioma removed from a healed skin lesion.

During the first decade after the exposure, the general health of the exposed

Marshallese appeared to be no different from that of the unexposed Marshallese. Apart from the skin effects, a lag in complete recovery of peripheral blood elements, and slight chromosome abberrations in peripheral blood cells, the only effect attributed to radiation (9) was an apparent increase in miscarriages and stillbirths in Rongelap exposed people during the first 5 years. However, the small numbers and the inability to evaluate early pregnancy terminations makes it difficult to be certain of this interpretation. Growth retardation observed in some of the children was ultimately explained by effects on thyroid function.

Late Radiation Effects

Nine years after the accident, a 12-year-old Rongelap girl was found to have developed a nodule in her thyroid gland. Within the next 3 years, 15 of the 22 Rongelap people who had been under age 10 years at the time of exposure had developed thyroid lesions. At that time, the first thyroid nodule in an exposed Rongelap adult appeared and in 1969, 15 years after the accident, the first thyroid nodule appeared among the exposed people of Utirik. It has become evident that thyroid abnormalities - which include benign and malignant thyroid tumors and thyroid failure - are the major late effects of the radiation received by the exposed Marshallese.

A few other late radiation effects - or possible radiation effects - have been noted. In 1972, a 19-year-old man, who was 1 year old when he was exposed and who had previously been treated for benign thyroid nodules, developed fatal acute myelogenous leukemia. It was estimated (3) that the chance that this case was radiation induced rather than spontaneous ranged from 5:1 to 15:1. In 1974, a 64-year-old Rongelap man died of stomach cancer, and in 1982, a Utirik woman, exposed at age 15 years, had surgical removal of a cranial meningioma which later recurred and caused her death. Nonlethal tumors possibly related to the radiation included a neurofibroma in the thyroid area of a Rongelap woman found at initial surgery, and single cases of successfully treated breast cancer and colon cancer in Utirik women. Since 1976, 2 exposed women, and possibly a 3rd have developed pituitary tumors (3, 5, 10). Screening for hyperprolactinemia revealed 1 other case with persistent prolactin elevation. The relation of these cases to radiation exposure is certainly not established. Deaths from radioinducible-type tumors have also occurred in the unexposed population during the 30-year follow-up, including an astrocytoma of the spinal cord, a bronchogenic carcinoma, a mammary carcinoma and a case of acute leukemia.

Thyroid Hypofunction

The first thyroid abnormality to appear in the exposed Marshallese people was radiation-induced thyroid atrophy, resulting in profound growth failure in two

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boys. However, the etiology was not recognized until after thyroid nodules began to appear. The reason was that the diagnosis of hypothyroidism, at that time based on PBI measurement, was obscured by an elevated iodoprotein level, later found to be prevalent in the Marshall Islands (2, 3, 11). Subsequent surveys by TSH measurement and TSH response to TRH, in addition to routine measurement of thyroid hormone levels, revealed 12 cases of subclinical thyroid hypofunction (12) that could not be attributed to prior thyroid surgery. A high proportion of the Rongelap people who were aged under 10 years when exposed had the most marked TSH elevations, which probably favored the development of thyroid nodules (4). Although the finding of hypothyroidism was surprising at first, reevaluation of the thyroid absorbed radiation dose by Lessard et al (1) now shows that the exposure was in a range known to be capable of causing thyroid failure.

Thyroid Neoplasia and Thyroid Nodules

As already mentioned, the development of nodular thyroid disease has been the major late effect of the radiation exposure of the Marshallese in terms of the number of people affected. It has also been the major cause of late morbidity, mainly because of the surgery required for diagnosis and management. However, there have been no deaths from thyroid cancer and no cases with metastases other than in cervical or mediastinal lymph nodes. Whether this is the result of early diagnosis and treatment, the age at which tumors developed, or other factors, is not known.

The prevalence, after 30 years, of thyroid nodules and thyroid cancers as a function of age in the exposed groups, and at all ages in the comparison group, are presented in Table 4. The histological diagnosis are the result of reevaluation of the pathological material by a panel of experienced thyroid pathologists. It is evident that the occurrence of nodules in all the exposed groups exceeded that in the comparison population. An excess of thyroid cancers also was seen in the Rongelap and Utirik exposed people. All were classified as papillary carcinoma, and occult carcinoma was excluded from the analysis. The time after exposure at which these nodules and cancers appeared are presented in Fig 1. After a latent period of 9 years, thyroid nodules began to accumulate in the Rongelap people who were exposed before the age of 10 years. The first thyroid cancer was diagnosed 11 years after exposure in a Rongelap woman who was 30 years old at the time of the accident. The first thyroid nodule in the Utirik people was detected 15 years after exposure and was the case in which opinion was divided between follicular carcinoma and atypical adenoma. In recent years, more cases have occurred in the Utirik people than in Rongelap people, perhaps because there are more individuals who remain at risk.

TABLE IV

THYROID LESIONS THROUGH 1986[a]

Age at exposure (years)	Total number	Thyroid atrophy	Thyroid nodule(s)[b]	Thyroid cancer[c]	Expected nodules[d]	Expected cancers[d]
Rongelap						
In utero	3	0	2	0	0.079	0.026
< 10	21	2	15	1	0.50	0.17
10-18	12	0	3	2	0.92	0.15
> 18	31	0	3	2	2.7	0.21
Sifo						
In utero	1	0	0	0	0.026	0.0087
< 10	7	0	2	0	0.18	0.061
10-18	0	-	-	-	-	-
> 18	11	0	3	0	0.98	0.075
Utirik						
In utero	8	0	0	0	0.21	0.070
< 10	56	0	8	1	1.5	0.49
10-18	19	0	7	2	1.4	0.24
> 18	84	0	8	1	7.5	0.58
All exposed	253	2	51	9	16	2.1
Comparison[e]	227	0	10	2	-	-

a) The number of cases in each category are listed - not the number of lesions, which were often multiple.

b) Excludes 3 exposed cases and 5 unexposed cases not surgically treated. Of the nodules not diagnosed as cancer, 4 cases (2 Rongelap and 2 Utirik) were classified as adenoma. The remainder were classified as adenomatous nodules.

c) All cases listed were papillary carcinoma. In one additional case opinion was equally divided between follicular carcinoma and atypical adenoma. Not included are 4 cases (1 Sifo, 3 Utirik) of occult papillary carcinoma in the exposed population and 2 cases in the comparison group.

d) Based on unexposed Marshallese, as reported by Lessard et al.(1).

e) Age- and sex-matched unexposed Rongelap people, including additions to replace those lost to follow-up.

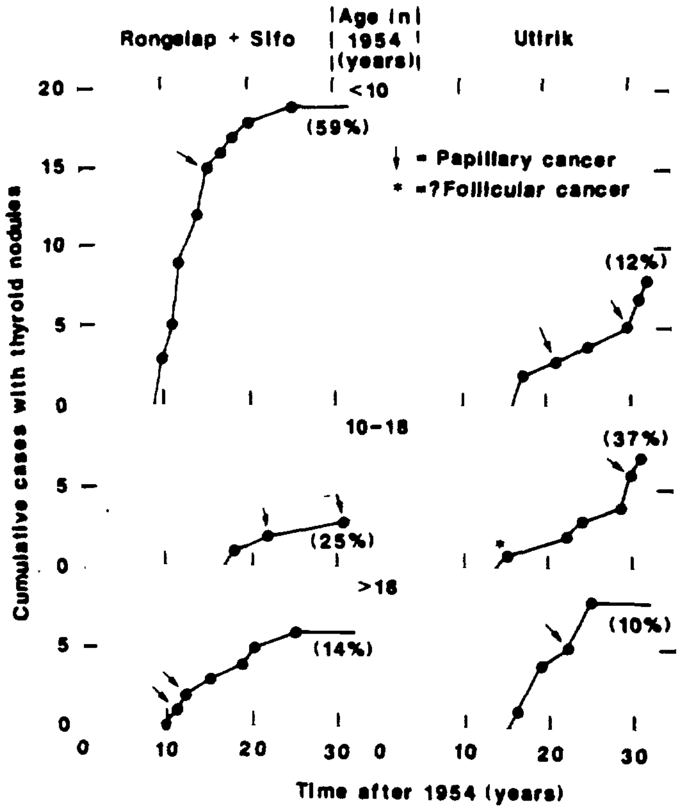

Fig 1. The accrual of cases with thyroid nodules and thyroid cancer in the exposed Rongelap people as a function of age of exposure in 1954. The group aged <10 years includes exposure in utero. Cases of thyroid atrophy without nodule formation (2 Rongelap boys aged under 10 years) are excluded. Each arrow indicates a case of papillary cancer. The asterisk indicates a case of possible follicular cancer (see text).

More females than males developed thyroid nodules, the ratio being about 3:1, and all the thyroid cancers in the exposed people occurred in women. This preponderance of thyroid nodules and cancer in women is the same as that seen in most other populations, with or without prior radiation exposure. The prevalence of cancers compared to benign nodules (18%) in the Marshallese is, however, lower than that seen after medical x-ray therapy (approximately 30%)(13).

Lessard et al (1), using their recalculated thyroid-absorbed dose, have reevaluated the risk of development of thyroid nodules and thyroid cancer in the Marshallese people. They also evaluated separately the risk from internal and external radiation (7). The mean risk coefficients for the age and island groups are presented in Table 5. These values have a high degree of uncertainty because of the small number of subjects, the standard deviations being in the range of ±150%. It is also to be noted that the incidence of thyroid cancer in the unexposed comparison population is considerably higher than that reported in another sea-level population in the Pacific: 59 cancers

TABLE V

RISK COEFFICIENTS FOR THYROID NODULES AND CANCER[a)]

Age at exposure (years)	Mean absorbed dose (rads)	Mean nodule time at risk (years)	Mean cancer time at risk (years)	Mean nodule risk coefficient (No. per 10^6 person.rad. years)[b)]	Mean cancer risk coefficient (No. per 10^6 person.rad. years)[b)]
Rongelap					
In utero	640	23	-	43.0	-
< 10[c)]	4000	13	15	15.0	0.73
10-18	1700	17	22	6.0	1.9
> 18	1300	17	13	0.44	3.4
All ages	2100	15	16	9.2	1.6
Sifo					
In utero	610	-	-	-	-
< 10	1100	22	-	11	-
> 18	410	18	-	37	-
All ages	670	19	-	20	-
Utirik					
In utero	130	-	-	-	-
< 10	490	25	21	3.6	0.89
10-18	220	22	22	27.0	19.0
> 18	170	22	22	4.8	1.3
All ages	280	23	22	5.9	2.5
All exposed					
In utero	290	23	-	21.0	-
< 10	1400	16	18	10.0	0.65
10-18	790	20	22	9.6	5.1
> 18	470	20	16	4.4	2.2
All ages	800	18	19	8.3	1.5

a) Source: Lessard et al (1).
b) 1 person.rad.year = 0.01 person.Gy.year.
c) Does not include 2 boys with thyroid atrophy.

per 10^4 person.year in the Marshall islands compared to 2 per 10^4 in Singapore (14). The latter value is close to that reported in the USA. If this is used instead of the comparison group, the resulting cancer risk for all Marshallese is 2.1 per 10^6 person.rad.year, 1 compared to 1.5 per10^6 person.rad.year in Table 5, so that a value of about 2 per 10^6 person.rad.year for the Marshallese is a good estimate from the current data. This value is not different from the weighted average of the risk in several exposed populations, including survivors of the Nagasaki and Hiroshima bombs, and persons exposed in childhood to x-ray therapy in the USA (1).

The estimated risk coefficients from external and internal radiation in the Marshallese is given in Table 6 for each age group and for the total exposed population. Two values per group are recorded, based on a 5-year or a 10-year latent period. The ratio of internal to external radiation risk ranges from 0.4 to 6.1 and is 1.5 for the entire population, suggesting that there is no identifiable difference between the risk from internal and external radiation.

TABLE VI

CANCER RISK COEFFICIENTS FOR INTERNAL AND EXTERNAL EXPOSURE ACCORDING TO AGE[a)]

Age at exposure (years)	Cancer risk coefficient (No. per 10^6 person.rad.year)[b]			
	10-Year latent period		5-Year latent period	
	External risk[c)]	Internal risk	External risk[d)]	Internal risk
< 10	3.3	1.4[e)]	2.5	1.0
10-18	3.3	8.0	2.5	6.3
> 18	1.0	6.1	1.3	3.3
Total	2.1	4.7	1.9	2.9

a) Source: Adams et al (5).
b) 1 person.rad.year = 0.01 person.Gy.year.
c) Based on Radioepidemiological Tables (15).
d) Based on NCRP Report 80 (16).
e) When the value 4.9 was used for the external risk coefficient (17), the internal risk coefficient was 1.3.

Relevance of the Marshall Islands Experience

The information collected on the Marshallese people constitutes an important body of data relating to the late effects of radiation absorbed by the thyroid gland. It shows convincingly that the induction of thyroid nodules and thyroid cancer is a major cause of late morbidity. Although it gives us some idea of the risk coefficient for internal radiation, the magnitude is uncertain owing to the lack of precise data about radionuclide intake, and because of the small population involved. Furthermore, the existence of mild hypothyroidism in a significant number of people might have enhanced the radiation effect through increased TSH secretion, a well-recognized cofactor in thyroid radiation effects. On the other hand, the high dose levels could have reduced the risk of thyroid-tumor induction by causing thyroid-cell death. It is also possible that the use of thyroxine therapy to lower TSH, instituted in the Rongelap exposed people in 1965, decreased the risk of thyroid tumorogenesis. Indeed, that was the aim of the therapy.

Nevertheless, these data are unique in providing some indication of the effect of radiation by short-lived isotopes of iodine and tellurium, which contributed 80 to 90% of the absorbed thyroid dose. The risk from external radiation is fairly well known (13, 17), and the risk to the thyroid from short-lived iodine isotopes is thought to be similar to the external radiation risk (17). The Marshallese data are compatible with that view. The data give no information, however, about the risk of radiation by iodine-131. This is the most important cause of thyroid radiation from nuclear accidents where the exposure is more distant from the source. The radiation risk due to iodine-131 is believed to be lower than that due to external radiation, probably because the exposure rate is slower.

As in all analyses of radiation exposure of the thyroid gland, it is very important to continue clinical observation into later life. In the natural history of sporadic thyroid cancer, the degree of malignancy, both by histologic type and by aggressiveness of the more differentiated types, becomes significantly worse after the age of 40 - 50 years (18). For optimal clinical care and for a clearer picture of radiation risk, it is essential to determine whether this is also the case with radiation-induced thyroid cancer. Even though many of the Rongelap people, and almost all of the children, have already had thyroid surgery, most of them have residual thyroid tissue that remains vulnerable to neoplastic change. Despite the apparent decrease in the rate of occurrence of thyroid nodules, continued observation of the exposed Marshallese people must be pursued.

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REFFERENCES

1. Lessard E, Miltenberger R, Conard R, et al. Thyroid absorbed dose for people at Rongelap, Utirik and Sifo on March 1, 1954, BNL 51882. Upton, NY: Brookhaven National Laboratory, 1985.

2. Conard RA, Knudsen KD, Dobyns BM et al. A twenty year review of medical findings in a Marshallese population accidentally exposed to radioactive fallout, BNL 50424. Upton, NY: Brookhaven National Laboratory, 1974.

3. Conard RA, Paglia DE, Larsen PR et al. Review of medical findings in a Marshallese population twenty six years after accidental exposure to radioactive fallout, BNL 51261. Upton NY: Brookhaven National Laboratory, 1980.

4. Conard RA. Late radiation effects in Marshall Islanders exposed to fallout 28 years ago. In: Boice JD, Fraumeni JF, eds. Radiation carcinogenesis: epidemiology and biological significance. New York: Raven Press, 1984; 57-71.

5. Adams WH, Engle JR, Harper JA, et al. Medical status of Marshallese accidentally exposed to 1954 Bravo fallout radiation: January 1983 through December 1984, BNL 51958. Upton, NY: Brookhaven National Laboratory, 1984.

6. Adams WH, Harper JA, Rittmaster RS, et al. Medical status of Marshallese accidentally exposed to 1954 Bravo fallout radiation: January 1980 through December 1982. BNL 51761. Upton, NY: Brookhaven National Laboratory, 1982.

7. Lessard ET, Miltenberger RP, Cohn SH, et al. Protracted exposure to fallout: The Rongelap and Utirik experience. Health Physics 1984; 46: 511-27.

8. Lessard ET, Greenhouse NA, Miltenberger RP. A reconstruction of chronic dose equivalents for Rongelap and Utirik residents - 1954 to 1980. BNL 51257. Upton, NY: Brookhaven National Laboratory, 1980.

9. Conard RA, MacDonald HE, Lowrey A, et al. Medical survey of Rongelap people five and six years after exposure to fallout, BNL 609(T179). Upton, NY: Brookhaven National Laboratory, 1960.

10. Adams WH, Harper JA, Rittmaster RS, Grimson RC. Pituitary tumors following fallout radiation and exposure. J Am Med Assoc 1984; 252: 664-6.

11. Rall JE, Conard RA. Elevation of serum protein-bound iodine level in inhabitants of the Marshall Islands. Am J Med 1966; 40: 883-6.

12. Larsen PR, Conard RA, Knudsen K, et al. Thyroid hypofunction after exposure to fallout from a hydrogen bomb explosion. J Am Med Assoc 1982; 247: 1571-5.

13. Shore RE, Woodard ED, Pasternack BS, Hempelmann LH. Radiation and host factors in human thyroid tumors following thymus irradiation. Helth Phys 1980; 38: 451-65.

14. Lee YS. Thyroid cancers in Singapore 1968-77. Trop Geograph Med 1982; 34: 303-8.

15. Ad Hoc Working Group to Develop Radioepidemiological Tables. Report of the National Institutes of Health, January 4, 1985, NIH Publication No. 85-2748. National Institutes of Health, 1985.

16. NRCP. Recommendations of the National Research Council on Radiation Protection and Measurements, Induction of Thyroid Cancer by Ionizing Radiation, NCRP Report No. 80. Bethesda, Maryland: NRCP, 1985.

17. Committee on the Biological Effects of Ionizing Radiation. The effects on populations of exposure to low levels of ionizing radiation: 1980, National Research Council Report. Washington DC, National Academy Press: 1980.

18. Cady B, Sedgwick CE, Meissner WA, et al. Rist factor analysis in differentiated thyroid cancer. Cancer 1979; 43: 810-20.