AN ASSESSMENT OF THYROID DOSE MODELS USED FOR DOSE RECONSTRUCTION

VOLUME II
A CRITICAL ASSESSMENT OF HISTORICAL THYROID DOSE ESTIMATES FOR MARSHALLESE EXPOSED TO TEST BRAVO FALLOUT

Contract No. 200-2002-00367
Task Order No. 10

A Final Report to the
Centers for Disease Control and Prevention
Atlanta, Georgia 30335

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May 14, 2007

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1.0 REVIEW OF PAST THYROID DOSE ESTIMATES

Total radiation dose to the thyroid from BRAVO fallout is the sum of all external and internal doses that were experienced by persons exposed on Rongelap, Ailinginae, and Utrik in the first 2 to 3 days following BRAVO Shot on March 1, 1954. Thus, contributing to external thyroid exposures were radiation from ground deposition, cloud immersion, and residual skin/clothing contamination.

However, for this report, the principal focus is thyroid exposures from **internalized** radioiodines. In the fission process, the iodine radionuclides (I-131, I-132, I-133, I-134, and I-135) are mainly produced as decay products of the precursor nuclides of Sb and Te. Although these precursors are less volatile than iodine itself, almost all the iodine radionuclides would be expected to condense late in the temperature history of the nuclear cloud, and thus on the surface of the fallout particles.

There are at least 24 radioisotopes of iodine with mass numbers from 117 to 140. All except I-127 are radioactive, with physical half-lives ranging from 1 second (I-140) to 16 million years (I-129). Table 3-1 identifies all important radioiodine fission products, their parents, daughters, and physical half-lives. However, only I-131, I-132, I-133, I-134, and I-135 are sufficiently long-lived or have a sufficiently long-lived fission-fragment precursor (i.e., Te-131m and Te-132) and, therefore, need to be included for thyroid dose assessment.

Table 1. Radioiodine Fission Products, Their Parents, Daughters, and Half-Lives

Mass	Tellurium		Iodine		Xenon		Cesium
129	32 d						
	↓						
	70 s	→	1.6×10^{7} y	→	Stable		
131	30 h	→					
	↓	}	8.02 d	→	Stable		
	25 s	→					
132	78 h	→	2.3 h	→	Stable		
133	63 s	→	20.8 h	→	5.3 d	→	Stable
134	43 s	→	52.5 s	→	Stable		
135	10 s	→	6.7 h	→	9 h	→	2×10^{6} y
137			19 s	→	3.4 s	→	30 y

At time of BRAVO, the ability to assess internal exposure to radionuclides was limited not only by instrumentation, but also by a paucity of human data that support our current understanding of the biokinetics of internalized radionuclides, and the interpretation of urine excreta and other in-vitro bioassay data.

1.1 EARLY THYROID DOSE ESTIMATES

Historically, the only urine sample that has been used to estimate thyroid doses for all three exposure groups was a single pooled urine sample that had been collected between day 16 to day 17 post-detonation from residents exposed on Rongelap Island. Since no urine samples were taken from persons exposed on Ailinginae and Utrik, thyroid dose estimates for these two groups were based on Rongelap data that were adjusted by means of scaling. Furthermore, due to the short half-lives of I-132, I-133, I-134, and I-135, exposure to these radioiodines had to be inferred for all three population groups.

Analysis and interpretation of this single pooled urine sample was described in a brief report authored by Payne S. Harris in 1954, which is enclosed herein as Appendix II-A. In his report, Harris estimated (1) a total intake of 56 μCi for I-131, (2) an intake of 5.1 mCi of "iodine equivalents," and (3) a total internal thyroid dose of 150 rep. In his report (page 6 of Appendix II-A), Harris would conclude the following:

> *As far as acute internal dose is concerned,* ***iodine*** *appears* ***most important*** *in cases of exposure at these early times after detonation. The absolute doses to the thyroid are appreciable but low compared to the partially or totally* ***ablating*** *doses of I-131 used for therapy of hyperthyroidism or carcinoma.* [Emphasis added.]

In time, Payne Harris' original thyroid dose estimate of 150 rep for Group I Rongelapese and his insinuation of this dose as being "…low compared to the partially or totally ablating doses of I-131…" would turn out to be in error.

Embedded in Harris' estimate of an initial intake of 56 μCi of I-131 and a total internal thyroid dose from all radioiodines of 150 rep were several critical model parameters that included the following:

(1) An assumed iodine uptake fraction of 0.2
(2) An estimated/assumed **adult** 24-hr urine excretion volume of 450 ml
(3) An assumed iodine excretion fraction of 0.001 for day 16–17 post-detonation

Of relevance to this report is Harris' assumption of the iodine uptake fraction of 0.2. A search of DOE archived documents indicates that a May 13, 1954, memorandum entitled, *Estimated Radiation Dose to the Thyroid of Natives from Rongelap,* that was written by the Atomic Energy Commission's (AEC's) Gordon M. Dunning to John Burgher (see Appendix II-B) provided the technical basis. In this memo, Dunning states the following:

> *The best estimated percentage absorption and deposition of iodine is* ***yet to be determined.*** *The best estimate I can turn up to date is still the 20% quoted in NBS Handbook 52.* [Emphasis added.]

Harris' original estimates of thyroid doses to Rongelapese were considered scientifically sound, and were cited in a series of medical survey reports issued by Brookhaven National Laboratory

(BNL) between 1955 and 1962 (Cronkite et al. 1955, 1956; Conard et al. 1958, 1959, 1960, 1962, 1963). For example, in the 1958 survey report entitled *March 1957 Medical Survey of Rongelap and Utrik People Three Years after Exposure to Radioactive Fallout*, Conard et al. provided the following assessment of internal radiation exposure, which closely parallels Harris' initial comments:

> *In an acute fallout situation, iodine-131 is probably the most important absorbed isotope to be considered during the early period. In the Marshallese, the 300 rep estimated to have been delivered to the thyroid glands (100 to 150 from I-131 and 175 r from the gamma dose) was* ***far too low to produce any acute effect****. . . .*
>
> *The incidence of leukemia and malignancy would be expected to be relatively low with the dose of radiation received by these people, and a significant number of cases would be seen only in a large population; therefore, the probabilities are good that such effects will* ***not*** *be observed in the Marshallese.* [Emphasis added.]

1.2 REVISED THYROID DOSE ESTIMATES

By 1964, three teenage girls who were exposed on Rongelap 10 years earlier had been diagnosed with thyroid nodules and underwent surgery. At this time, previous BNL medical surveys as early as 1961 had also identified two boys who had been exposed at 15 and 18 months of age and showed severe growth retardation and classical signs of cretinism (Conard et al. 1965).

These unexpected findings triggered an AEC review of earlier thyroid dose estimates, which specifically focused on individuals who had been exposed as children (James 1964). James' dose calculations assumed that the ingestion of I-131 (and other radioiodines) of children was the same 56 μCi body intake calculated for adults by Harris (1954). However, due to smaller thyroid size, James (1964) derived probable thyroid doses between 700 and 1400 rads to Rongelap girls ages 3 to 4 at time of BRAVO. Table 2 summarizes ranges of thyroid doses for external and internal exposures estimated by James (1964).

Table 2. Thyroid Doses (Rads) to Rongelap Girls Age 3–4*
(Source: James 1964)

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

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

In brief, the need to revise thyroid doses to children was triggered by clinical findings in the early 1960s that were inconsistent with the original dose estimates derived by Harris (1954). Important to note, however, is that these revised child thyroid doses were nevertheless based on the original intake value of 56 μCi and the 11.2 μCi thyroid uptake value derived by Harris for adults in 1954 from a single pooled urine sample.

Of interest is that James' revised estimates of child thyroid doses were not accepted by everyone. For example, the following is a handwritten comment that appeared on the face page of one of several 1964 archived reports by James:

> *One of the most flagrant examples of retrospective science ever committed. James didn't have the guts to say 'my figures aren't worth a damn, but you made me do it!'* [signed] *DB*

For verification, a copy of the report's face page and the accompanying handwritten note are reproduced here as Exhibit 1. While the commentator's identity remains uncertain, it is reasonable to conclude that "D.B." was an internal reviewer of the "James Report," who disagreed with the report's findings, and knew or suspected that Ralph James may have yielded to pressure from persons within his organization.

EXHIBIT #1

Cover Page of James (1964) Report Containing D.B.'s Handwritten Note

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UCRL-12273

ESTIMATE OF RADIATION DOSE TO THYROIDS OF THE RONGELAP CHILDREN FOLLOWING THE BRAVO EVENT

401299

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December 16, 1964

ABSTRACT

An estimate is made of the radiation dose to the thyroids of Rongelap children following the Bravo event of March 1, 1954. The available experimental data are used to estimate the dose under two alternate assumptions of mode of intake: (a) all of the intake was by inhalation, and (b) all of the intake was by oral ingestion. It is concluded that the most probable dose to the thyroid of a 3 - 4 year old girl is in the range 700 - 1400 rad.

One of the most eloquent examples of atmospheric science ever committed. James deserves to say, my figures are known as a dream, but you made me do it!

DBS

By 1974, 17 of 19 children (or 89.5%) exposed prior to the age of 10 on Rongelap were diagnosed with thyroid lesions. Clinical tests also revealed metabolic hypothyroidism, as demonstrated by elevated levels of thyroid stimulating hormone (TSH) in 53% of the subjects.

In subsequent years, additional thyroid nodules and carcinomas were observed that not only included persons exposed as adults, but individuals with assumed lower doses who had been exposed on Utrik.

Figure 1 provides a timeline for the surgical removal of palpable thyroid nodules through 1990 for the two groups. A distinguishing feature is the delayed onset and late occurrence of thyroid nodules among Utrik residents when compared to the exposed Rongelap population. Table 3 further defines palpable nodules by type among the three exposure groups.

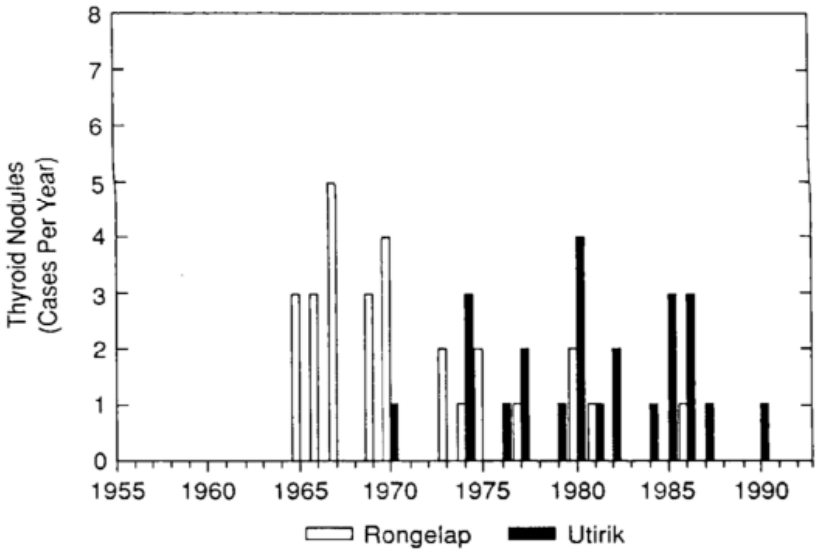

Figure 1. Surgically Confirmed Thyroid Nodule Cases per Year
(Source: Howard et al. 1997)

Table 3. Palpable Thyroid Nodules Diagnosed at Survey through 1990
(Source: Howard et al. 1997)

	Adenomatous nodules	Adenomas	Papillary cancers	Follicular cancers	Occult cancers
Rongelap (67)	17	2	5	—	—
Ailinginae (19)	4	—	—	—	1
Utrik (167)	10	5	4	1	6
Comparison (227)	4	1	2	—	2

Clinical observations made in the late 1970s and early 1980s were once again considered incompatible with existing dose estimates. By 1980, the BEIR III report of the National Research Council's Committee on the Biological Effects of Ionizing Radiation had derived a thyroid cancer risk of 1.89 excess cases per million person-rad-years (PRY). This value was nearly four times lower than the 7.0 derived risk value for Rongelap and nearly 10 times lower than the 17.8 derived risk value for Utrik that had been estimated earlier by Conard (1974).

In a 1974 survey report, Conard (1975), extrapolated the total thyroid doses estimated by James (1964) to older age groups and to people exposed on Sifo Island (Ailinginae) and Utrik Island, as given by the following values:

Group and Age in 1954	Thyroid Dose (rads)
Rongelap:	
1 year	> 1500
2–9 years	800–1500
≥ 10 years	340–800
Ailinginae:	
< 10 years	280–450
> 10 years	140–190
Utrik:	
< 10 years	60–100
> 10 years	30–60

1.3 BROOKHAVEN NATIONAL LABORATORY'S FINAL REVISION OF THYROID DOSE ESTIMATES

DOE's dilemma to periodically revise estimates of thyroid doses was acknowledged by Edward Lessard in an undated, unpublished report issued by BNL (DOE Accession No. 0403041), in which the following statements appear:

> *Variation between atolls in risk of radiation-induced thyroid cancer and the difference when compared to other irradiated groups [e.g., Japanese A-bomb survivors] had become an important scientific and health-related question with the considerable* ***political overtones****. Early in 1977, Bond, Borg, Conard, Cronkite, Greenhouse, Naidu, and Meinhold, all members of Brookhaven National Laboratory (BNL) and Sondhous, University of California, College of Medicine, initiated a reexamination of the technical issues. In 1978, formal program objectives and funding were supplied to BNL by the Department of Energy's Division of Biological and Environmental Research.* [Emphasis added.]

In 1985, Lessard et al. published their report that employed a more comprehensive approach, which included the following explanation (Lessard et al. 1985):

> *…Thyroid absorbed dose tabulated here was estimated from results on* ^{131}I *activity excreted in urine* [Payne Harris' data] *and the specific nuclide composition of BRAVO fallout. Surface and airborne activity, fallout granule*

size, and exposure rate at times after the detonation were developed for 142 nuclides at Rongelap and Utirik on the basis of the reported nuclide composition on day 25 post-detonation. Over 70 documents were reviewed for information regarding exposure-rate readings, film-badge readings, fallout composition, dose and dose rate, body burdens, urine analyses, gastrointestinal tract contents, bone marrow and thyroid dose estimates, and activity measurements in soil, water, marine life, and land animals. Results from the meteorology study and archival soil study were also reexamined and compared to fallout composition results.

A tabulation of the estimates of thyroid absorbed dose, age at exposure and specific nuclides was done for each location. For an adult male, the thyroid absorbed dose from iodine and tellurium nuclides was 7.7 times the absorbed dose due to ^{131}I at Rongelap, 10 times at Sifo Island and 4.7 times at Utirik Island. James [1964] *assumed the total thyroid absorbed dose was 2.6 times the absorbed dose due to ^{131}I (Ja64). The factor 2.6 would be appropriate for slightly older fallout than that experienced at Rongelap, Utirik or Sifo Islands. Thyroid absorbed dose was based on ingestion intake.*

On the basis of this revised approach, Lessard et al. (1985) derived internal thyroid doses for Rongelap, Sifo (i.e., Ailinginae), and Utrik Islands as summarized in Table 4, and concluded the following:

Medical observations concerning thyroid abnormalities have been tabulated along with the new thyroid dose estimated for each person. From these results, the mean cancer risk rate in the exposed population of 251 people was 150 thyroid cancers per million person-gray-years at risk (1.5 ± 2.5 thyroid cancers per million person-rad-years at risk).

The revised Marshall Islander dose estimates corresponded to a new risk coefficient that was now compatible with the existing value of the Japanese A-bomb survivors, as defined by BEIR III (NRC 1980).

Table 4. Summary of Internal Thyroid Absorbed Dose Estimates by Age and Location As Reported by Lessard (1985)

Exposure Group	Rongelap Island (Rads)	Sifo Island (Rads)	Utrik Island (Rads)
Adult Male	1,000	280	150
Adult Female	1,100	290	160
Fourteen-Year Old	1,400	410	220
Twelve-Year Old	1,600	450	240
Nine-Year Old	2,000	540	300
Six-Year Old	2.400	640	340
One-Year Old	5,000	1,300	660
Newborn	250	--	48
In Utero, 3rd trimester	680	490	98
In Utero, 2nd trimester	--	--	260

1.4 CITATION OF LESSARD'S DOSE ESTIMATES IN THE SCIENTIFIC LITERATURE

Although Lessard's dose estimates were reported in 1985 and pre-date the 1990 BEIR V Report by 5 years, the BEIR V Committee Report cites clinical thyroid data reported by BNL 27 years after exposure (Conard 1984) and thyroid dose estimates given in BNL's 26-year medical survey (Conard et al. 1980).

In response to these data, the National Academy of Sciences (NAS) Committee stated the following:

> *The thyroid status of the Marshall Islanders 27 years after exposure is summarized in Table 5-4. Although the dose estimation is* ***open to question****, the prevalence of hypothyroidism, thyroid nodules and proven thyroid cancer all appear to increase with dose . . .* [Emphasis added.]

In spite of the skeptical attitude taken by the NAS Committee, there have been no further reviews or revisions to dose estimates derived by Lessard et al. (1985). Not surprisingly, in subsequent BNL reports as well as journal articles authored by BNL scientists, Lessard's 1985 thyroid dose estimates continue to be cited (Adams et al. 1989; Robbins and Adams 1989; Conard 1992; Howard et al. 1995; Sun et al. 1997; Howard et al. 1997; Musolino et al. 1997, Cronkite et al. 1997).

Numerous references to Lessard's dose estimates can also be found in documents issued by prominent scientific groups, Federal agencies, and by independent scientists. Among these are the following:

- National Council on Radiation Protection and Measurements (NCRP). 1985. Report No. 80, *Induction of Thyroid Cancer by Ionizing Radiation*.
- U.S. Nuclear Regulatory Commission (U.S. NRC). 1985. NUREG/CR-4214 – *Health Effects Model for Nuclear Power Plant Accident Consequence Analysis*.
- National Research Council (NRC). 19994. *Radiological Assessment for Resettlement of Rongelap in the Republic of the Marshall Islands*.
- International Atomic Energy Agency (IAEA). 1998. *Radiological Conditions at Bikini Atoll, Prospects for Resettlement.*
- Agency for Toxic Substances and Disease Registry (ATSDR). 2004. *Toxicological Profile for Iodine*.
- National Cancer Institute (NCI). 2004. *Estimation of the Baseline Number of Cancers Among Marshallese and the Number of Cancers Attributable to Exposure to Fallout from Nuclear Weapons Testing Conducted in the Marshall Islands*.
- Simon and Graham 1996. "Dose Assessment Activities in the Republic of the Marshall Islands." *Health Physics* Vol. 71, No. 4, pp. 438–456.
- Simon et al. 1997. "A Comparison of Independently Conducted Dose Assessments to Determine Compliance and Resettlement Options for the People of Rongelap Atoll." *Health Physics* Vol. 73, No. 1, pp. 133–151.
- Takahashi et al. 1999. "Thyroid Nodules, Thyroid Function, and Dietary Iodine in the Marshall Islands." *International Journal of Epidemiology*, Vol. 28 pp. 742–749.
- Takahashi et al. *Thyroid Disease in the Marshall Islands. Findings from 10 Years of Study* 2001. Tohoku University Press, Jendai, Japan.
- Gilbert et al. 2002. "Health Effects from Fallout." *Health Physics* Vol. 82, No. 5. p726.
- Simon and Bouville. 2002. "Radiation Doses to Local Populations near Nuclear Weapons Test Sites Worldwide." *Health Physics* Vol. 82, No. 5. p706.
- Takahashi et al. 2003. "The Relationship of Thyroid Cancer with Radiation Exposure from Nuclear Weapon Testing in the Marshall Islands." *Journal of Epidemiology*, Vol. 13, No. 2, pp. 99–107.
- Beck et al. 2006. "Review of Dose Estimation for Epidemiological Studies of the Radiological Impact of Nevada Test Site and Global Fallout." *Radiation Research*, Vol. 166, pp. 209-218.

2.0 THE NEED TO RE-EXAMINE THE SCIENTIFIC BASIS OF PAST THYROID DOSE ESTIMATES ASSOCIATED WITH TEST BRAVO.

Since the first estimate of 150 rep to the adult thyroid for persons exposed on Rongelap by Harris in 1954, dose estimates have been revised and expanded to include other Rongelapese based on age and sex, as well as to persons exposed on Ailinginae and Utrik.

It appears that the impetus to revise/expand earlier dose estimates was the unexpected emergence of clinical thyroid pathologies that could not be explained on the basis of prevailing thyroid dose estimates and risk coefficients. In effect, revisions to dose estimates suggest a process of "data fitting," in which a dose was derived that would support the observed clinical data. Thus, various assumptions and model parameters were used in past BNL dose models that were inappropriate, unsupported, or conflicted with known facts.

A review of the scientific literature shows that thyroid dose estimates derived in 1985 by Lessard et al. remain unchanged and continue to be referenced to this day. In this section, key elements of Lessard's dose model are examined and data are presented that support the need to revise dose estimates derived by Lessard et al. (1985).

2.1 AN ASSESSMENT OF LESSARD'S THYROID DOSE MODEL

At the core of Lessard's dose estimate is a single pooled urine sample collected from Rongelapese on March 16 and 17, 1954, and analyzed by radiochemical means, as reported by Harris (1954). A copy of the original Harris report is enclosed herein as Appendix II-A. In his report, Harris derived an intake of 56 μCi I-131 on BRAVO+1 day for the adult Rongelapese, and a peak thyroid content of 11.2 μCi. The assumed peak thyroid content of 11.2 μCi implies a thyroid uptake fraction (i.e., f_2) of 0.2.

In the 1985 report, Lessard et al. provided the following statements that served as the foundation of all derived thyroid dose estimates:

> *…Harris indicated a mean activity of 0.48 kBq (1.31×10-2 μCi) of I-131 in the Rongelap adult 24-hr urine taken on the 17th day post-detonation…and an adult mean peak thyroid content of 414 kBq (11.2 μCi) (Ha54). This peak estimate was calculated on the assumption that 0.1% of stable iodine burden on the first day would be eliminated via urine between the 15th and 17th days…*

On the basis of these data, Harris had derived an intake of 56 μCi I-131 on B+1 day, which for an assumed thyroid uptake fraction of 0.2 corresponded to a peak thyroid content of 11.2 μCi I-131. Important to note here are two issues. First, Lessard et al. accepted Harris' mean activity of 0.48 kBq (1.31 × 10-2 μCi) of I-131 in the Rongelap adult 24-hr urine taken on the 17th day post-detonation without verification.

Embedded in Harris' value (i.e., 1.31×10^{-2} μCi of I-131) are several critical calculational elements that SC&A had challenged in a previous draft report (entitled *Historical Dose*

Estimates to the GI Tract of Marshall Islanders Exposed to BRAVO Fallout) and issued to the CDC for review in August 2006. However, due to the fact that SC&A's draft report has not been peer-reviewed, and for reasons of expediency, potential errors regarding the 1.31×10^{-2} μCi I-131 content in the 24-hr urine will not be discussed herein.

Secondly, and relevant to this report, is Lessard's explanation for raising Harris' initial intake of 56 μCi to 93 μCi I-131, as given by the following:

> *…Two models, one developed by Johnson (1981) and the other by* ***ICRP (ICRP 79),*** *were used to calculate the fraction of an initial I-131 intake by ingestion that would be eliminated on a given day post-intake…On the basis of 0.48 kBq (1.31 x* 10^{-2} *μCi) in adult urine on the* 17^{th} *day post-intake a 3440 kBq (93 μCi) intake was estimated for I-131.* [Emphasis added.]

Thus, Lessard's thyroid dose estimates as given in Table 22 were defined by the ICRP 30 iodine biokinetic model and its attendant dose conversion factors (DCFs), as cited in Table 21 of the BNL report (Lessard et al. 1985). For dose reconstruction, use of the ICRP 30 iodine model, however, requires that the following conditions are met:

- The exposed are healthy euthyroid adults with thyroid weights of 20 g for male and 17 g for female
- Have an average dietary intake of 225 μg per day of stable iodine
- Exhibit an iodine uptake fraction of 0.3.

Presented in Sections 3.0, 4.0, and 5.0 below are historical data which suggest that conditions assumed in the ICRP-30 model were not met for most individuals of the exposed populations. As a result, previously derived thyroid doses are likely to have been substantially underestimated.

3.0 THYROID WEIGHTS AMONG MARSHALLESE AND THEIR IMPACTS ON THYROID DOSE

For reference adult euthyroid males and females, the ICRP assumes a thyroid weight of 20 grams and 17 grams, respectively (ICRP 1975). However, these generic deterministic values represent a wide range of thyroid weights that reflect differences in dietary habits, metabolic health of the thyroid, and possible genetic factors.

Dunning and Schwarz (1981) reviewed thyroid mass among "normal" adults (>18 years) and found that mass varied from a low of 2 grams to 62 grams, with a mean of 18.3 grams. More extensive data that assessed thyroid mass by gender and age include the studies of Mochizuki et al. (1963), Paukow et al. (1985), and Killough and Eckerman (1986).

In the study by Pankow et al. (1985), a total of 1,400 normal U.S. human thyroid glands were taken at autopsy between 1970 and 1980 and evaluated for weight and dimensions. Results of this evaluation showed that there was no statistical difference to results reported in the earlier study by Mochizuki et al. (1963). When the two data sets were combined and treated as one, three distinct adult thyroid gland weights were identified based on age and sex (Table 5).

Table 5. Thyroid Mass by Age and Sex
(Source: Pankow et al. 1985)

Group (yrs.)	Mean wt (g)	Standard Error (g)
Females (20–69)	14.38	0.2
Males (20–69)	16.62	0.2
Males (30–60)	18.69	0.2

Although these studies suggest thyroid mass values that are significantly below those assumed by the ICRP, neck palpation and autopsy data are considered less accurate then ultrasonography. Current ultrasonography has largely replaced palpation, and is considered the preferred method, as stated by the Food and Nutrition Board (FNB 2000):

> *Ultrasonography defines thyroid size much more precisely and reliably. The technology – safe, practical, and easily performed in the field – is replacing palpation in most studies.*

A review of the literature identifies several studies where ultrasonography has also shown thyroid mass values that are consistently lower than what has been assumed by the ICRP for reference man/woman (Table 6).

Table 6. Thyroid Masses for Select Population Groups

Source	Country	Thyroid Mass (g)	
		Men (range)	Women (range)
ICRP 23 (1975)	U.S.	20 g	17
Apostaei and Miller (2004)	U.S.	17.5	14.9
Gutekunst et al. (1986)	Sweden	7.7 (2.5–34)	11.3 (3.3–27.4)
Berghout et al. (1987)	Holland	8.7	12.7

Thyroid Mass among Marshallese

In the early time period following BRAVO, ultrasonography was not available to BNL investigators who conducted periodic medical surveys on the exposed population groups. For this reason, no data are available that specify thyroid mass values among the exposed subjects at time of exposure.

In a recent study by Takahashi et al. (1999), 300 of the 306 subjects from Majuro who had participated in the iodine excretion study were also evaluated for thyroid volume by means of ultrasound examination. These 300 subjects were categorized as follows:

- Normal Iodine Status (n = 232)
  - no thyroid nodules (n = 102)
  - with thyroid nodules (n = 130)

- Moderate to Severe Iodine Deficiency (n = 68)
  - no thyroid nodules (n = 26)
  - with thyroid nodules (n = 42)

The distributions of thyroid volumes for these four groups are shown in Figures 2 and 3. For all 300 subjects, the median volume was 10 ml and the most frequent (i.e., mode) value was 8 ml. Inspection of Figures 2 and 3 shows that there were size differences that reflect the presence of thyroid nodules and dietary iodine deficiency. As expected, the largest thyroid volumes were seen among subjects who were diagnosed with nodules and low iodine excretion levels.

Of relevance to this report are the generally low median and mode values of 10 ml and 8 ml for the entire study cohort, which are about a factor of 2 lower than values assumed by the ICRP model that was used for dose reconstruction by Lessard et al. (1985). For a given intake of radioiodine, the thyroid dose is inversely proportional to the mass of the thyroid. On the reasonable assumption that current thyroid volumes are applicable to the exposed populations at time of BRAVO, Lessard's thyroid dose estimates (based on ICRP values) are underestimated by a factor of 2.

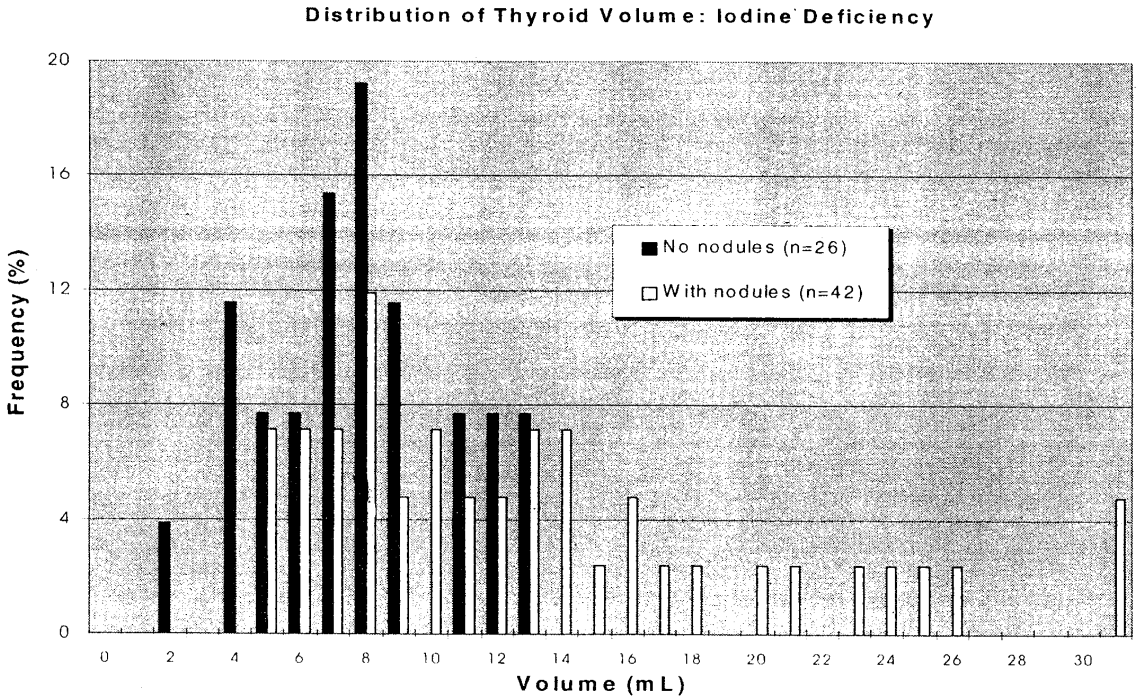

Figure 2. The Distribution of Thyroid Volumes among 68 Study Participants from Majuro with Iodine Deficiency
(Source: Takahashi 1999)

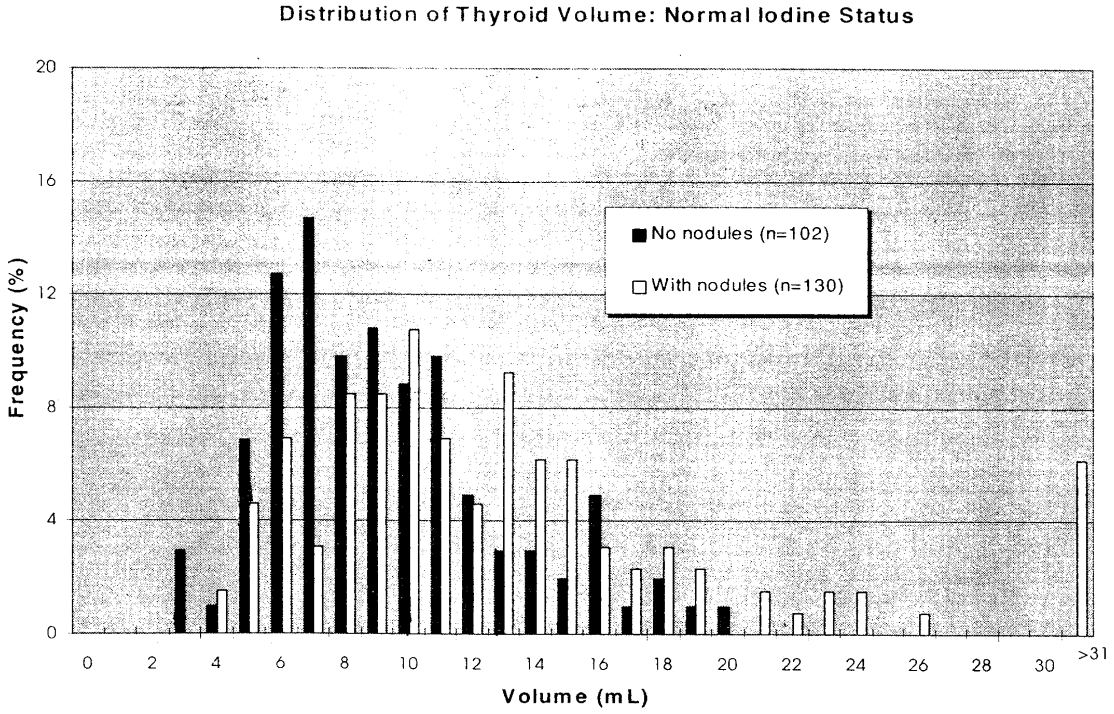

Figure 3. The Distribution of Thyroid Volumes among 232 Study Participants from Majuro with Normal Iodine Excretion
(Takahashi 1999)

4.0 DIETARY IODINE DEFICIENCIES AMONG MARSHALLESE

The unexpected thyroid neoplasms observed among teens in the early 1960s prompted the BNL medical team to conduct a comprehensive study of thyroid function tests in their 11- and 12-year medical survey (Conard et al. 1967). Included among the tests were (1) thyroid uptake of radioiodine-132, and (2) urinary excretion of stable/dietary iodine.

4.1 MEASUREMENTS OF RADIOIODINE UPTAKE

The uptake of I-132 by the thyroid was tested in 1965 on 21 Marshallese; 12 exposed and 9 **non-exposed** controls with no known thyroid abnormalities. The I-132 in amounts of 5–10 μCi was administrated by mouth before breakfast, and counts were obtained over the neck area with a collimated 1-inch NaI crystal at ½ , 1, 2, 3, and 4 hours. In addition, a single 3-hr urine sample was assessed for I-132.

For the 9 **non-exposed** control subjects, the average radioiodine uptake fraction was 0.52, with a range of 0.26 to 0.77. These data indicate that the majority of persons had a significantly higher value than the ICRP 56 f_2 value of 0.3 applied by Lessard et al. 1985.

4.2 1965 IODINE URINARY EXCRETION STUDIES

By means of the iodine recycle model described by Riggs (1952), analysis of a 24-hr urine sample for iodine content provides a reliable method for estimating the daily dietary intake. Twenty-four-hour urine samples from 28 subjects were analyzed at the Boston Medical Laboratory for iodine in 1965. Table 7 defines the subjects by age and sex, and cites the subjects by their iodine excretion value in ascending order. Thus, subject with the ID #59 was a 46-year old female whose 24-hour urine contained 19.5 μg iodine. Her excretion value corresponds to a daily intake of less than 35 μg iodine, with an associated uptake fraction of 0.796. All but 5 of the 28 subjects had f_2 values greater than the 0.3 value assumed in the ICRP-30 model. These data are further analyzed in Table 8, which shows that females had significantly lower dietary intakes and correspondingly higher f_2 values than males. Also noteworthy is that the mean and range of f_2 values derived from the 28 urine samples are virtually identical to the I-132 uptake values calculated for the 9 non-exposed/control subjects described above.

Table 7. Iodine Excretion Data for 28 Marshallese and Their Estimated Thyroid Uptake Factions

Subject ID No.	Age	Sex	Weight(kg)	I-Urine/d (µg/d)	Derived Iodine Intake (µg/day)	Derived I Uptake Fraction (f_2)
59	46	F	39	19.5	34.7	0.796
835	32	F	51	34.7	49.9	0.686
8	14	F	41	37.2	52.4	0.672
843	37	F	50	40.5	55.7	0.652
932	31	F	50	45.4	60.6	0.626
20	19	M	54	48.4	63.6	0.611
41	56	M	54	50.4	65.6	0.601
11	62	M	52	56.3	71.5	0.574
45	44	F	55	61.0	76.2	0.555
15	19	F	52	66.1	81.3	0.535
84	11	M	29	70.1	85.3	0.520
53	20	F	49	75.0	90.2	0.503
920	34	M	62	76.4	91.6	0.499
855	61	M	66	82.4	97.6	0.480
822	19	M	57	90.1	105.3	0.458
58	71	F	55	105.8	121.0	0.418
840	36	M	68	106.8	122.0	0.416
40	41	M	55	114.4	129.6	0.399
12	30	F	62	130.0	145.2	0.369
27	38	M	65	136.2	151.4	0.358
853	61	M	69	145.1	160.2	0.344
942	51	F	60	163.5	178.7	0.317
928	53	F	57	164.2	179.4	0.316
895	36	F	67	177.1	192.3	0.300
73	30	M	71	178.2	193.4	0.299
50	46	M	84	190.3	205.5	0.285
833	33	M	64	197.4	212.6	0.278
51	37	M	44	279.3	294.5	0.214

Table 8. Summary Data for Iodine Intake and Thyroid Uptake by Gender

Group Category	# of Persons	I-Dietary Intake (µg/d) Mean	I-Dietary Intake(µg/d) Range	I-Uptake Fraction(f_2) Mean	I-Uptake Fraction(f_2) Range
All Persons	28	113.8	34.7–279.3	0.467	0.214–0.796
All Females	13	101.3	34.7–177.1	0.519	0.300–0.796
All Males	15	136.7	63.6–294.5	0.422	0.214–0.611

In response to these data, BNL scientists in the 11- and 12-year BNL survey report (Conard et al. 1967) stated the following:

> *. . . In general, it had been expected that individuals living close to the sea and eating seafood and fish would show relatively higher iodine intake.*

1974 Dietary Intake and Excretion Studies. Apparently, the unexpected low excretion levels observed in the 11- and 12-year medical survey prompted additional studies as part of the 20-year medical survey of the exposed Rongelapese (Conard et al. 1975). During the 1974 survey, 24-hr urine samples were again analyzed for iodine, which showed daily excretions that ranged from 25 μg to 266 μg, with a mean value of 126.7 μg and a standard deviation ± 74.5 μg. Although the mean excretion value of 126.7 μCi/d had increased, the range of values observed in 1974 was comparable to that observed earlier in 1965. Due to the fact that the 1974 dataset did not identify the subjects by age or sex, a reasonable explanation may be that a disproportionate number of males were represented among the 19 subjects analyzed in 1974. (Note: Table 8 identifies a mean excretion of 136.7 μg/d among males in 1965.)

In concert with the 1974 iodine excretion study, BNL scientists also collected seven 24-hour sample diets that were analyzed for their iodine content. For the seven sample diets, an estimated mean intake of 84.7 + 36.6 μg per day was calculated. It should be noted that the highest intake of 152 μg/day corresponded to Sample Meal 1, which is the only diet containing a non-local food item (i.e., rice).

Exhibit #2 contains both the urine excretion data and the dietary intake data, as reported by the BNL in the 20-year medical survey report (Conard et al. 1974).

EXHIBIT #2

Appendix 9

A. Urinary Iodine Excretion

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

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

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

In summary, results of two independent urinalyses conducted in 1965 and 1974 show iodine excretion values that were well below expected values, as given by the following remarks (Conard et al. 1975, p. 58):

> *The **urinary excretion** is somewhat lower than the U.S. mean of 190 μg/day in **1941** [Reference 94]. On the basis of the few diets analyzed, the daily **intake** seems to be within the recommended range of 50 to 75 μg* [see Exhibit #2 above]. *These iodine are somewhat lower than would be expected in an oceanic population but are much higher than seen in areas of endemic goiter.*
> [Emphasis added.]

In spite of BNL's statements that expressed surprise at the observed low excretion values, there appears to be an attempt to downplay the importance of these findings by favorably comparing these values to those in "areas of endemic goiter," and further suggesting that a daily intake of 50 to 75 μg represents a recommended range. The U.S. Food and Drug Administration (FDA), the FNB, and the World Health Organization (WHO) recommend daily **intakes** of 90 μg for children, 150 μg for adults, 200 to 220 μg for pregnant females, and 290 μg for lactating females.

Equally of interest is the inappropriate comparison of BNL's 1974 data to the mean U.S. excretion of 190 μg/day for the year 1941, which references Bruger et al. 1941 ("The Iodine Content of Blood, Urine, and Saliva of Normal Persons in the New York City Area," *J Lab Clin Med* 26:1942–1944). From data previously presented in Section 5.0 of Volume I of this report, it will be recalled that at the time of the BNL's 1975 21-year medical survey, numerous studies had been published, including the comprehensive study by Oddie et al. (1970). Key findings in this study are captured in the following statements (Oddie et al. 1970, pp. 663–664):

> *Endemic goiter has been found, at one time or another, in several areas of continental North America . . . Inspection shows a good correlation between these goiter areas and the lower iodine intake areas in the **present** study. However, values for mean iodine intake in these areas are now in excess of 240 μg daily, usually ranging between 260 and 390 μg daily . . . Iodine intake is highest in the southwest, where values range up to 738 μg mean intake daily.*
>
> *. . . The iodization of table salt in the United States at a level of 1 part /10,000 has been estimated to provide an iodine intake of **500 μg** daily, assuming an average daily iodized salt intake of 6.5 g. The variation in mean intake observed in the present survey (from 240 to 740 μg daily) indicated that the original paucity of dietary iodine intake in the United States has indeed been corrected and that the desired average intake of 500 μg daily has been achieved in the southwest and in much of the southeastern United States.*
>
> *With this increase in intake, radioiodine uptake values in euthyroid subjects have been falling progressively; in many areas, the lower limit of normal 24-hour uptake now is between 5 and 10% and in the southwest it has recently fallen below 5%. **The marked geographic variations in iodine intake still evident in***

the United States indicate that normal standards for thyroidal radioiodine uptake must be locally determined. [Emphasis added.]

Recent Study Data Regarding Dietary Intakes of Iodine among Marshallese. Late in 1999, Takahashi et al. published a study that assessed the current dietary iodine levels in the Marshall Islands. Takahashi's findings validate the earlier data of Conard et al. (1967, 1975). Salient aspects of the study include the following:

- Analysis of urinary excretion of iodine was used to evaluate typical dietary intakes of individuals. Because it was not practical to obtain 24-hour urine samples, spot samples were acquired, and the iodine-to-creatinine ratio was determined and used as an index of the daily iodine excretion.

- Urine was collected from three population groups; 310 adults presently living in an "urban environment" on Majuro Atoll,; and 55 adults and 68 children living in an "outer environment" on Likiep Atoll.

Table 9 summarizes the iodine results of the study. For the Majuro study group of adults, only 25% had excretion levels considered normal, while 53%, 21%, and 1% were judged mildly deficient, moderately deficient, and severely iodine-deficient.

Among those examined on Likiep Atoll, iodine deficiency was even more pronounced; (1) of the adult participants, 13% showed **severe** iodine deficiency, with 47% exhibiting moderate and 33% mild deficiency (only 7% of the adults exhibited **normal** dietary levels), (2) for the 68 children of Likiep, iodine deficiencies were noted in all but 13%, with 24%, 56%, and 7% showing mild, moderate, and severe deficiencies, respectively.

Table 9. Categories of Urinary Iodine Excretion for 310 Majuro Adults and 55 Adults and 68 Children from an "Outer-Island" (Likiep Atoll) Based on WHO Guidelines

Iodine Excretion Category	Iodine:creatinine (nM/mM)	% Study Subjects (sample size)		
		Majuro Adults (n=310)	Likiep Adults (n = 55)	Likiep Children (n = 68)
Normal	>89	25	7	13
Mild deficiency	45–89	53	33	24
Moderate deficiency	22–45	21	47	56
Severe deficiency	<22	1	13	7

Takahashi's assessment of urinary excretion among present-day Marshall Islanders employed WHO (1994) criteria quantified in Table 10. WHO classifies the dietary intake values as (1) normal, (2) mild deficiency, (3) moderate deficiency, and (4) severe deficiency, and provides quantitative values that are defined in terms of the amount of **iodine** excreted per day, as given in Table 10.

Table 10. WHO Criteria for Assessing Dietary Iodine Deficiency

Classification	Urinary Output of Iodine (μg/l)
No deficiency	≥ 100
Mild deficiency	50–99
Moderate deficiency	20–49
Severe deficiency	< 20

The unexpected higher frequency of dietary iodine deficiencies among the Likiep participants did not go unnoticed to the investigators, who offered the following comments (Takahashi et al. 1999):

> *. . . This finding **contradicts** our expectations that the more **traditional** lifestyle on the outer atolls . . . where lifestyles are based on fishing and traditional food gathering practices would [be expected to] provide children with more iodine than the diets in the semi-industrialized urban areas of Majuro and Ebeye. This observation may suggest that the **traditional** lifestyle may cause iodine deficiency by itself and **that the Marshallese may have suffered from iodine deficiency for a long time.*** [Emphasis added.]

Suspicion that local diets may be low in iodine was confirmed by Takahashi and co-investigators as part of their study (Table 11). Analysis of local foods that represent the Marshallese diet showed that no vegetative foods or drinking water contained measurable levels of iodine, and fish was the only significant source of dietary iodine. Interviews with Marshallese further revealed that fish is **not** consumed as frequently or in quantity, as researchers had expected.

Table 11. Iodine Concentration in Selected Components of the Marshallese Diet
(Source: Takahashi et al. 1999)

Type of Food Product	Number of Samples	Mean Concentration (μg/g)
Coconut	6	<0.01
Jekaru (fermented coconut juice)	2	<0.01
Pandanus (fruit)	1	<0.01
Papaya	1	<0.01
Breadfruit	1	<0.01
Rice	2	<0.01
Drinking Water	1	<0.01
Fish	10	0.66

In summary, BNL medical surveys of the Rongelap group in 1965 and 1974 (Conard et al. 1967 and 1975) and the more recent study by Takahashi et al. (1999) have consistently shown low dietary intakes and excretions of stable iodine for a majority of Marshallese.

For unexplained reasons these highly relevant survey data were not acknowledged by BNL scientists in their approach to thyroid dose reconstruction, as reported by Lessard et al. (1985). Their use of the ICRP 30 dose model and its associated thyroid DCF(s) must, therefore, be regarded as inappropriate. Table 6 above indicated that of the 28 subjects assessed in 1965, 23 subjects (or 82%) demonstrated uptake fractions greater than the ICRP 30-assumed value of 0.3. Table 7 also suggests that the critical group among the three populations exposed on Rongelap, Ailinginae, and Utrik Atolls were females of child-bearing age. Of special concern within this group are females who were either pregnant or lactating at time of BRAVO, and for the years before thyroxine replacement therapy was initiated. For the most iodine-deficient individuals, use of the ICRP 30 model assumes an iodine uptake fraction that is low by more than a factor of 2.

5.0 DIETARY DEFICIENCIES OTHER THAN IODINE THAT MAY HAVE AFFECTED THYROID METABOLISM AMONG MARSHALLESE

In a recent report issued by the FNB (2000), the standing committee on the Scientific Evaluation of Dietary Reference Intakes reviewed the scientific literature and established intakes for various vitamins and minerals, including iodine. On page 268 of Chapter 8: *Iodine*, the FNB stated the following:

> *Deficiencies of **vitamin A**, **selenium**, or **iron** can each exacerbate the effects of **iodine deficiency**.* [Emphasis added.]

The functional role for many minerals is that of a cofactor to a specific enzyme that is uniquely linked to a metabolic pathway. This section presents data that strongly suggest that, in addition to iodine deficiency, Marshallese may have also been dietary-deficient in vitamin A, iron, and/or selenium.

5.1 MINERAL CONTENT OF ATOLL SOILS

Dietary minerals are generally obtained by consuming various foods that include food crops, which extract these minerals by root uptake from the soil. A diet that is principally derived from local food crops will, therefore, reflect deficiencies in the mineral content of local soils. It was previously shown that food crops grown in the Marshall Islands contain only nominal levels of iodine (Conard et al. 1975, Takahashi et al. 1999). Presented below are data that briefly identify other soil deficiencies.

Low atoll soils of the Pacific have been evaluated for their chemistry, mineralogy, and ability to support food crops in a number of studies. Stone et al. (2000) characterized the soils of the Marshall Islands and provided the following conclusions:

> *Atoll soils differ in several ways from almost all other soils that have been written about or discussed in agricultural literature . . . atoll soils are made almost entirely . . . of calcium carbonate, with small amounts of magnesium carbonate . . . soils of deep ocean atolls have no clay . . which is indeed important in continental soils . . .*
>
> *Green plants require several nutrients or chemical elements for growth and fruiting . . . the six plant nutrients most likely to be in short supply on atolls* [include nitrogen, N; phosphorous, P; potassium, K; **iron, Fe**; manganese, Mn, and zinc, Zn.]

Similar conclusions were stated by Morrison (1990):

> *The data here also clearly indicate the very low levels of certain **micronutrients** found in the soils of low atolls. **Iron**, manganese, copper, and zinc are all present*

*in **extremely low** (total) amounts such that the available supplies of these elements must be minimal in the absence of **external** supplies*. [Emphasis added.]

Early studies by Fosberg and Carroll (1965) included analyses of minor elements in soils in the northern Marshall Islands that included the atolls of Jemo, Bikar, Ujae, Kwajalein, and Wotho. A summary of their data pertaining to minor soil components are given in Table 12.

Table 12. Ranges in Amounts of Minor Elements in Samples of Soil Materials from the Jemo Soil Series on Bikar, Jemo, Ujae, Kwajalein, and Wotho Atolls of the Marshall Islands

For elements above double line, determinations were made on 45 samples; for those below lines, on 25 samples.

Element	Range (percent)	Frequency (No. of times found)
Boron	0.0X, 0.00X	23, 22
Barium	.0X, .00X, .00X	1, 28, 15
Cobalt	.000X	4
Chromium	.00X, .000X	17, 28
Copper	.0X, .00X, .000X	2, 5, 38
Iron	.X, .0X, .00X	2, 33, 10
Manganese	.0X, .00X, .000X	1, 23, 20
Molybdenum	.00X, .000X	1, 4
Nickel	.0X, .000X	1, 27
Strontium	.X, .0X	32, 13
Zinc	.0X, .00X, .000X	10, 5, 1
Aluminum	.0X, .00X	13, 9
Silicon	.X, .0X	1, 24
Silver	.000X	1
Titanium	.00X, .000X	5, 16
Vanadium	.00X, .000X	10, 14
Yttrium	.00X, .000X	2, 3

Similar findings were reported in a more recent study by Gessell and Walker (1992) in behalf of soils at Bikini and Enewetak Atoll. Table 13 provides summary data of composite soil samples representing the top 6 inches that are commonly regarded as the root-zone. For comparison, data are also included that represent average values for U.S. continental soils (Bowen 1979). With the exception of copper, atoll soils suffer from varying levels of mineral deficiencies for all other micronutrients. Most pronounced are soil deficiencies of iron and manganese. Important to note here is that while none of the atoll studies analyzed soil for **selenium**, it is only reasonable to conclude that atoll soils are also deficient in **selenium** content.

Table 13. Elemental Analysis of Bikini and Enewetak Soils
(Source: Gessell and Walker 1992)

Island		Dist. Class	Percent				ppm						
Composite Soils, Surface 15 cm			N %	P %	Ca %	Mg %	Na	K	Fe	Mn	Cu	Zn	Se
	Vegetation												
Ikuren	*Pisonia*	A	0.31	0.34	36.8	0.73	940	70	37.9	1.80	29.4	12.0	ND*
Japtan	*Pisonia*	A	0.84	4.38	31.9	0.96	1,040	260	92.2	8.40	10.3	18.1	ND
Enue	Coconut grove	A	0.42	0.33	36.2	0.80	1,030	90	138	2.00	29.3	10.4	ND
Bikini	*Pandanus*	B	0.60	0.95	31.7	2.29	1,010	100	73.5	2.90	44.9	12.7	ND
Nam	*Tournefortia*	C	0.10	0.04	35.2	2.08	1,090	80	35.0	0.80	21.7	20.0	ND
Avg. U.S. Continental Soils**			2	0.8	1.5	5.0	5,000	14,000	40,000	1,000	30	90	0.4

* ND = not done.
** Source: Bowen, H.J.M 1979

5.2 DOCUMENTED IRON DEFICIENCIES AMONG MARSHALLESE

The documented iron deficiency in atoll soils is consistent with medical survey studies that found low hematocrit levels in both the exposed and unexposed Marshallese subjects (Conard et al. 1956, 1958, 1959, 1960, 1962, 1963, 1965, 1967, 1970, and 1975). Table 14 identifies hematocrit values cited by the BNL in the 1957 medical survey (Conard et al. 1958). For comparison, average hematocrit values for adult U.S. men and women are 45% (38–54%) and 40% (36–47%), respectively.

Table 14. Hematocrit Values by Age and Sex for Exposed and Control Subjects in 1957

Age/Sex	Rongelap (%)	Ailinginae (%)	Utrik (%)	Unexposed Rongelap (%)
Males:				
• 3 – 10 yr	35.6 ± 2.4	37.5	37.0 ± 3.8	35.6 ± 2.4
• >15 yr	38.7 ± 3.2	40.6 ± 1.5	40.2 ± 2.4	41.0 ± 3.1
Females (all ages)	35.4 ± 2.6	36.5 ± 3.2	35.9 ± 2.9	35.9 ± 2.4

Anemia was also in evidence by low **total** blood volume (BV) and red cell volume (RCV) in non-exposed Marshallese. Table 15 compares summary data from 20 **non**-exposed Marshallese with 19 Caucasians residing in the Pacific Ocean area (Conard et al. 1967).

Table 15. Total Blood and Red Cell Volume for Non-Exposed Marshallese and Pacific Caucasians
(Source: Conard et al. 1967)

Group	Average Red Cell Volume (Liter)	Average Total Blood Volume (Liter)
Marshallese – Non-exposed (20)	1.303	3.102
Pacific Area Caucasians (19)	1.983	4.582

In response to these and other observations, BNL clinicians stated the following:

From the 1957 BNL Medical Survey:

> *The hematocrit values showed a tendency toward **anemia** in both the exposed and unexposed groups . . . There were 53% of the exposed and 46% of the unexposed people who had hematocrits of less than 37%.* [Emphasis added.]

And, from the 1975 Medical Survey:

> *Varying degrees of **anemia** have been seen occasionally, particularly in women of **childbearing age***. [Emphasis added.]

Studies by Zimmermann and Korhle (2002) have shown that iron deficiency impairs thyroid hormone synthesis by reducing the activity of the heme-dependent thyroid peroxidase (see Figure 3 of Vol. I of this report). Beard (1990) also showed a causal relationship between iron deficiency, anemia, and reduced levels of thyroxine in human subjects. Similar positive results were observed by Zimmermann et al. (2000), who treated goitrous/iron-deficient anemic children with 60 mg ferrous sulfate. The study concluded that iron supplementation significantly improves the efficacy of iodized oil in goitrous children with iron-deficiency anemia.

5.3 SELENIUM DEFICIENCY IN MARSHALLESE DIETS

Although none of the previously-cited soil studies specifically assessed for selenium, it is reasonable to conclude that the observed generic mineral deficiencies of atoll soils includes the mineral selenium. However, indirect evidence of selenium deficiency among Marshallese is presented in Section 7 of this report.

Selenium in trace quantities is essential to plants and mammals for normal growth and health. Dietary intakes of less than 6 μg/day are considered deficient, and intakes of 10 to 200 μg/day represent the normal range.

Like most minerals, selenium is obtained from the consumption of various food products, which in turn reflect the mineral content of soils. The selenium content of soils within the U.S. varies between 0.01 μg/g to 12 μg/g, with an average value of 0.4 μg/g of soil (Bowen 1979). Like most minerals found in soil, selenium is primarily associated with the inorganic clay component. Thus, for Marshallese on Rongelap and Utrik whose diet consisted principally of locally grown foods, a third dietary deficiency that affects thyroid function can be assumed.

The Metabolic Role of Selenium. Once released from the thyroid gland into the circulating blood, thyroxine (T_4 or tetraiodo-thyronine) and triiodothyronine (T_3) rapidly attach to several binding proteins, including thyroxine binding globulin (TBG) transthyretin, and albumin. The protein-bound hormone molecules attach to target cells throughout the body. Important to note is that T_3 has significantly greater hormonal potency than T_4, and it is at this time that a large fraction of T_4 is converted to T_3. The monodeiodination of T_4 to T_3 is the major source of peripheral T_3 and reverse T_3 (rT_3) (Figure 4). Selenium is an essential cofactor for deiodinase

enzymes (selanodeiodinases) that convert T_4 to T_3 (Berry and Larsen 1992; Larsen et al. 1998; Duffield et al. 1999).

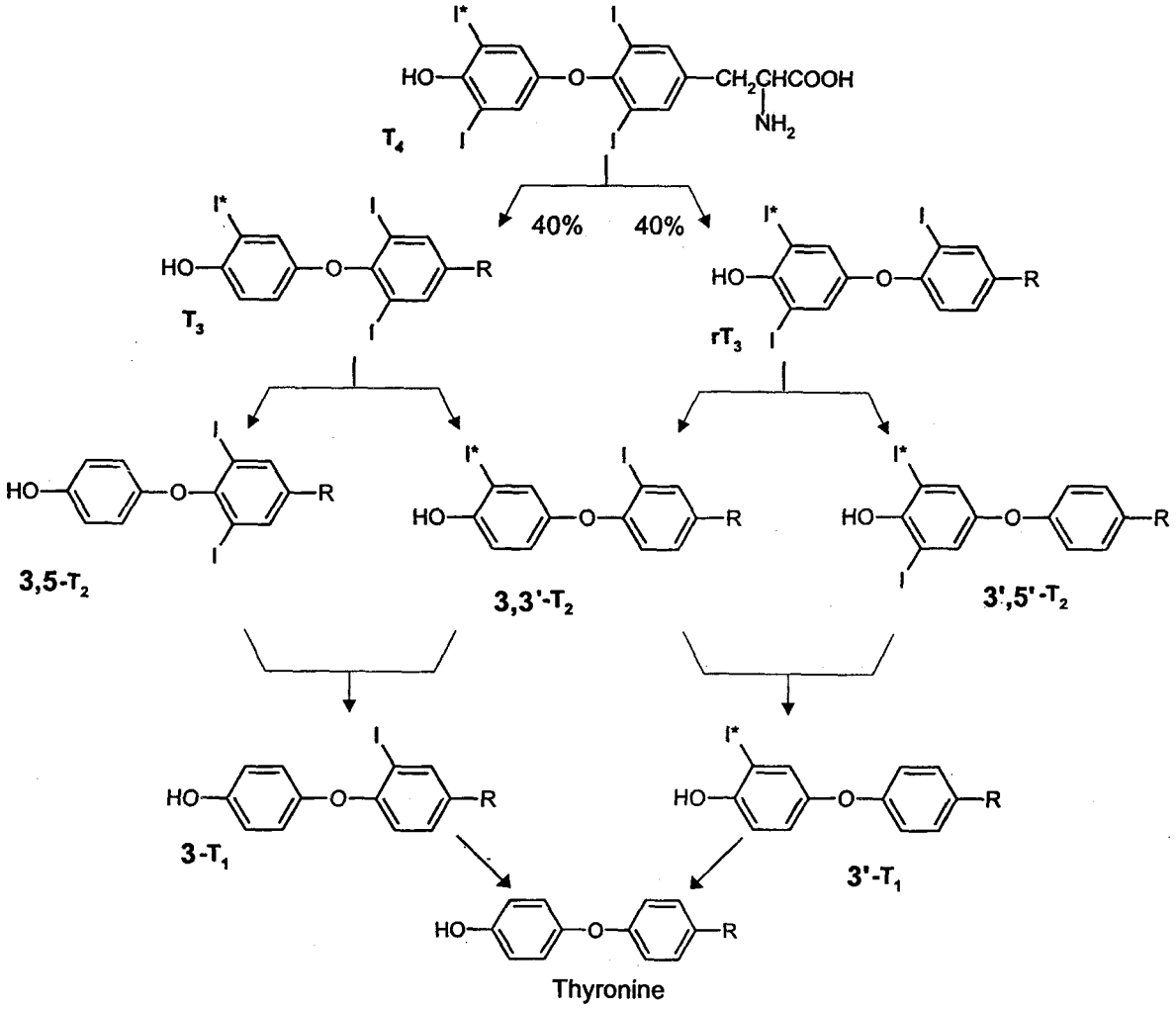

Figure 4. Major Deiodination Pathways of Thyroid Hormones in Peripheral Tissues

A dietary deficiency in selenium would, therefore, impair the conversion of T_4 to the more potent T_3 hormone and reduce the efficiency of the thyroid gland. A reduced production of T_3 also has another major impact on thyroid function. When peripheral T_3 levels are low, the pituitary responds by releasing thyroid stimulating hormone (TSH), which is the major regulatory/ stimulator of the thyroid. Thus, a dietary deficiency in selenium can have the same effect as a dietary deficiency in iodide, inasmuch as either can limit T_3 production, which raises TSH levels. In turn, an elevation in TSH raises the uptake fraction (or f_2) of iodide and indicates a state of biochemical hypothyroidism. Since the metabolic role of iodine and selenium are independent of each other, a deficiency in both is likely to have an additive effect.

5.4 DOCUMENTED VITAMIN A DEFICIENCY

In the 4-year BNL medical survey report (Conard et al. 1959), as well as in the 6-year BNL medical survey report (Conard et al. 1960), the following brief statements were made that suggested dietary deficiencies in vitamin A among the exposed population:

Four-year Survey, p. 8:

> *. . . A complaint of night blindness of several months duration among 10 children and 1 adult was investigated and is reported below.*

Six-year Survey, p. 18:

> *. . . At the times of 1959 and 1960 surveys of the Marshallese, the people appeared to be generally in a state of good health and nutrition. There was* ***no*** *indication of vitamin deficiency such as had been observed in 1957 in the children when about 10 had night blindness associated with vitamin A deficiency. The improvement may be due in part to the agricultural program on the island resulting in the availability of papaya and squash.*

The best known and generally the earliest physiologic effect of vitamin A deficiency (i.e., hypovitaminosis A) is the loss of night vision (i.e., nyctalopia). The retinal rods responsible for night vision contain a pigment (i.e., rhodopsin) derived from vitamin A that breaks down when light strikes the rod. In vitamin A deficiency, rhodopsin regeneration fails (Scrimshaw 1979.)

Beside night vision, there are numerous other metabolic functions that require vitamin A, including those of the thyroid. Vitamin A is essential for thyroid function by aiding the thyroid's ability to absorb iodine, as well as enhancing the conversion of T_4 to T_3 (Morley et al. 1980). In a double-blind, randomized clinical study of children with iodine deficiency disorders (IDD) and vitamin A deficiency (VAD), children were treated with either iodized salt and 200,000 international units of vitamin A or iodized salt alone. In children with IDD and VAD, the intervention with both iodized salt and vitamin A resulted in significant reductions in TSH levels and thyroid mass when compared to children receiving only the iodized salt (Zimmermann et al. 2000).

5.5 CONCLUSIONS

Minerals critical to normal thyroid function include iodine, selenium, and iron, along with vitamin A. Clinical data provide evidence that varying dietary deficiencies existed in the general Marshallese population, which reflect the very low mineral content of sandy atoll soils.

Individually or in combination, these dietary deficiencies lead to a state of **biochemical hypothyroidism** that is clinically characterized by elevated serum levels of TSH and an increased thyroid uptake fraction (f_2) of iodine/radioiodine.

Important to note is that biochemical hypothyroidism (as opposed to primary hypothyroidism) does not lead to overt thyroid insufficiency and goitrous conditions. As a compensatory mechanism, biochemical hypothyroidism does, however, reduce the thyroid's reserve capacity. Under conditions of reduced thyroid reserve capacity, any additional insult and/or demand on the thyroid may have adverse effects.

For persons exposed to BRAVO fallout, the added insult of high radiation doses to the thyroid and the added demands of pregnancy on the maternal thyroid may have resulted in adverse pregnancy outcomes and serological findings effects that have been misinterpreted by previous investigators.

Presented in Sections 6.0 and 7.0 of this report are post-BRAVO data pertaining to adverse pregnancy outcomes and serological findings that have puzzled BNL scientists, but may very likely represent states of thyroid insufficiencies caused by dietary factors and/or thyroid exposure to radiation.

6.0 REASSESSMENT OF ADVERSE PREGNANCY OUTCOMES

Among the populations exposed to BRAVO fallout, hypothyroidism was diagnosed at different levels. **Clinically overt** (or primary) hypothyroidism was diagnosed in two Rongelap boys with severe growth retardation (cretinism). Hypothyroidism was also observed in twelve other subjects diagnosed with thyroid nodules and growth retardation.

More moderate forms of thyroid hypofunction can only be diagnosed by serological methods that assess levels of T_4 and TSH. Typically, **biochemical hypothyroidism** is characterized by low to normal levels of T_4, but elevated levels of TSH. Under this condition, borderline thyroxine levels may preclude overt expressions of hypothyroidism, but only under elevated stimulation of TSH. Under conditions of biochemical hypothyroidism, the thyroid's reserve capacity may approach zero, and any additional demand on the thyroid can no longer be met.

At sufficiently large doses of radiation, damage to individual cells results in cell death. Death to a substantial fraction of thyroid cells, in turn, will lead to hypothyroidism. The extent of hypothyroidism may range from very mild or subclinical to severe, and reflects the thyroid's reserve capacity, the magnitude of radiation dose, and the fraction of thyroid cells destroyed.

6.1 THE DEMANDS OF PREGNANCY ON THYROID FUNCTION

The state of pregnancy is accompanied by complex hormonal changes in the mother, the placenta, and the fetus during the 9-month gestation period. A significant number of these involve the interaction of the maternal thyroid with the developing embryo/fetus. Numerous studies have documented these changes in euthyroid and in hypothyroid pregnant subjects. Due to the complexity of this subject, only a brief discussion is presented herein.

During the first trimester, there is a dramatic increase in estrogen levels, largely derived from placental production. Elevated levels of estrogen stimulate the synthesis of thyroid-binding globulin (TBG) in the liver that raises circulating TBG levels to twice normal. As a consequence, total T_4 and T_3 levels increase and reach peak levels at 10 to 15 weeks of gestation, and remain at this level for the duration of pregnancy. The increase in total (and free) T_4 and T_3 are driven not only by the stimulating effects of TSH on the maternal thyroid, but more importantly by human chorionic gonadotropin (HCG), which rises rapidly in the first few weeks following conception. The stimulating effect of HCG on the maternal thyroid is due to the fact that HCG is structurally similar to TSH. Like TSH, the HCG hormone is composed of two polypeptide chains; an alpha chain and a beta chain. Significantly, the alpha chain is identical to that of TSH. This partial structural homology allows HCG to act as a surrogate to TSH in stimulating the thyroid, as evidenced by a decrease in TSH levels in the first trimester when HCG levels peak (see Figure 5) (Glinoer 1997).

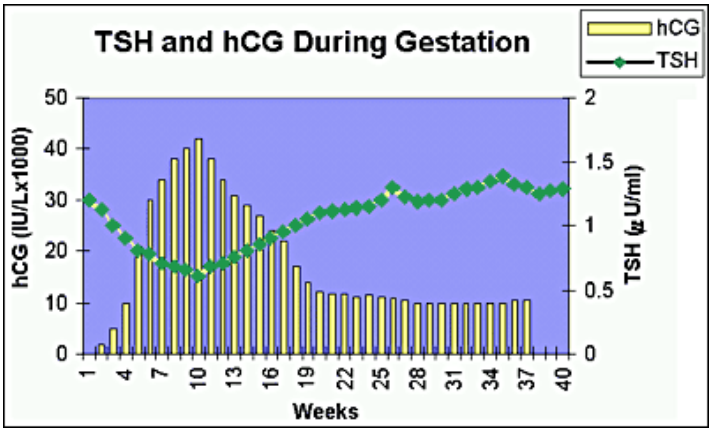

Figure 5. Thyroid-Stimulating Hormone and Human Chorionic Gonadotropin during Gestation

Lastly, the increased demands on the maternal thyroid during pregnancy are affected by **increased** renal excretion of circulating iodide caused by the physiological increase in maternal glomerular filtration. (Morreale de Escobar et al. 1987).

In summary, increased demands on maternal thyroid function during pregnancy are modulated by three independent but interrelated factors; (1) an increase in HCG concentrations that stimulate the thyroid gland, (2) increased production of thyroxine-binding globlulin (TBG), T_4, and T_3 induced by estrogen, and (3) an elevated increase in urinary iodide excretion, which reduces the available pool of iodide for thyroid uptake.

Maternal hypothyroidism during early pregnancy and its impacts on pregnancy outcomes are well documented in the scientific literature (Hetzel 1979, Hetzel 1983). Over the years, the WHO has focused its attention on IDD on a global level and defined these disorders by life-stage (WHO 1999). Table 16 identifies IDD impacts of maternal hypothyroidism on the fetus.

Table 16. The Iodine Deficiency Disorders
(WHO 1999)

Effects on the fetus	Abortions Stillbirths Congenital Anomalies Neurological Cretinism Psychomotor defects
Effects on the child and adolescent	Goiter Juvenile hypothyroidism Impaired metal function Retarded mental and physical development Diminished school performance
Effects on the adult	Goiter and its complications Hypothyroidism Impaired mental function

According to the American Association of Clinical Endocrinologists, nearly 1 out of 50 women in the United States is diagnosed with hypothyroidism during pregnancy, and nearly 6 out of every 100 miscarriages can be attributed to thyroid deficiency (Perry 2004).

A study of 244 pregnant and hypothyroid women showed a rate of stillbirths that was double that of control subjects (Niswander 1972, deZegher et al. 1995).

Perhaps the most frequent impact of maternal hypothyroidism is on psychoneurological and developmental deficits in the progeny. Maternal thyroid hormone is critical in the myelination of the central nervous system during the initial phases of gross brain development and during the spurt in forebrain neuroblast proliferation (Boyages et al 1993, Larsen 1989, Man 1991, Haddow et al. 1999). Substantial reductions in IQ scores have been reported among children born to hypothyroid mothers (Bleichrodt and Born 1994, Haddow et al. 1999). Together, these studies imply that maternal thyroid insufficiency adversely affects neuronal differentiation and fetal brain development.

Under extreme conditions of maternal dietary deficiency, congenital hypothyroidism in the offspring may result in cretinism (Goyens et al. 1987, Vanderpas et al. 1990). Endemic cretinism induced by iodide deficiency in-utero refers to individuals having a typical constellation of signs and symptoms, which at birth include increased hair, low forehead, puffy features, umbilical hernia, enlarged tongue, and sluggish behavior. Other abnormalities may include deaf-mutism, mental retardation, and evidence of spastic paraplegia. If, after birth, a state of hypothyroidism is allowed to continue, stunted body and skeletal growth and mental retardation result.

It must also be noted that congenital hypothyroidism and cretinism may result when normal/ euthyroid mothers are exposed to large doses of radioiodine during pregnancy. Under these conditions, the transfer of sufficient radioiodine across the placenta after the first trimester (when fetal concentration of iodine begins) can result in the complete destruction of the fetal thyroid. Inadvertent administration of high doses of I-131 in women for treatment of thyrotoxicosis or thyroid cancer has occurred in limited numbers when their pregnancy was unknown. In most such instances, exposure occurred early in the first trimester with limited consequences. Several cases, however, have been reported where the radioiodide was administered at the end of the first or beginning of the second trimester (Lightner 1977, VanHerle 1975, Hamill 1961, Fisher 1963, Green 1971, Stoffer 1976). Considerable destruction of fetal thyroid resulting in infant hypothyroidism and other developmental abnormalities was observed in several cases. The relatively low frequency of permanent damage to the fetus is likely related to the fact that fetal uptake is very low until mid-gestation, and inadvertent administration of I-131 at such an advanced stage of gestation is infrequent.

The FNB, a unit of the Institute of Medicine (IOM) of the National Academies, is the parent committee that develops recommendations for daily intakes of nutrients. The Board is responsible for overseeing the development of the Recommended Dietary Allowances (RDAs) and Estimated Average Requirements (EARs). In its recent report (FNB 2000), the Board cited the following iodine EAR and RDA values during pregnancy:

Age of Pregnant Female (yr)	EAR	RDA
14 – 18	160	220
19 – 30	160	220
31 – 50	160	220

The FNB defines these criteria as follows; (1) the EAR is the average daily intake of iodine needed to meet the nutritional requirements of **half** of the **healthy euthyroid** pregnant females, and (2) the RDA is the average daily dietary intake needed to meet the nutritional requirement of 97% to 98% of **healthy euthyroid** pregnant females.

As previously discussed, medical survey data taken in the spring of 1965 (Conard et al. 1965) had shown urine excretion values and dietary intakes of iodine (see Tables 7 and 8 and Exhibit #2) that were well below FNB's EAR/RDA values. In the fall of 1965, all Rongelap-exposed persons were started on thyroid hormone supplementation/replacement therapy. Thus, for the post-BRAVO period of 1954 through 1965, exposed females of child-bearing age may have experienced pregnancies under conditions of hypothyroidism induced by radiation injury to the thyroid that was exacerbated by dietary deficiencies in iodine, iron, selenium, and/or vitamin A.

6.2 PREGNANCY OUTCOMES DOCUMENTED BY BROOKHAVEN NATIONAL LABORATORY MEDICAL SURVEYS AMONG RONGELAPESE

Since the time of BRAVO, medical surveys were conducted at discrete times. Earlier surveys were confined to the more heavily exposed Rongelapese subjects who were exposed on Rongelap Island/Ailinginae Atoll. Moreover, early surveys were limited in scope. Thus, in the 2-year post-BRAVO survey report, no mention was made regarding adverse pregnancy outcomes (Conard et al. 1956).

In the 3-year survey report (Conard et al. 1958), the following clinical findings were acknowledged:

> *In utero effects and effects on pregnancy. Four women were pregnant at the time of exposure, two in the first, one in the second, and one in the third trimester. These pregnancies progressed uneventfully to normal-term deliveries. The babies all appeared normal in every way, and no microencephaly was present as has been reported in some babies irradiated in utero in Japan. Since the event, nine other normal births have taken place. One miscarriage and two infant deaths have occurred, but this incidence does not seem greater than that in unexposed Marshallese people based on limited numbers of observations.*
>
> *Fertility. It is entirely possible that a temporary loss of fertility may have occurred shortly after exposure in some of the people. However, careful investigation of the possible effects of the radiation exposure on fertility has not been possible. . . . Pregnancies among the exposed Rongelap women during the past three years are within the range 18 to 32 per 1000 population reported in the above survey.*

In the 4-year medical survey report (Conard et al. 1959), the following clinical data were reported:

> *. . . The exposed and unexposed [i.e., Rongelap Controls] groups each contain 19 women of childbearing age (15 to 44 years) . . .*
>
> *During the past year healthy babies were born to 4 irradiated women and 6 unexposed women. . . . Three miscarriages occurred in the exposed women, two at 3 months and one at about 6 months. In all three cases, this was the second miscarriage since exposure. However, two of the women have had one normal pregnancy since the accident. One of the unexposed women had a miscarriage, and another had a full-term baby that died within a month, apparently of diarrhea in infancy. (Between the March survey and the return survey in May 1958, one exposed woman had a full-term baby that died shortly after birth of unknown cause.)*

Years later, in the 11- and 12-year medical survey, Conard et al. (1973) provided the following summary statement:

> *As had been noted earlier, the exposed women had a somewhat **greater incidence** of miscarriages and stillbirths over the first 4 years post exposure (see Table 8). During 1954–58 the **exposed women** had 8 miscarriages in 49 pregnancies (16.3% incidence) during the 4-year period 1956–60. A χ^2 test for significance showed that total miscarriages and still-births were **significantly greater** (at the 5% level) in the exposed women compared with unexposed during the first 4 years, but there was no significant difference after this period.*
> [Emphasis added.]

Table 8 as referenced in the quotation above is reproduced herein as Table 17 for the years 1954 up to 1965, when thyroid hormone replacement began for all exposed Rongelapese. The data not only support a higher incidence rate, as stated by Conard et al. (1959), but Conard's assessment significantly underestimated the true incident rate among "exposed **women,**" due to the fact that Conard et al. (1959) defined "**exposed"** ". . . to include **non**exposed females married to **exposed males**." Thus, the contribution to adverse pregnancy outcomes by exposed females is obscured. This issue was subsequently resolved by data presented in the 20-year medical survey report (Conard et al. 1975).

Table 17. Births and Fetal Deaths by Year in Exposed* and Unexposed for 1954 through 1964

Year	No. Women Aged 15–45		Total Pregnancies		Live Births		Miscarriages		% Pregnancies Terminated in Miscarriage	
	Exposed*	Control	Exposed*	Control	Exposed*	Control	Exposed*	Control	Exposed*	Control
1954	19	NA	1	NA	0	NA	1	NA	100	NA
1955	20	NA	6	NA	5	NA	1	NA	17	NA
1956	20	29	6	9	4	7	2	2	33	22
1957	21	30	5	11	2	9	3	2	60	18
1958	22	30	14	9	8	8	6	1	43	11
1959	22	29	6	10	5	9	1	1	17	10
1960	24	29	10	10	9	8	1	2	10	20
1961	23	29	7	10	6	10	1	0	0	0
1962	24	30	4	6	4	5	1	1	25	17
1963	27	32	8	6	7	5	1	1	12	17
1964	26	32	6	13	6	11	0	2	0	15

* "Exposed" as defined by BNL includes **non**-exposed females who had children by exposed fathers.

For the 20-year report, Conard et al. (1975) segregated the **exposed** from the **unexposed** females for the 5-year period between 1954 and 1958, and reported the following:

Group	Total No. of Pregnancies	No. of Pregnancy Terminations	% Pregnancy Terminations
Exposed females and unexposed males	11	6	54.5
Unexposed females and unexposed males	39	5	12.8

Nevertheless, Conard (in 1992) and Cronkite et al. (again in 1997) largely dismissed earlier observations of adverse pregnancy outcomes and failed to recognize the possible link to maternal thyroid insufficiency as the causal factor:

In Conard 1992, p. 22; Cronkite et al. 1997, p. 183:

> *During the first years, there was an increase in miscarriages and stillbirths in the exposed Rongelap women, but the numbers were small and* ***it is uncertain if this increase was related to radiation effects.***
>
> *. . . Although inheritance of radiation-induced genetic mutations has been seen in animal studies, such effects have not been demonstrated unequivocally in humans. Studies on large numbers of children born of exposed Japanese parents of Hiroshima and Nagasaki, many of whom received larger amounts of radiation than the Marshallese, have not demonstrated any clear-cut genetic effects.*
> [Emphasis added.]

In spite of all the data that had been collected by BNL scientists that included thyroid dose estimates in hundreds to more than 1,000 rads, clinical evidence of thyroid pathologies/ hypothyroidism, and dietary deficiencies in iodine (that was likely further exacerbated by deficiencies in iron, selenium, and vitamin A), **the role of impaired thyroid function among pregnant females was never considered by BNL scientists as a risk factor that might explain the observed adverse pregnancy outcomes among the exposed population groups**.

Enclosed as Exhibit #3 is a case study that was cited in the 13-, 14- and 15-year medical survey study (Conard et al. 1970). Exhibit #3 identifies a 29-year old female exposed on Ailinginae Atoll to BRAVO fallout as a 15-year old. At the time of the medical survey in 1968, the subject was diagnosed with a thyroid tumor. Her medical history cites one normal pregnancy, two stillbirths, and one miscarriage. Important to note was the subject's anemic condition; clinical tests on thyroid function are obscured by the fact that the subject was on hormone replacement therapy at time of tumor diagnosis.

EXHIBIT #3

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

DISCHARGE SUMMARY

NAME	UNIT NO.
Rongelap 70	8-18-8[illegible] 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. } thyroid tumor

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 (Alimgnae). 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. (Note: these dose estimates have been significantly increased

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. } miscarriages & stillbirths } anemia

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

DISCHARGE SUMMARY

NAME | UNIT NO. 8-18-8[illegible] R

PAVILION 1 OPD

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.

thyroid hormone replacement therapy

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.

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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 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

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Exhibit #3 (Continued)

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

NAME | UNIT NO. 8-18-88 R

PAVILION 1 OPD

-3-

Marshall Islands.

FINAL DIAGNOSIS: Neurofibroma.

DISCHARGE MEDICATIONS: No medication necessary.

Robert A. Conard

Robert A. Conard, M.D.

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

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7.0 RE-ASSESSMENT OF ELEVATED PROTEIN-BOUND IODINE OBSERVED AMONG EXPOSED AND UNEXPOSED MARSHALLESE

In the past, a common approach for measuring the concentration of circulating thyroid hormones (T_4 and T_3) in the plasma or serum was to measure the collective iodine content of protein-bound iodine (PBI). A summary review of clinical data for PBI in euthyroid subjects was reported by Richmond (1962) and Wayne et al. (1964) that brackets the time of BRAVO exposure and early medical surveys conducted by BNL. Table 18 provides summary data regarding circulating levels of PBI as reported in 19 separate studies.

Table 18. Values for Protein-Bound Iodine in Euthyroid Subjects
(Source: Richmond 1962)

Author of Study	PBI (μg/100 ml serum)		
	No. of Cases	Mean	Range
Riggs 1947	55	5.1	3.5–7.5
Perry and Cosgrove 1949	34	5.9	4.0–9.3
Kydd et al. 1950	83	5.3	3.8–8.6
Starr et al. 1950	100	5.5	4.0–8.5
Barker et al. 1951	68	5.1	3.4–8.0
Tucker and Keys 1951	402	5.8	2.6–11.1
Hallman et al. 1951	37	5.4	3.2–7.6
Sunderman and Sunderman 1953	65	5.0	2.9–7.9
Zak et al. 1952	120	7.3	3.5–11.3
O'Neal and Simms 1953	10	5.2	–
Winikoff 1954	106	5.1	2.7–8.0
Zieve et al. 1954	50	6.7	4.6–9.3
Blackburn and Power 1955	530	5.2	2.5–8.3
Sanz et al. 1956	12	5.2	3.8–6.0
Astwood 1957	117	–	4.0–8.0
Levy 1959	49	–	3.5–7.4
Vanotti and Beruad 1959	30	5.1	4.2–6.1
Tanaka & Starr 1959	103	–	5.0–7.0
Wayne et al. 1964	130	4.9	3.0–7.5

PBI measurements, however, do **not** exclusively represent iodine contained in T_4 and T_3, but also include other circulating iodine compounds, such as mono-iodotyrosine (MIT), diiodotyrosine (DIT), and thyroglobulin, as well as free iodide. Thus, PBI values in normal healthy euthyroid subjects yield an iodine level of which about 10% of the total PBI (or about 0.6 μg I per 100 ml) is **not** associated with the hormones T_4 and T_3 (Wayne et al. 1964).

In 1957, Man and Bondy developed a method that selectively extracted iodine from the thyroid hormones. Their method, the **butanol extractable iodine (BEI)**, is based on the fact that only

iodine contained in T_4 and T_3 is isolated, whereas iodine and other circulating iodinated compounds are not. In effect, the butanol extractable iodine is a true measure of TBG.

As noted in Table 18, PBI levels show significant variations among seemingly euthyroid subjects, which prompted Wayne et al. (1964) to conclude the following:

> *. . . The PBI concentration does not show a linear relationship with the amount of thyroid hormone produced per day . . .* [and] *if the PBI is composed largely of biologically inactive compounds a normal or even a high level may coexist with* ***clinical hypothyroidism.*** [Emphasis added.]

At the time, the identity and role of circulating selenodeiodinases in the conversion of T_4 to T_3 and catabolism of T_3, DIT, and MIT had not been elucidated. Nevertheless, Wayne et al. (1964) correctly speculated that " . . . dietary factors influencing PBI levels are suggested by the finding of increased values. . ."

PBI Levels Reported for Marshallese. As part of the 11- and 12-year medical survey, Conard et al. (1967) reported the following data that included multiple serum PBI measurements and a single serum **butanol-extracted iodine** measurement. These data are reproduced herein as Table 19.

Table 19. Protein-Bound Iodine and Thyroxine Binding Globulin Values Reported by Brookhaven National Laboratory
(Conard et al. 1967)

Year	Group	Serum Protein-Bound Iodine		No. of Samples	Percent over 8 µg/100 ml
		Average (µg/100 ml)	Range (µg/100 ml)		
1959	Rongelap	6.2	4.1–9.2	12	16
1962	Rongelap	8.6	4.6–12.0	14	64
1963	Rongelap	8.1	1.9–12.0	29	66
1964	Rongelap	7.1	2.0–10.2	11	
1965	Rongelap (exposed)	7.6	4.1–11.9	31	36
1965	Rongelap (unexposed)	7.0	3.9–10.7	19	42
1966	Rongelap (exposed)	6.1	3.1–11.8	19	28
1966	Utrik (exposed)	11.8	6.9–28.7	25	92
1964	U.S. Medical Team	4.9	2.5–6.9	10	—
		Thyroxine-Bound Globulin/BEI			
1959	Rongelap	4.9	2.7–8.7	12	

BNL clinicians interpreted these unusually high PBI levels with the following explanation (Conard et al. 1967, pp. 28–29):

> *. . . Until the recent development of hypothyroidism in two boys, it had been the consensus of all physicians who examined these people that they were euthyroid. A conceivable explanation for the high PBI could be an elevation of thyroxine-*

binding proteins in serum which, as in the congenital elevation of thyroxine-binding globulin described by Beierwaltes and Robbins, causes an increase in the serum PBI without hyperthyroidism. The levels of the TBG in the Marshallese serum measured by Robbins, however, were within normal limits. The discrepancy between PBI and BEI suggested the presence of an iodoprotein in serum.

. . . there are some results which show that most of the iodinated amino acids in this protein are ***monoiodotyrosine*** *and* ***diiodotyrosine. The iodoamino acids are devoid of physiological activity.*** *. . .* ***The reason these individuals have such an iodoprotein in the blood is not clear.*** [Emphasis added.]

At the time of the BNL studies, the metabolic conversion of circulating T_4 to T_3 was not fully understood. It was the classical study by Oppenheimer et al. (1972) that identified the critical role of Type I iodothyroinine deiodinase, which is required for this conversion.

About 30% of T_4 is converted to T_3 by monodeiodonation in peripheral tissues. In fact, T_4 is generally regarded as a pro-hormone, and T_3 the primary tissue hormone, which is less firmly bound to plasma proteins and enters the cells more readily (DeGroot et al. 1958). While a small (~ 0.2) fraction of T_4 and T_3 is excreted in feces via the liver, bile, and gut, sequential **deiodination** and metabolic breakdown within cells and in the plasma liberate iodide, where it is free to follow the same fate as newly absorbed dietary iodide.

Therefore, deiodination of T_4 serves not only as a critical mechanism for the production of extra-thyroidal T_3, but also for the deactivation by catabolic breakdown of circulating DIT and MIT that contribute to the recirculating pool of iodide. These deiodination reactions, as previously shown in Figure 3 of Volume I of this report, are catalyzed by three different **selenium-dependent** deiodinases enzymes (selenodiodinases), as described by Larsen and Berry (1994) and Larsen et al. (1998).

Based on our current understanding of the catabolic deiodination processes mediated by selenium-dependent enzymes (selenodiodinases), it is probable that the observed high levels of PBI in the general Marshallese population are largely the result of circulating DIT and MIT, which were **not** subject to the normal catabolic deiodination process due to a deficiency in **selenium** among Marshallese.

The FNB of the National Academy of Sciences – National Research Council recommends a daily selenium intake of 20 to 30 μg for children, 55 μg for adult females, and 70 μg for adult males. The suspected dietary deficiency in selenium is consistent with the known dietary deficiencies of iodine, iron, and vitamin A, and reflects the mineral deficiencies in atoll soils.

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APPENDIX II-A: PAYNE HARRIS 1954 REPORT

58854

A SUMMARY OF THE RESULTS OF URINE ANALYSES ON RONGELAP NATIVES, AMERICANS AND JAPANESE FISHERMEN TO DATE

The urine samples used for the studies were pooled samples collected from a cross-section of the Rongelap native population, from a cross-section of the American group on Rongerik atoll, and supposedly from a cross section of the Japanese fishermen exposed on 1 March. Certain parameters important to the analyses are contained in the following table.

	Sample Source	Date of Collection	Average Volume Excreted Per Day Per Person
1.	Rongelap natives	March 16 and March 17	450 ml (measured average at time of collection)
2.	Rongerik Americans	March 18	1 liter
3.	Japanese fishermen	March 28 and April 19	Assumed to be 1 liter

The analyses can be broken down into three parts based on the different measured activities. Studies were made on alpha activity, gamma activity beta activity. The measurement of alpha activity was concerned with the chemical determination of plutonium. The studies of gamma activity were primarily concerned with identification and measurement of I^{131} and with identification by spectrum determination of certain other gamma emitting isotopes. The beta activity studies were concerned with the chemical separation and identification of Sr^{89}, Ba^{140}, Ca^{45}, and Ru^{103}.

The results of the plutonium analyses are shown below. The samples were run using different aliquots in an attempt to get meaningful results.

Sample Source	d/m/24 hr sample average from all samples measured
Americans	0.7
	0.7

These results have questionable significance. It is the practice at this laboratory to ignore levels below 1 dpm as repeated sampling in such cases have indicated no significance can be attached tothe results. However, if the above is considered an absolute upper limit of activity and considering that for humans 0.03% of the total body load is excreted on the day of collection, a calculation of the body load can be made: The results indicate a body burden of 1.6×10^{-2} µg which is less than 3% of the maximum permissible level.

The studies of gamma activity in the urine essentially turned out to be a study of I^{131} activity. By the use of a liquid scintillator system it was possible to determine gamma activity of whole urine and various fractions thereof. It was found that the total gamma activity could be divided into a volatile fraction with 70% of the activity and a non-volatile fraction with 30%. However, the whole urine decayed over a period of at least two weeks with a half-time of 8 day. corresponding to that of I^{131}. It was possible to collect the volatile activity by distillation into $Na_2S_2O_3$ after repeated acidification and addition of iodide carrier. The distillates were then measured for gamma activity and the results corrected to time of exposure.

The parameters necessary for the various corrections and the numbers used are shown in the following:

(a). Radioactive half-life - 8 days

(b) Per cent excreted on day of collection - 0.1%.

(c) Counter efficiency - 39% (checked with known sample of I^{131}).

(d) Per cent uptake on day of exposure - 100% (assumes ingestion as primary mode of exposure and complete separation of I^{131} from CuO fallout material).

Sample Source	counts/sec per liter day of counting	I^{131} in body on day of exposure
Rongelap natives	145	2.1×10^6 dps 56 μc
Rongerik Americans	20	6.4×10^5 dps 17.5 μc
Japanese	background	unknown

The above results do not take into account body burdens of short-lived iodine isotopes nor the iodine produced from various tellurium mothers. If the data from Hunter and Ballou on the various isotopes of iodine and tellurium are considered (and weighted for beta energies), the body levels in terms of I^{131} equivalents and dose to the thyroid may be estimated. Also, considering 100% uptake an exposure in terms of fissions can be determined.

Population Group	Body Burden of Iodine in I^{131} Equivalents	Dose to Thyroid	Exposure in fissions
Rongelap natives	5.1 mc	150 rep	5.5×10^{13}
Rongerik Americans	1.9 mc	50 rep	1.8×10^{13}

The thyroid dose was calculated assuming 20% maximum uptake in 24 hours and considering the more rapid decay introduced by the short half-lived isotopes.

The beta activities of a number of isotopes were studied. A repeat iodine analysis for the detection of beta activity was made on the native urine. Recovery was poor (~50%) in the separation method used. However, the final result indicating a body burden of 40 μc is to be compared with the 56 μc determined by gamma activity. The reliability is certainly less for beta measurement.

Up to the present time the total beta activity of the samples may be separated into two fractions on the basis of decay. The first portion of the decay

Past April 15 the half-life is 54 days and is apparently controlled by Sr^{89} contamination. Apparently other isotope contributions are small if having comparable half-lives or if having half-lives long compared to the two above.

The data may be summarized and put in tabular form as shown below.

Sample Source	Isotope	Disintegrations per min/liter corrected to day of exposure	Body burden at time of exposure	Fissions exposure
Rongelap natives	Sr^{89}	11500	2.2 μc	1.2×10^{13}
	Ba^{140}	2500	0.34 μc	1×10^{13}
	Ru^{103}	320	0.013 μc	---
	Ca^{45}	940	0.19 μc	---
Rongerik Americans	Sr^{89}	1870	0.42 μc	2.3×10^{12}
	Ba^{140}	890	0.27 μc	7.8×10^{12}
	Ru^{103}	180	0.015 μc	---
	Ca^{45}	90	0.04 μc	---

Several parameters must be considered in the derived information shown above. These parameters and the numbers used are shown in tabular form in the following:

(a) Radioactive half-times

1. Sr^{89} - 54 days
2. Ba^{140} - 12.8 days
3. Ru^{103} - 42 days
4. Ca^{45} - 152 days

(b) Per cent of initial body burden excreted on day of collection

1. Sr^{89} - 0.2%
2. Ba^{140} - 0.3%
3. Ru^{103} - 0.5%
4. Ca^{45} - 0.2%

(c) Per cent of exposure dose excreted on day of collection

1. Sr^{89} - 0.1%
2. Ba^{140} - 0.01%

(d) Volume of urine excreted/24 hrs/person

1. Natives - 0.45 liter

2. Americans - 1 liter

(e) Efficiency, self absorption, and yield corrected for before entry into the table.

The Japanese results have not been included in the above table in terms of any particular isotope because of recurring difficulties in the strontium method. If the highest total beta counts in dry-ashed, plated, 100 ml aliquots of urine are used a table as shown below may be formed.

Sample Source	Date of Collection	Date of Count	Counts/min per liter	% Sr^{89} in Body Excreted on Day of Collection
Rongelap natives.	16 Mar.	17 June	2040	
		7 July	1570	0.2%
	17 Mar.	17 June	1860	
		7 July	1420	
Japanese fishermen	19 April	16 June	400	
		7 July	460	0.1%
	28 Mar.	16 June	340	
		7 July	280	0.15%

If it is assumed that the counts shown above are an accurate reflection of the Sr^{89} activity as indicated by the decay of these samples, the body burden of the Japanese fishermen may be related to that of the natives or Americans. Considering the assumed differences in 24-hour volume excreted, it appears that the body burden of the Japanese is equal to or slightly greater than that of the natives. Thus, it appears that the Japanese exposure in terms of fissions was essentially equal to that of the natives.

In summary it may be concluded that the total body burdens of the natives and Japanese are approximately the same and specific isotope measurements in the case the natives indicate an exposure to $\sim 3 \times 10^{13}$ fissions. In the case of the Ameri

$\times 10^{12}$ fissions is indicated. If it is assumed that errors

are random, which can only be suspected and not proven, uncertainties of a factor of 2 can be applied to the above. These results certainly depend on the excretion numbers used. The numbers used, of course, are derived in part from animal data but are the same as those used for humans in the case of exposure through ingestion. In these cases it appears that ingestion is the only reasonable mode of entry into the body if time of fallout, particle size, and wind parameters are considered.

From these data several conclusions may be drawn. These may be annotated as follows:

1. From the analyses to date it appears that the total body load of fission products is about the same for the Japanese and for the natives. Also, the body burden of each of these groups is about four times that of the Americans.

2. In view of the widely variant habits of cleanliness, living and eating of the three exposed groups and the correlation of urine levels found, the external dose to the Japanese may be estimated as about the same as the external dose to the natives.

3. As far as acute internal dose is concerned, iodine appears most important in cases of exposure at these early times after detonation. The absolute doses to the thyroid are appreciable but low compared to the partially or totally ablating doses of I^{131} used for therapy of hyperthyroidism or carcinoma.

4. As has been expected it appears that strontium will be the bone-seeking isotope of most interest from the long-range aspect. In these cases it appears that strontium was at its maximum essentially at maximum permissible limit in the body.

5. Considering the estimated level of strontium and other isotopes at their maxima in the body, it can only be concluded that the internal hazard is negligible in these cases over the long-range point of view. Animal studies on

mice and sheep at certain field tests support this conclusion.

6. As has been predicted from animal studies, it may be further concluded that external radiation is the controlling factor in the fallout exposure situatio In order for internal dose to become an appreciable hazard, the external dose must be overwhelming.

UNCLASSIFIED

APPENDIX II-B: GORDON M. DUNNING, HEALTH PHYSICIST, BIOPHYSICS BRANCH, DIVISION OF BIOLOGY AND MEDICINE MEMO TO DR. JOHN BUGHER, M.D., DIRECTOR, DIVISION OF BIOLOGY AND MEDICINE

403830

Dr. John C. Bugher, M.D., Director
Division of Biology and Medicine

May 13, 1954

Gordon M. Dunning, Health Physicist
Biophysics Branch, Division of Biology and Medicine

ESTIMATED RADIATION DOSE TO THYROID OF NATIVES FROM RONGELAP

This memorandum is in reply to your request for an estimate of additional doses to the thyroid of the Rongelap natives due to the fact that tellurium, as a precursor to iodine, may be present in the gut after ingestion of fallout material. The tellurium, in turn, might disintegrate into radioactive iodine while in the gut, with subsequent deposition of the iodine in the thyroid.

There are some 17 radioactive isotopes of tellurium but only 7 of these are produced in fission. Of these, 6 are not of interest (4 have too short a half-life, 1 leads to stable iodine-127 and 1 leads to iodine-129 with a half-life of $2\text{-}4 \times 10^7$ years). The remaining radioisotope is tellurium-132, with a half-life of 77 hours leading to iodine-132 with a half-life of 2.4 hours. (Incidentally there is no tellium precursor that is of interest here.)

Without having the original data of LASL I have accepted their estimate that there were ingested and/or inhaled the products of 5×10^{13} fissions, assumed they were all ingested, and then proceeded to calculate the dose to the thyroid from (a) I^{131} (b) each short-lived iodine isotope of interest and (c) the added dose coming from $T^{132}\text{-}I^{132}$. The calculations show that $T^{132}\text{-}I^{132}$ will produce an added dosage of about 26%.

The best estimated percentage absorption and deposition of iodine is yet to be determined. The best estimate I can turn up to date is still the 20% quoted in NBS Handbook 52. However, I will continue to search for additional information. In the meantime the table below indicates the magnitude of doses to the thyroid if one assumes 20, 50, and 100% absorption and deposition. Incidentally, it may be noted that the calculations below based on 20% (the number assumed by LASL) show estimated doses to the thyroid from I^{131} and from shorter lived iodine isotopes to be in good agreement with those estimated by LASL, i.e., 50 reps for I^{131} and 80 reps for short-lived iodine isotopes.

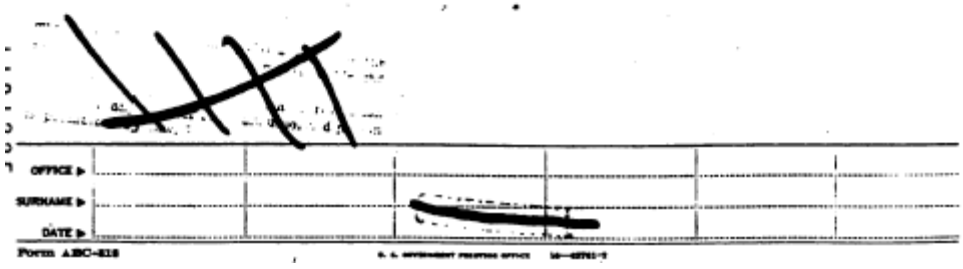
DOSE TO THYROID (REPS)

	Assuming 100% Retention		Assuming 50% Retention		Assuming 20% Retention	
I^{131}	255	255	128	128	51	51
I^{132}	27*	(54)**	14*	(27)**	5*	(10)**
I^{133}	370	370	185	185	74	74
I^{135}	60	60	30	30	12	12
$I^{132}(Te^{132})$	185*	(370)**	93*	(185)**	37*	(74)**
Total	897*	(1109)**	450*	(555)**	189*	(221)**

* If assume that one-half of the I^{132} (half-life 2.4 hours) present in the gut is deposited in the thyroid.
** If assume all of the I^{132} (half-life 2.4 hours) present in the gut is deposited in thyroid.

Most probable estimate of ratio of doses to thyroid is:

$$\frac{I^{132,133,135} + I^{132}(Te^{132})}{I^{131}} \simeq 2.5$$



ANNEX

Calculations of Dose to Thyroid

I^{131}

Assume: inhalation and/or ingestion of 5×10^{13} fissions

At D + 1 there are 0.017 d/m/100 fissions

or 8.5×10^{7} d/m/5×10^{13} fissions

or 38.3 μc intake of I^{131}

or 1.4×10^{12} atoms intake of I^{131}

(Average energy 0.22 Mev)

$$\text{Dose (reps)} = \frac{(1.35 \times 10^{12})^{*}(0.22)(1.6 \times 10^{-6})}{(20)(93)} = 255 \text{ reps}$$

* Correction for biological decay.

I^{132}

Assume: inhalation and/or ingestion of 5×10^{13} fissions

At D + 1, I^{132} intake is 1.1×10^{11} atoms

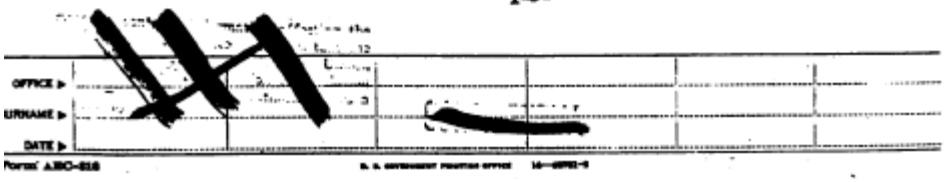
The average mean energy of I^{132} is about 0.55 mev or 2.5 times that of I^{131}.

Thus, the energy equivalant to I^{131} would be

$(1.1 \times 10^{11})(2.5) = 2.75 \times 10^{11}$ atoms of I^{131}

However, due to the short half-life of I^{132} (2.4 hrs) assume that the energy equivalent of 1.5×10^{11} atoms of I^{131} reaches the thyroid.

Thus, the ratio of doses $\frac{I^{131}}{I^{132}} = 9.0$



I^{133}

Assume: inhalation and/or ingestion of 5×10^{13} fissions

At D / 1, I^{133} intake is 1.24×10^{12} atoms

The average mean energy of I^{133} is about 0.36 or 163 times that of I^{131}.

Thus, the ratio of doses $\frac{I^{131}}{I^{133}} \simeq 0.7$

I^{135}

Assume: inhalation and/or ingestion of 5×10^{13} fissions

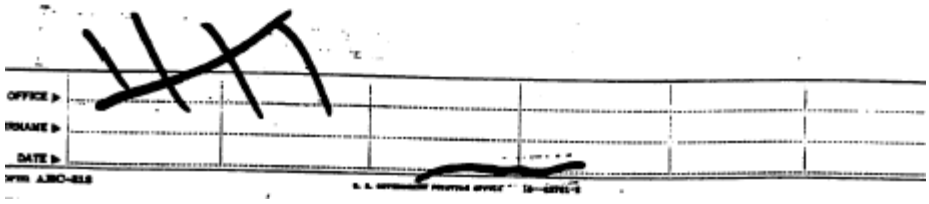
At D / 1, I^{135} intake is 2.36×10^{11} atoms.

The average mean energy of I^{135} is about 0.3 mev or 1.36 that of I^{131}.

Thus, the energy equivalent to I^{131} would be

$(2.36 \times 10^{11})(1.36) = 3.2 \times 10^{11}$ atoms of I^{131} energy equivalent.

Thus, ratio of doses $\frac{I^{131}}{I^{135}} \simeq 4.2$



$$_{52}Te^{132} \xrightarrow{77\ hr.} {}_{53}I^{132} \xrightarrow{2.4\ hrs.} {}_{54}Xe^{132}\ (stable)$$

Assume: inhalation and/or ingestion of 5×10^{13} fissions.

At D / 1, Te^{132} intake 100 μc

or 1.5×10^{12} atoms

Assume: the time spent in the gut is 77 hrs.

Then, 7.3×10^{11} atoms of Te^{132} will have disintegrated into 7.3×10^{11} atoms of I^{132}.

The average mean energy of I^{132} is about 0.55 Mev or 2.5 times that of I^{131}.

Thus, the energy equivalent to I^{131} would be

$(7.3 \times 10^{11})(2.5) = 1.8 \times 10^{12}$ atoms of I^{131}.

However, due to the short half-life of I^{132} (2.4 hrs), assume that only the energy equivalent of 1×10^{12} atoms of I^{131} reaches the thyroid.

Thus, the ratio of doses $\frac{I^{131}}{I^{132}(Te^{132})} \cong 1.4$.

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