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INHALATION OF RADIOIODINE FROM FALLOUT:
HAZARDS AND COUNTERMEASURES

ENVIRONMENTAL SCIENCE ASSOCIATES

PREPARED FOR
DEFENSE CIVIL PREPAREDNESS AGENCY

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FINAL REPORT

INHALATION OF RADIOIODINE
FROM FALLOUT: HAZARDS
AND COUNTERMEASURES

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KEY WORDS

	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Radioiodine						
Iodine						
Blocking agents						
Fallout particles						
Fallout shelters						
Thyroid uptake						
Thyroid irradiation						
Stockpiling						
Civil Defense						
Decontamination						

II

INHALATION OF RADIOIODINE FROM FALLOUT:
HAZARDS AND COUNTERMEASURES

SUMMARY

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For
Defense Civil Preparedness Agency
Office of the Secretary of Defense
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by

Richard Cole

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SUMMARYA. Objectives

1. To evaluate the nature and extent of the intake of radioiodine in the Marshall Island populations, following the CASTLE BRAVO nuclear test on March 1, 1954 (fallout from which produced extensive thyroid damage in most of the heavily exposed (Rongelap) young children), with emphasis on the possibility that inhalation was a major route of entry.
2. To estimate, on the basis of the literature on fallout phenomenology, the extent and rate of volatilization of iodine from siliceous fallout particles.
3. To calculate potential inhalation intake of radioiodine by populations in shelter during and following nuclear attack and, based on this, the expected ionizing-radiation doses to the thyroid, particularly for young children; to indicate the immediate and the long-term consequences of such irradiation of the thyroid.
4. To evaluate the use of blocking (oral administration of stable iodide) as a countermeasure to the intake of radioiodine, particularly by inhalation; specifically, to define required levels and scheduling of blocking-agent administration, and to establish implications of side effects resulting from administration of iodide in large quantities.
5. To recommend procedures for the use of blocking, if required, and to estimate national costs of such protection.

B. Assumptions

1. Arriving local (as opposed to delayed) fallout contains no particles small enough to be inhaled directly. Therefore, inhalation in shelter (on predicted fallout contours) can occur only if there is a significant level of volatilization of radioiodine from fallout particles, whether trapped in the shelter ventilation system or deposited outdoors.

2. In the light of the extreme uncertainty in the major input variables in the assessment of inhalation intake (mainly in the extent and rate of volatilization of iodine from fallout particles), it is safe to adopt an eclectic approach in assembling input information for the investigation. That is, extreme accuracy in data and calculational models is not required; in fact, order-of-magnitude approximation may often be sufficiently accurate. Thus, convenience in finding and applying source data is the guiding principle, and it dictates the choice of most of the following assumptions.

3. Characterization of the shelter intake of radioiodine on particles trapped in the ventilation system, and association of that intake with fallout-radiation contours, is based on the following assumed characteristics of the fallout:

- a. Thermal-neutron fission of U-235.
- b. No induced activities (transuranics or soil products).
- c. No loss (in particular, of radioiodine) by fractionation; similarly, no fractionation with particle size (however, fractionation losses are taken into account in interpretation of the calculated results).
- d. Transport/arrival model of Clark and Cobbin (1963).
- e. $t^{-1.2}$ decay for air-ionization rate of total fallout mixture.
- f. Radioiodine abundance, decay, and activity relationship to total R/hr as given by Kochendorfer (1969).
- g. All particle sizes present in arriving fallout being swept into the shelter ventilation system, according to their volume concentration in the arriving fallout (no aerodynamic discrimination according to size).
- h. Log-normal activity/particle-size distribution.

4. Inhalation intake of radioiodine, per individual, is independent of the shelter ventilation rate, both for particles trapped in the ventilation system and for those deposited outdoors.

5. Characterization of thyroid uptake of radioiodine and of the resulting ionizing-radiation dose to the adult thyroid, for a given intake, is based on the reported work of Mechali, et al. (1966), which rests on:

a. 75% of "inhaled" intake "retained"; 25% by direct passage through lungs, 50% by temporary retention in upper respiratory passages, followed by swallowing (the other 25% being immediately expelled by the lungs).

b. A simplified 3-compartment model for iodine processing in the body (3 first-order rate processes).

c. Radioiodine mixture from fast-neutron fission of Pu-239.

d. Inhalation intake including that of radiotellurium, especially Te-132.

e. Total absorption of contained-radioiodine beta energy by the thyroid; zero absorption of contained-radioiodine gamma energy.

6. The young-child's thyroid is 1/10th the size (mass) of the adult thyroid (2 g vs. 20 g); the child's respiration rate is 1/3 that of the adult's (0.17 cfm vs. 0.5 cfm). The thyroid uptake (expressed as percent of intake) is the same for both age groups. Thus, for inhalation in the same environment, the radioiodine ionizing-radiation dose to the child thyroid is 3.3 times that to the adult thyroid.

7. Because of its relatively low beta energy and because of the inhomogeneous structure of the thyroid, iodine-131 is only about one-tenth as effective as the other radioiodines (or as x-radiation), on a rad-for-rad basis, in producing damage to the thyroid. Therefore the most dangerous period is the first week after the nuclear explosion; after this only the longer-lived (and less-effective) I-131 remains in quantity.

8. The shelter intake of radioiodine volatilized from particles deposited outside the shelter is based on "sampling" from an outside environment characterized by one-dimensional upward turbulent diffusion of released iodine vapor in an infinite horizontal field. This is a worst-case assumption for both a normal atmosphere and a temperature inversion.

9. Shelter intake of radioiodine vapor during a temperature inversion occurs in three regimes:

- a. Upward diffusion to fill the inversion layer.
- b. Uniform, constant-concentration distribution of released radioiodine throughout the inversion layer during the remainder of the inversion.
- c. Upward diffusion (dissipation) after the break-up of the inversion.

C. Findings

1. The generally accepted (literature) dosimetry, both whole-body and thyroid, for the Rongelap children is compatible with all the clinical measurements and all symptoms, early and long-delayed; therefore, there is little to be gained by attempting to refine the intake and dose calculations.

2. For the American military personnel on Rongerik Atoll at the time of CASTLE BRAVO, the high levels of I-131 and of the non-volatile radionuclides found in the early pooled urine samples resulted from massive intake, presumably a mixture of inhalation and ingestion, on shot day and on D+1, before the men were evacuated from the Atoll.

3. The lower levels of the non-volatile radionuclides found later in individual Rongerik urine samples resulted from the same source, and were therefore valid input data for an attempted correlation of body burdens with individual location and behavior during the first two days.

4. No single factor (with respect to individual behavior or exposure) appears to account for the variation in the measured body burdens of the non-volatile radionuclides in the Rongerik men. The fallout was ubiquitous enough (even inside the buildings) for ingestion (or quasi-inhalation, by impaction) to have been a factor. Also, the high seawater solubility of the coral-type fallout could have led to rapid dissolution of radioiodine(s), followed by volatilization at the water surface. Thus, inhalation of radioiodine was a possibility also. Finally, the friability of the dry coral-type particles could have led to the formation of smaller, directly inhalable, particles; thus, even the non-volatile radionuclides could have been inhaled directly.

5. On the basis of limited direct information, it is concluded that the radioiodine volatilization rate from dry siliceous fallout particles is of the order of 0.00025% per day.

6. Where siliceous fallout particles are exposed to water, radioiodine may be leached out of the particles at a much higher rate than the direct-volatilization rate. The apparent half-life for leaching of the available radioiodine may be only about 1.5 days, but the available radioiodine may be only a fraction of the total radioiodine distributed through the near-surface layers of the particle. Once dissolved in water, iodine (even as iodide, which can be oxidized) is immediately available for volatilization.

7. Where coral-type fallout particles are exposed to water, very rapid release of the radioiodine to the water is likely, so that the volatile-radioiodine threat may be considerable in such a situation. Also direct volatilization of iodine from such particles, even when they are "dry", may be faster than in siliceous particles.

8. Nuclear weapons exploded at the surface over limestone or in central sections of cities, where there may be high concentrations of concrete, may produce fallout particles containing calcium oxide and related calcium compounds in concentrations high enough to produce more extensive and more rapid release of radioiodine to water (compared to that from totally siliceous particles) and, perhaps, more rapid direct volatilization from "dry" particles.

9. Under worst-case assumptions, the radioiodine inhalation threat from particles trapped in shelter ventilating systems is minimal, particularly when the resulting thyroid dose is compared with that resulting from whole-body irradiation by penetrating radiation (even with a shelter protection factor of the order of 40).

10. It appears that the only situation in which inhalation of radioiodine by shelter populations can create a threat that must be countered is one in which: (a) a long-lasting temperature inversion accompanies or immediately follows the arrival of high levels of local fallout; and (b) there are significantly large areas of shallow water nearby, into which fallout particles (particularly if they contain significant percentages of calcium oxide and related compounds, deriving from limestone or concrete at the burst point) can deposit.

11. Because the important variables in this specific kind of threat situation are almost impossible to parameterize for planning purposes, it is not feasible to calculate, for a given assumed attack, the number of individuals who will be (ultimately) injured if not protected, at the time of fallout arrival, against the effects of intake of radioiodine. Nevertheless, the threat of such injury appears serious enough, and the cost of the appropriate (blocking) countermeasure low enough, that it is worth planning such a countermeasure.

12. Blocking with stable iodide works by a combination of isotope dilution and saturation of the thyroid against further uptake of iodine in any form.

13. In the civil-defense context, it appears that blocking will be useful only if it reduces the uptake of iodine to 1% of normal, or less. This implies the administration of 200 mg of potassium iodide just prior to, or at the start of, the arrival of fallout, to protect against a massive intake of radioiodine on the first day.

14. Protection against continuing inhalation during the remainder of the first week would be provided by the administration of an additional 400 mg of KI at the rate of 100 mg per day over the next 4 days, or spaced over the next 6 days.

15. The cost of this first-week protection (in the form of a stockpile of 1.2 billion 100-mg tablets, stored in the shelters) for 200 million people would be, at most, \$1.2 million, or less than a penny per person so protected.

16. If blocking iodide is to be stockpiled, in any event, against the possible massive early inhalation threat, then it seems worthwhile to be prepared to counter the other, less-important or more-easily-controlled, threats (delayed fallout, quasi-inhalation during outdoor activities, contaminated-milk intake) by the stockpiling of additional iodide. 80-day protection against any or all of these would require (at 200 mg of KI on D-day, followed by 100 mg per day for the next 79 days) 8.1 g of KI per person, which for 200 million people would cost at most \$16.2 million, or about \$0.08 per person.

17. Possible side effects of prolonged administration of blocking-iodide (iodine allergy, iodide goiter, etc.) must be taken into account. However, the important consideration is the possible outcome if blocking is not used. That component

of the population that is at the greatest risk (to ionizing-radiation-produced thyroid pathology) is the infant or very young child, and perhaps the fetus in utero. The general adult population is least at risk.

18. For all members of the population, the administration of a one-time blocking dosage of 200 mg of KI appears to be quite safe; even administration of a total of 600 mg over the course of 5-7 days appears safe for the vast majority of the population. Administration of 100 mg daily for a period of say 2 months should be safe for most of the population; for those for whom it may be known to be risky (individuals suffering from renal or cardiac failure or from pulmonary tuberculosis), decisions would depend on circumstances.

19. Incidence of the major known side effects of prolonged administration of high levels of stable iodide is statistical, rather than deterministic; that is, predisposition to the effects is probably involved, but the nature of the predisposition is not known. However, if, for example, an unknown 10% of a population may exhibit such side effects, it is undoubtedly better to protect 100% of the population against a high risk of radioiodine uptake, whose consequences are usually irreversible, and take the risk of side effects in the unknown 10%, particularly since most of the side effects are reversible, disappearing on cessation of excess-iodide intake.

20. The most dangerous side effect for healthy members of the population is the occurrence of iodide goiter in some infants born to mothers who have ingested large quantities of stable iodide (500-1000 mg or more daily) over long periods (usually of the order of years). The rate of incidence of this condition for a population of pregnant women so exposed is unknown, but probably low; however, where it does occur, the risk of fatal respiratory obstruction in the newborn is extremely high. It is not known whether daily intake as low as 100 mg can produce the effect. If there is a known (high) risk of radioiodine uptake by the fetal thyroid (that is, if it is known that the mother is being exposed to radioiodine intake), it would always be wise (statistically) to administer blocking iodide to the pregnant woman. Where the risk of radioiodine intake is not certain, the picture is not as clear. However, the neonatal iodide-goiter syndrome is very unlikely to develop after only a one-time, or even a one-week, administration of stable iodide to the pregnant woman. Therefore, the best prophylaxis might be to protect her (and thereby the fetus) against massive early intake of radioiodine, by one-week administration of iodide, and then take special pains to protect the mother against subsequent intake of radioiodine for the remainder of

her pregnancy, so as to obviate the need for further administration of blocking agent to her.

21. As a general rule, the nursing infant should receive blocking agent directly; if the mother is receiving blocking agent, her milk may contain more radioiodine than it would if she were not so protected.

22. The philosophy of selective administration of blocking agent can be applied to the entire shelter population. That is, in the face of uncertainty at early times after attack, protect everyone by first-week administration of blocking agent; continue administration as needed, depending on habits and on subsequent ingestion history, and on increasing knowledge (perhaps measurement) of the radioiodine threat.

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* Affiliations given in "REFERENCES," following main text.

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SECTION I

INTRODUCTION

On March 1, 1954, the BRAVO event of Operation CASTLE contaminated several occupied areas in the Marshall Islands to levels high enough to require early evacuation of the populations, and, for some populations, medical follow-up to this day (Conard, et al., 1967 and 1970). The most heavily exposed group, some 64 Marshallese inhabitants of Rongelap atoll, received an estimated 175 rad whole-body dose before being evacuated on March 3. While internal contamination was of concern (indeed, measurements were made of iodine-131 in pooled urine samples about two weeks after exposure), the consensus of the scientists and medical people at the time was that internal burdens of radionuclides were not likely to produce injury, even in the heavily exposed group.* Indeed, as of 1960 it was accepted that "the Marshall Island people were apparently fully recovered from their accidental exposure to thermonuclear fallout in 1954" (Le Roy, 1969).

However, in 1963 an asymptomatic thyroid nodule was discovered during routine examination of a (then) 12-year-old girl, and in the following years additional such cases were discovered during the annual medical surveys. By 1969, 15 of the 19 children who had been under 10 years of age at the time of exposure had developed thyroid nodules, and another 2 had severe atrophy of the gland (Conard, et al., 1970). Fourteen of the nodule cases involved benign nodules, while 1 was a malignant tumor. The method of treatment of all 15 nodule cases was surgery, either total or sub-total thyroidectomy.

* All early symptoms were due to whole-body exposure doses and to skin-deposited fallout particles.

1-2

No children in the less-heavily-exposed populations (whole-body doses up to about 70 rad) have developed thyroid abnormalities to date. Adults in the Rongelap group have developed thyroid lesions, but the rate of incidence is less than one-tenth that in the children.

It seems clear that the cause of the thyroid abnormalities was (beta) irradiation of the thyroid by the mixture of radioiodines concentrated in that gland after ingestion and/or inhalation. For roughly equal intake of radioiodine by children and adults (and there is some evidence that this occurred) a 2.5 gram child thyroid would have received about eight times the exposure dose of the 20-gram adult thyroid. After the onset of thyroid abnormalities in the Rongelap children, an estimate of their thyroid dose from I-131, 132, 133 and 135 was made (James, 1964), based on the measurement of I-131 in pooled urine mentioned earlier. The "most probable" child thyroid dose was found to be between about 500 and 1250 rad* from radioiodine uptake alone, to which must be added 175 rad of whole-body exposure. The thyroid dose in children was therefore taken to be about 1000 rad, including the whole-body exposure.

It is likely that there is more trouble ahead for the Rongelapese, particularly those who were adults at the time of exposure, and for the less-heavily-exposed populations, particularly the children. If the latency period for neoplasms in general, and cancers in particular, is inversely related to dose, then additional statistically expected neoplasms for the Rongelap adults and for the other populations may occur in the future. It is known also that (expected) thyroid neoplasms are much more likely to occur

* The lower limit of the range was the most probable dose if inhalation was the route of entry; the upper limit was based on ingestion.

during adolescence and pregnancy, periods of great metabolic stress, than at other stages of development; this could be part of the explanation of the bunching of effects for the Rongelap children (14 cases of nodules in a 3-year period, centered 11 years after exposure).

The experience of the Marshallese children illustrates one combination of conditions leading to a serious radioiodine threat; by its very occurrence it suggests that worse radioiodine threats can occur in nuclear warfare. The children were exposed, for the first two days, to all of the radiological effects of the fallout field ("standard" exposure rate of the order of 100 R/hr* at 1 hr). They received whole-body exposure doses of the order of 175 rad, because they were essentially unsheltered. The air they breathed, during and after fallout deposition, was unfiltered. They ate contaminated food and drank contaminated water.

Had the "standard" exposure rate at Rongelap been even a factor of three higher, more than half the exposed individuals would have died, within a few weeks to a month, from the whole-body gamma-radiation exposure. The survivors would have been seriously ill and would have experienced other long-term effects, so that the internal radioiodine problem might have been relatively unimportant. In nuclear warfare, a large fraction of the area of the continental United States could experience "standard" exposure rates that are a factor of 30 or more higher than the Rongelap figure; in such areas

* This number is not known with any accuracy, since it is based on back-extrapolation from the earliest radiac reading, taken at H+36 (H+50?) hr, and the exponent in the decay equation during the first day or so may be quite different from 1.2. However, the exposure dose cannot be far from the reported value. This will be discussed subsequently in more detail.

most of the unsheltered (protection factor less than 10-20) individuals would die within one month or so. Approved (marked) shelters having a protection factor of forty would reduce the shelter-period whole-body gamma-radiation exposure, even in such fields, to about what the Rongelap children received (and subsequent decontamination of occupied areas would continue the protection after the shelter period). However, the radioiodine intake could be, as a first approximation, a factor of 30 higher than that at Rongelap if survivors were to eat contaminated food, drink contaminated water, or milk produced by cows grazing on contaminated pasture, and breathe unfiltered air during and after fallout deposition. Thus the relative importance of the internal radioiodine threat is enhanced by improved shelter in higher-"standard"-exposure-rate locations and by the absence of controls on ingestion or inhalation of fallout radioactivity.

Complete protection would be provided if (1) only filtered air were breathed and (2) only pre-attack-packaged food and water were consumed, during the entire period of potential danger from radioiodine (roughly 30-40 days, or 4-5 half lives of the longest-lived fission-product iodine radioisotope, 8-day ^{131}I). Since (vapor) filtration of air is not a standard procedure in marked fallout shelters, any radioiodine in the gaseous state would reach the shelterees; particles stopped on particle filters or in the ventilation ducting could still release their radioiodine in gaseous form over time, and thus be a continuing threat.* Furthermore, marked fallout shelters are stocked with food and water for two weeks or less of occupancy; this leaves part of the ingestion-threat period uncovered also.

* Thus, in the inhalation context there are two important questions: (1) Are there shelter situations in which fallout particles themselves can be inhaled; and (2) To what extent is fallout-particle radioiodine released by volatilization?

For delayed (small-particle) fallout, it has been shown that under the exposure combination type experienced by the Marshallese (food, water and air all contaminated) the radioiodine uptake from inhalation is minor compared to that from ingestion (Wehmann, 1963). Thus the major portion of the radioiodine threat could be countered by pre-attack shelter storage of additional consumables, particularly water and powdered milk, to take care of the special needs of infants and children. Nevertheless, if volatilization of iodine is significant one can visualize situations in which, given essentially complete protection against whole-body gamma radiation and radioiodine (and other radionuclide) ingestion, the youngest shelterees (and, to a lesser extent, the adults) might still inhale sufficient radioiodine to suffer serious problems 10-15 years later, and perhaps earlier than that.

Installation of effective vapor filtration and stocking of additional food and water in all marked shelters would be a fairly expensive or cumbersome countermeasure. The suggested oral administration of stable ("blocking") iodine to reduce the thyroid uptake of radioiodine appears to be much less expensive. Whether such a countermeasure is needed, exactly what it would consist of, and how expensive it would be, were the subjects of the research reported here.

In the next section of the report, the scope and emphasis of the study will be presented and justified, and the method of approach will be laid out.

SECTION II

SCOPE AND APPROACH

In the civil-defense context, as has been mentioned, the total problem could involve intake of radioiodine(s) by inhalation, and by ingestion of water and milk. However, the emphasis in this study has been on the inhalation problem, for the following reasons:

1. By and large, the ingestion problem is more amenable to dealing with after the fact; that is, the ingestion threat can be delayed by use of stored products first, so that, at worst, ingestion of contaminated material will not occur until there has been some radiological decay*. In particular, the more-dangerous short-lived radioiodines would be gone.

2. From a phenomenological point of view, analysis of the ingestion problem is more straightforward than that of the inhalation problem. That is, the nature and magnitude of the ingestion problem are dependent primarily on fallout levels, to a lesser extent on the physical nature of the fallout, and not at all on the details of the arrival process. Because of the delay in the use of contaminated foods, only I-131 would be of concern. Thus, calculation of the ingestion situation can be handled by a brief phenomenological analysis of existing data (I-131 intake at low fallout levels) primarily for low-yield explosions on siliceous soil in the continental United States, followed by scaling up to higher fallout levels. On the other hand, inhalation phenomenology is complex, depending on details of the fallout-arrival process, ingress of particles or vapor into ventilation systems, and vaporization at early times, when the mixture of radioiodines is still rich.

3. The ingestion problem, for phenomenological situations close (technically) to those in the civil-defense context, has been and is being examined in other studies, in civil defense and other contexts.

* In any event, food production itself consumes time. For radioiodine ingestion, the critical foodstuff would be cow's milk; availability of that product would depend on a chain of events that would be inhibited in intense fallout fields.

4. The blocking countermeasure would be more effective against the inhalation threat than against the ingestion threat. A safe quantity of stable iodide* taken at one time, is known to provide protection for several days; thus, the relatively short-lived inhalation threat can be countered cheaply, conveniently and safely. On the other hand, the more-protracted ingestion threat would require repeated administration of the same levels of iodide, a course that is not assuredly safe, particularly for infants.

The structure of the report follows from this emphasis on inhalation. The first area to be discussed, in Section III, is the Marshall Island experience following CASTLE BRAVO. Questions which are considered therein are:

1. What actually happened to the Rongelap infants? That is:
 - a. What was their mode of intake of radioiodine? In particular, could inhalation have been an important factor?
 - b. What was their total intake and uptake of all the radioiodines? How well has their thyroid dose been characterized?
 - c. Was the subsequent pathology consistent with the estimated thyroid dose?
2. Is enough known from the above and from similar questions applied to the adults to provide a basis for judging their prognosis?
3. To what extent is the Rongelap experience a model for predicting the radioiodine threat in the CONUS civil-defense context? The answer to this question must include consideration of differences in fallout-particle phenomenology in the two locations, as well as of differences in location, protection and habits of the people involved.

* Say one capsule, several hundred mg, of potassium iodide.

The discussion in Section III includes supporting information based on: (1) the experience of the American military personnel on Rongerik, also heavily contaminated by early fallout from CASTLE BRAVO; and (2) the history of the Utah children who were exposed to radioiodine intake as a result of Nevada Test Site detonations in the 1950's and 1960's. The discussion is backed up by material in several of the appendices to this report, whose specific content will be indicated later in this description of the report structure. However, in general this supporting information includes: nuclear, physical and chemical properties of the radioiodines in fallout particles; thyroid function (including iodine-uptake rate and extent); thyroid pathology, with particular reference to the effects of ionizing radiation; and a detailed assessment of the source of the measured individual body burdens of radionuclides in the Rongerik servicemen.

In Section IV, the inhalation problems in the civil-defense shelter environment are characterized, in terms of two potential sources of inhalable radioactivity: (1) gaseous radioiodines originating from particles trapped in the ventilating system; and (2) those originating from particles deposited outside the shelter. In addition to some of the kinds of uncertainty already well characterized by Stocum (1968) for the iodine-in-milk route of ingestion, the uncertainties as to the extent of volatilization of radioiodines from siliceous fallout particles must be considered in the assessment of the threat of radioiodine inhalation in shelter. The latter uncertainties are shown, in Section IV, to dominate the assessment.

Blocking (administration of stable iodide to reduce the uptake of radioiodines) as a countermeasure for both inhalation and ingestion is evaluated in Section V. Discussed

therein are dosages and schedules of administration, effectiveness in reducing potential beta-ray dose to the thyroid, and possible side-effects of the countermeasure.

The work is summarized in Section VI, which contains conclusions and recommendations.

Appendix A is a detailed attempted assessment of the source of the measured individual body burdens of radionuclides in the Rongerik servicemen, by means of an attempted correlation between those measured levels and the estimated exposures of the individual men to possible sources of intake.

Appendix B is a comprehensive evaluation of the measured extent of volatilization of radioiodine(s) from fallout particles in the SUNBEAM series at the Nevada Test Site in 1962.

Appendix C contains the basic information on the nuclear and physicochemical properties of the fission-product radioiodines in fallout particles.

Appendix D is a presentation of the methodology used in the estimation of the extent of penetration of gaseous radioiodine in the shelter environment, with some results.

Appendix E is a brief summary of background information on normal-thyroid function, iodine-uptake modes and extent, and the models used to characterize the behavior of iodine in the thyroid and in the thyroid hormones.

Appendix F is a brief summary of background information on thyroid pathology, with particular reference to that induced by ionizing radiation. It includes estimates of risk per rad of thyroid exposure dose, for both benign and malignant tumors.

SECTION III

THE MARSHALL ISLAND EXPERIENCE*3.1. Introduction

The salient features of the experience of the Marshall Islanders exposed to fallout in 1954 have been presented in Section I of this report. In Section II, it was indicated that there are key questions about that experience which have to be answered if conclusions are to be drawn about the civil-defense implications of what happened on Rongelap Atoll (where a population of 64 received whole-body doses of about 175 rad from penetrating radiation), and to a lesser extent on Ailinginae (corresponding doses of about 70 rad). Unless it is noted otherwise, the discussion following will refer to the first group only.

The purpose of the development that follows is to answer those questions, at least within the accuracy actually required in the present evaluation. It is anticipating a little, but it should be pointed out that, as the development in Section IV and Appendices B and D will show, the uncertainties in the extent of volatilization of radioiodines from siliceous** fall-out particles (in a continental-United-States (CONUS) context) will dominate the evaluation so overwhelmingly that large inaccuracies in documenting the Marshallese experience can be tolerated. It turns out that some of the important features of that experience are known fairly accurately anyway.

* This Section includes some information on thyroid problems (1) in Utah children following weapon tests at the Nevada test site and (2) in the Hiroshima/Nagasaki survivors.

**Or particles formed by detonations over limestone or concrete masses.

The long list of references with Dr. R. A. Conard as sole or senior author (plus Robbins, Rall and Conard, 1967) document the medical history of the Marshallese, and provide some of the required fallout phenomenological background also. The thyroid-dose calculations used by Conard, et al. were made by James (1964), based on urine-radioiodine measurements on the Marshallese that had been made by Harris (1954). Supporting information on the phenomenology of the fallout* from Castle BRAVO, some of which had provided input to the estimates of Conard, et al., can be found in Sondhaus and Bond (1955), Cronkite, Bond and Dunham (1956), Sharp and Chapman (1957), Cohn, et al. (1955/1957) and J.C.A.E. (1957).

Figure 3.1 is a dose-contour map of the Marshall Islands following CASTLE BRAVO, taken from Effects of Nuclear Weapons (1962). The Rongelapese caught on their home atoll were on their home island, at the southern tip of the atoll, on D-day; the American station on Rongerik atoll also was at the southern end of that atoll. Thus, both populations were at the safest points in their respective atolls, and both were exposed to about the same fallout exposure rate. Contours are the estimated total doses (air exposure doses) accrued in the open through H+96 hr; the numbers are thus higher than the maximum doses actually accrued by the Rongelapese on their home atoll (175 rad) or on Ailinginae (70 rad), since they were all evacuated to Kwajalein on D+2 (Rongelap group left at H+51, Ailinginae group at H+54); similarly, the 28 American military personnel on Rongerik Atoll were evacuated to Kwajalein on D+1, one group leaving at H+30, another at H+34.

* Fallout-arrival times, measured exposure rates at later times, fallout decay rates and average energies, etc.

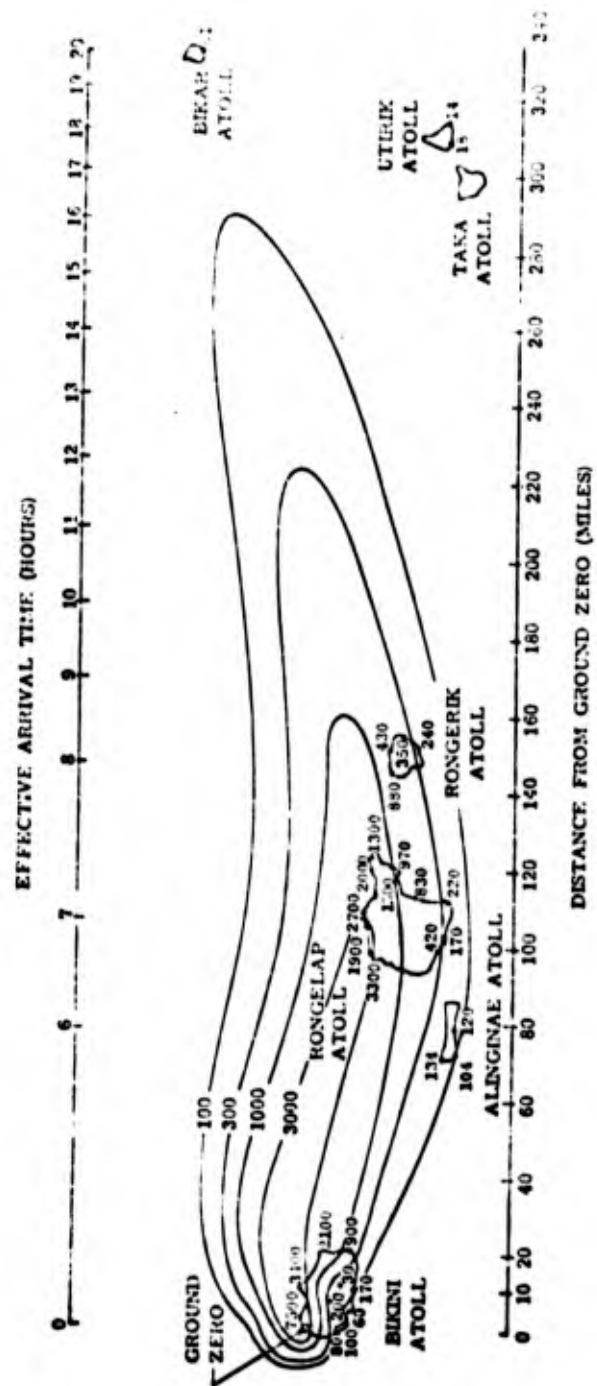


FIGURE 3.1
 ESTIMATED TOTAL-DOSE CONTOURS IN ROENTGENS AT 96 HOURS AFTER THE BRAVO
 TEST EXPLOSION

Accrued whole-body doses were calculated by Sondhaus and Bond (1955), based on exposure-rate measurements in the field at the time of evacuation and subsequently, and on extrapolation backward in time on the basis of decay rates measured by other projects on collected fallout samples. Their calculated doses were backed up by readings on dosimeters in use at Rongerik. Use of the $t^{-1.2}$ law can lead to serious errors at early times, where the contribution of induced transuranics may be high, leading to slower decay. In particular, calculation of the so-called standard intensity, or hypothetical exposure dose rate at H+1 hr, may be quite inaccurate.* However, dose calculations do not have to go back that far, since fallout did not start to arrive at Rongerik until about H+7 (about H+4 at Rongelap).

The calculated whole-body dose of 175 rad for the Marshallese on Rongelap cannot be off by very much. The widespread early symptoms of radiation sickness (2/3 had nausea during the first two days; 1/10 had vomiting and diarrhea) and the levels of the clinical measurements imply that the average dose could not have been very much lower than 175 rad (certainly more than 100 rad); conversely, the absence of any early fatalities (say within several months) implies that the average dose could not have been very much higher than 175 rad (certainly much less than 300 rad). Radiologists may argue that the demonstrated dose range is narrower than this. (James (1964) has put the range at 150 - 200 rad). The fact that the Americans on Rongerik, with a calculated whole-body dose averaging 78 rad (backed up by dosimeters), had no

* True standard intensities on Rongelap and Rongerik (after correction for inaccuracies in instrument calibration) would have been about 150 R/hr if later readings had been extrapolated back on the basis of the $t^{-1.2}$ law. A more-reasonable value (slower decay during first two days) is about 100 R/hr at 1 hr, for both places. With some reported decay constants, the number can be as low as 50 R/hr at 1 hr.

early symptoms supports a lower limit of much more than 100 rad on the Rongelap dose.

3.2. Thyroid Pathology and Dosimetry

The stimulus for the present evaluation was the late development of thyroid abnormalities in the Marshallese (Rongelap) population; the most pronounced effect was in those who had been less than 10 years old at the time of exposure. Of the 19 in this category in the most heavily exposed group (700-1400 rad to the child thyroid gland, all but 175 rad being from radioiodines taken up by that gland), 15 had developed benign thyroid nodules by 1968; 2 others had exhibited stunted growth and thyroid atrophy with almost complete depression of thyroid function. Thus, 17 out of 19 (89.5%) had severe thyroid damage. Since then (1969), one of the 15 children with thyroid lesions has developed a malignant lesion of the thyroid. On a straightforward percentage basis, the incidence of benign lesions in those over 10 years old at time of exposure (3 in 34^{*}, or 8.8%, by 1969) is about a factor of 10 below that in the children.^{**} The difference is accounted for by the fact that the thyroid dose in the older group was much less than that of the children; in the teenagers and adults, roughly the same quantity of radioiodine was taken up and distributed throughout a much larger gland (ca. 20 g for adults, ca. 2.5 g for very young children). Two individuals in the older group have exhibited malignant lesions of the thyroid; one in 1965, with no prior benign lesions.

* The basis of the calculation is the surviving population as of 1969. Between 1954 and 1969 there had been 11 deaths in the over-10-yr-old-in-1954 group. None was related to thyroid pathology.

** As of the Spring, 1971 medical survey, there were no further changes in any of the quoted incidence numbers (Conard, 1971).

the other in 1969, with prior benign lesions.*

The uncertainty in dosimetry for the internal emitters is only partially illustrated by the quoted range of 700-1400 rad to the child thyroid (as calculated by James, 1964). The sole basis for that dosimetry is one urine sample (pooled over the entire population of 64: children and adults, male and female), taken on D+17. On the basis that the 16th-day urine output of stable iodine (or decay-corrected I-131) is 0.1% of the thyroid burden 24 hr after intake,** Harris (Los Alamos Scientific Laboratory, data cited in Cohn (1955/1957) and Cronkite, et al. (1956)) extrapolated the measured activity back to give a thyroid burden of about 11.2 μCi of I-131 one day after exposure. This is an average value for the whole Rongelap (on Rongelap) population. As a first approximation, it can be taken to be the average for the adult population. James (1964), in calculating thyroid doses from this one measurement, assumed two extremes in the mode of intake: all from inhalation, or all from ingestion. He calculated adult and child thyroid doses for each extreme, and then stated that the actual dose for each population must lie between the extremes, since intake presumably was by a mixture of the two modes. The range of 700-1400 rads given above for the children is the

* The major reasons for the failure on the part of the medical survey team to anticipate the development of the thyroid pathology were: (1) the thyroid doses computed in 1954 had been in a region considered safe on the basis of I-131 diagnostic dosage history (see Appendix F); and (2) the normal protein-bound iodine (PBI) levels of the Marshallese are high compared to United States levels; thus the depression in the Rongelap children's PBI's resulting from the early thyroid damage led to levels that looked normal (see Appendix E). According to Robbins, Rall and Conard (1967), normal Marshall Island PBI's are in the range 3.9 - 12.0 $\mu\text{g}\%$, compared to U.S. levels of 2.5 - 6.9. (The measure of abnormality in the U.S. is the excess over 8. $\mu\text{g}\%$). Later measurements and experiments showed that the elevated portion of the normal Marshallese PBI was extra-thyroidal, rather than hormonal, in origin.

** The basis for this assumption is discussed in Appendices A (Section A.3) and E (Section E.5).

result of this scheme; however, each endpoint itself has a large uncertainty, as will be shown.

First, because of uncertainty in the 16th-day 0.1% excretion number used in estimating the initial thyroid burden (see Appendix A, Section A.3 for details), James indicated that the adult I-131 thyroid burden should be in the range 5.6-22.4 μCi (11.2 μCi being a geometric mean value). For calculation on the basis of inhalation alone, he assumed that inhalation occurred only during fallout arrival, and that the volume of air respired by a 3-4 yr old girl was about 0.3 times that for an adult. From the second assumption, the range for the initial I-131 thyroid burden of such a child would be 1.7 - 6.7 μCi , geometric mean 3.4 μCi . For calculation on the basis of ingestion alone, he assumed that the primary source was contaminated drinking water; since water was being rationed, it was reasonable to assume that children and adults all consumed the same volume of water. For ingestion, therefore, both children and adults were assumed to have an initial I-131 thyroid burden in the range 5.6 - 22.4 μCi , geometric mean 11.2 μCi . One-third of the ingestion was assumed to occur at H+10, two-thirds at H+30. For the 3-4 yr old child, James took the thyroid mass to be 2.5 ± 0.6 g, a number determined for a large population of New York children, so he used a range 1.9 - 3.1 g.

He now had to compute, in addition to the doses from I-131 for his ranges of assumed values, the doses from the other radioiodines. Items contributing to the differences in dose from the various isotopes are: (1) the fission yields of the different chains (see Appendix C); (2) the average energies deposited in the thyroid per disintegration for the different radioiodines (see Appendices C and E (Section E.6)); and (3) radioactive decay of each isotope before inhalation or ingestion (see Appendix C).

Summing up over I-131, I-133 and I-135, and taking account of all the ranges enumerated above, James computed the entries for his exposure table, reproduced here verbatim as Table 3.1.

TABLE 3.1
Summary
Thyroid Dose (Rads) to Rongelap Girls Ages 3 - 4

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

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

Note that while the most-probable child's-thyroid dose (radioiodine plus whole-body) is in the range 700 - 1400 rad (685 - 1445 rad, as calculated), the possible range is 350 - 3500 rad. Partial resolution of the uncertainty could come from a better understanding of the mode of intake, which would be important in its own right in civil-defense planning. This question is considered later in this Section and in Appendix A.

For purposes of dose/effects evaluations, most authors have used a geometric mean (close to the arithmetic mean) of the most-probable range, i.e. a dose of 1000 rad to the child thyroid.* However, a small error has crept into the literature.

* In Appendix F, it is demonstrated that this thyroid dose is consistent with the pathology that developed over the years.

in that some have erroneously added the whole-body dose of 175 rad, to give a total thyroid dose of 1175 rad (for example, Conard, et al. (BNL 50220, 1970)). This error is, of course, inconsequential in view of all the uncertainties associated with the dosimetry, but it can lead to confusion in comparisons of dose/effects numbers of different authors.

By calculations like those leading to Table 3.1, James (presumably assuming 20 g as the mass of the adult thyroid) computed that the most-probable thyroid dose for the Rongelap adults was 160 rad* from radioiodines alone (approximately the same value for pure ingestion as for pure inhalation, since intakes were assumed the same for both modes for adults), to which must be added 175 rad whole-body, to give a total most-probable dose of about 335 rad. For the adult thyroid dose, there would be a much narrower total range of uncertainty than that for the children (say over a factor of 4-5, rather than a factor of 8-10).

3.3. Mode of Intake of Radioiodines

In subsection 3.2, it was pointed out that James (1964) had used original-source data of Harris, on the I-131 content in an early pooled Rongelap urine sample, in his calculation of the Rongelap thyroid doses. When a copy of the unpublished Los Alamos memorandum (Harris, 1954) became available to this project, it was seen that there was additional information which might shed light on the mode of intake. This was the reported information on the American military personnel exposed at Rongerik Atoll, information that raised disturbing questions. The data showed that the 28 American servicemen on Rongerik, who had an average whole-body dose of 78 rad, had a radioiodine burden leading to a thyroid dose of 50 "rep", while the 45 Rongelap adults, who had an average whole-body dose of 175 rad, had a radioiodine burden leading to a thyroid dose of 150 "rep".

* James notes that the Los Alamos (Harris) estimate of the adult thyroid dose had been 150 rad.

Thus, the Rongerik/Rongelap ratio for intake is about 1/3, while the corresponding ratio for whole-body dose is about 1/2.5. This rough equivalence implies that either (1) the major route of intake of I-131 was inhalation; or (2) the Rongerik servicemen were eating food and/or drinking water that was as contaminated as that on Rongelap; or that both events were important factors in the intake. Early summaries (Cohn, et al. (1957), Cronkite, et al. (1956)) had indicated that the Rongerik food and water supplies consumed before evacuation of the servicemen on D+1 were not heavily contaminated by fallout; certainly not to the extent seen at Rongelap, where rainwater falling on an already contaminated roof on the afternoon and evening of shot day was diverted to a collecting cistern and drunk immediately. Quotations from the definitive report (Sharp and Chapman, 1957) indicated that the Americans on Rongerik had radiacs which indicated the arrival of fallout, that they covered up and took shelter in aluminum Butler buildings as soon as they could, and that they showered and changed clothing, all of which imply that ingestion should not have been a significant route for radioiodine uptake. However, Sharp and Chapman also reported the presence of large quantities of fallout particles inside the Rongerik buildings. Furthermore, the presence of non-volatile radionuclides (Sr-89, Zr/Nb-95, Ba/La-140) in the individual urine samples of the Rongerik Americans at D+44 (Cohn, et al., 1957) implied ingestion, or a quasi-ingestion (impaction/swallowing) route. Again, these levels were roughly proportional to those for the Rongelapese.* Since the data of Cohn, et al.

* An even more significant indicator of ingestion, or of quasi-ingestion, was Harris' (1954) finding of roughly proportional (Rongerik/Rongelap) quantities of Sr-89 and Ba-140 in the pooled Rongerik urine sample. However, in both populations, equivalent fissions based on I-131 were about a factor-of-5 higher than those based on the non-volatiles.

was obtained for the Rongerik individuals, rather than for pooled samples, it could be checked against their reported movements and habits during the exposure period.

Appendix A is an evaluation of the possible modes of intake of the non-volatile radionuclides in the Rongerik Americans, by means of tests of hypotheses based on measured burdens for specific individuals, coupled with the reported whereabouts and behavior of the same individuals during the critical period. It contains also an assessment of the possibility that the measured urine levels on D+17 (I-131 and non-volatiles) and D+44 (non-volatiles only) represented low-level intake of late-arriving worldwide fallout, rather than massive intake at Rongerik on D-day and D+1. The conclusions of Appendix A are as follows:

1. The high levels of I-131 and of the non-volatile radionuclides found by Harris (1954) in the pooled Rongerik urine sample taken on D+17 (CASTLE BRAVO) resulted from massive intake, presumably a mixture of inhalation and ingestion (or impaction/ingestion), on shot day and on D+1, before the servicemen were evacuated to Kwajalein.

2. The lower levels of the non-volatile radionuclides found by Cohn, et al. (1957) in individual Rongerik urine samples taken on D+44 (CASTLE BRAVO) resulted from the same source, and were therefore valid input data for an attempted correlation of "body burdens" with individual whereabouts and behavior during the first two days.

3. No single factor (with respect to individual whereabouts, behavior or exposure) appeared to account for the variation in the measured "body burdens" of the non-volatile radionuclides in the Rongerik servicemen. Fallout on Rongerik was so ubiquitous, apparently, that it would have been almost impossible for the men to avoid ingestion, to say nothing of inhalation (the latter being a possibility if there had been volatile forms of radioiodine in the air, as a result of the peculiar properties of coral-type fallout*, particularly when

* Per the discussion of Section IV and Appendix B, there is no firm evidence that iodine associated with dry siliceous fallout will volatilize to a significant extent.

exposed to the large shallow areas of adjacent lagoon and ocean).

In the context of the civil-defense problem, involving fallout from detonations primarily over siliceous soils, the physicochemical properties of the coral-type fallout seen in the CASTLE series cannot be considered to be representative. CASTLE land-surface bursts produced fallout particles that, upon arrival at the surface, were mixtures of calcium carbonate and hydrated calcium oxide, both highly soluble in acid solutions, the latter moderately soluble in water also. Rain and/or surface moisture might have dissolved some fraction of the particulate, thus freeing the iodine; it might also be that the fallout which deposited in the ocean and the lagoons was a very large source of volatile iodine. Volume III of the "Fallout Data Compilation" (Kawahara, et al., 1966) contains some information on the chemical and physical state of the radioiodine in CASTLE-series fallout. For four of the high-yield detonations, the oxidation state of the iodine was established by a relatively insensitive method; the predominant species was found to be iodide. In fallout from coral-surface bursts, (sampled in "total"-collectors which contained rain and/or sea spray) most of the iodine (50-80%) was found in the solid phase of the recovered fallout samples*. In the fallout from water-surface bursts, collected the same way, all the iodine was found in the liquid phase (whereas as much as 25% of the total activity in the same water-surface-burst fallout samples would settle in ordinary centrifugation).

Thus, even if volatilization did occur at Rongelap and Rongerik (and there is only limited evidence that it did), this would not imply that there would be significant volatilization in the CONUS attack situation.**

* Nevertheless, a significant fraction (20-50%) of the iodine from particles landing in the water would have dissolved, and thus been available for volatilization.

**Possible exceptions - surface detonations: (a) on limestone; (b) in cities with massive concrete structures. Note also that attacks on Hawaii and other partially coral areas create similar problems.

3.4. Marshallese Dose/Effects Results

The dose/effects basis of risk calculations is characterized and discussed in Appendix F, Section F.2. Some of the input data for the Marshallese situation have been presented in Section 3.2 above, where it has been noted that Conard, et al. (BNL-50220, 1970) and others had inadvertently used 1175 rad as the average thyroid dose for the children, rather than the 1000 rad calculated by James (1964). In the following discussion, incidence or risk-per-rad numbers, as calculated and reported by Conard, et al. will be used, to avoid confusion in comparisons with the literature; calculations for additional Marshallese populations will be done on the same (1175-rad to Rongelap children) basis. Strictly speaking, however, children's risk-per-rad numbers thus presented in this Section should be raised by about 20%, even though much larger errors are inherent in the Marshallese thyroid dosimetry and in published risk-per-rad data for other types of ionizing-radiation exposure.

Conard, et al. calculate (BNL-50220, 1970) Marshallese-pathology incidence on the basis of surviving population at the time of the last medical survey; also, they do not count individuals who had been in utero at the time of exposure in 1954. On March 1, 1954 the total population exposed on Rongelap itself was 64, plus 3 in utero. Of the 64, 19 were under 10 years old; all of them, plus the 3 in utero, survived to the time of the last reported medical survey (1969). Of the 45 people who had been over 10 years old in 1954, 11 had died in the intervening years, none from thyroid-related causes. The thyroid pathology to date for the most heavily exposed group (Rongelap itself) (as of the Spring, 1971 medical survey there were no new developments for any of the Marshallese) is shown in the following table, which is a modified form of Conard, et al.'s. Table 14.

RONGELAP GROUP			
Age at Exposure	Thyroid Dose, Rad	Nodules/Survivors (All Kinds)	Malignancies/Survivors
0 - 10 yrs	1175 (1000+175)	17/19 (89.5%)*	1/19 (5.3%)
> 10 yrs	335 (160+175)**	3/34 (8.8%)	2/34 (5.9%)
All	635 (Wtd. avg.)	20/53 (37.6%)	3/53 (5.7%)

The exposed Ailinginae group consisted of 18 individuals, plus 1 in utero. Of the 18, 6 were under 19 years old in 1954; all of them, plus the 1 in utero, are still alive. Of the 12 people who had been over 10 years old in 1954, 4 died in the intervening years, none from thyroid-related causes (although two had developed thyroid pathology: one a carcinoma, and the other adenomatous lesions discovered only at autopsy). The thyroid-pathology table for the Ailinginae group follows:

* As counted, nodules include the two cases of thyroid atrophy, with accompanying hypothyroidism and stunting. None of the 3 in utero individuals developed thyroid problems. It would be interesting, in the light of the discussion in Appendix E (Section E.4) on fetal uptake, to know what the fetal ages of the 3 individuals were at the time of exposure. Dyer and Brill (1969) find that the thyroid dose in a 22-week fetus is 6 rad per μCi of I-131 administered to the mother. Thus a 22-week Marshallese fetus would have received about 1000 rad from all the radioiodines. (If the mother had taken up 20% of her intake, then the 11.2 μCi first-day burden (adult thyroid) noted earlier in this Section implied an intake of 56.2 μCi of I-131 alone. Thus the fetal thyroid dose from I-131 alone would have been about 340 rad; that from all the radioiodines of the order of 1000 rad, per James' (1964) factors).

** Children 10-20 yrs old at exposure probably had about 500 rad from radioiodines. There are no nodules in that group.

AILINGINAE GROUP			
Age at Exposure	Thyroid Dose, Rad*	Nodules/Survivors (All Kinds)	Malignancies/Survivors
0-10 yrs	464 (395+69)	0/6 (0.0%)	-
> 10 yrs	132 (63+69)	1/8 (12.5%)	-
All	274 (Wtd. avg.)	1/14 (7.1%)	-

The Utirik group, which received about 14 rad whole-body, consisted of 157 individuals at the time of exposure. In 1969, there were 127 still alive; of these, 99 were examined in the 1969 survey. Of the 40 children (0-10 yrs old in 1954) examined, none had thyroid nodules; of the 59 adults (>10 yrs old in 1954), 3 had thyroid nodules, one of the 3 cases being a carcinoma.**

From the Rongelap data Conard, et al. have concluded that the development of 3 malignant thyroid tumors in a population of 53, coupled with the high incidence of benign nodules, "makes the etiological role of radiation exposure increasingly probable." Failure of the Ailinginae children to develop any neoplasms is surprising in view of their presumed thyroid dose; however, Conard (personal communication, 1970) believes that the food and water supply on Ailinginae was better protected than that on Rongelap, so perhaps the Ailinginae thyroid doses were smaller than indicated in the table.

Conard, et al. state: "The incidence of thyroid nodularity in the exposed Marshallese is considerably higher than that reported by Pincus and Hempelmann in their studies of

* The Ailinginae thyroid dose from radioiodines was calculated by Conard on the basis of proportionality to that for the Rongelap group (in the ratio of the whole-body doses from penetrating radiation; the Ailinginae whole-body dose was 69 rad). There had been no urine measurements on the Ailinginae group.

** A recent follow-up survey on the Rongerik Americans (Howell, 1971 and 1972) showed no evidence of thyroid pathology in the 12 survivors found or in the histories of the 3 known to have died in the intervening years.

populations who had been exposed to therapeutic x-irradiation of the neck region at a young age. However, on a risk per rad basis, the incidence of 51 cases per 10^6 persons per rad per year for the Marshallese is quite comparable with 24 for one group and 64 for a second group calculated by Pincus and Hempelmann." (Further details on earlier risk data can be found in Appendix F, Section F.2; that section of this report places the Marshallese data in the context of that other risk-per-rad information also.) The present author has attempted to confirm Conard, et al.'s. risk calculations, using the reported nodule incidence, a pathology-development period of 15 years (1954-1969), and the thyroid doses given in the above tables. For the Rongelap survivors of all ages (53 individuals) this calculation gives 40 cases per 10^6 persons per rad per year. If one adds in the Ailinginae and the Utirik survivors of all ages, this calculated risk parameter becomes 37. It is only for the 19 surviving Rongelap children alone that one can reproduce the value of 51 by a straightforward calculation. Perhaps the straightforward linear calculation is not justified, in any event, when the actual incidence is close to 100%.

With respect to the risk of malignancy, Conard, et al. state: "Based on the present incidence of thyroid malignancy in the high exposure Rongelap group, the risk of this malignancy developing - per 10^6 persons per rad per year - is 3 cases for the children exposed at < 10 years of age, 10 cases for the older people, and 5.6 cases for the group as a whole. The risk in the Marshallese children is not inconsistent with that reported by others."* The present author calculates

* However, the higher risk for the adults is in the wrong direction, if one accepts the literature conclusions discussed in Appendix F, Section F.2. It may be that the Marshallese children have not yet reached the peak latency period for malignancy; on the other hand, the fact that all those with benign lesions are on a lifelong course of thyroid-hormone administration should help to protect them from further pathology.

3, 11.7, and 5.9, respectively, for the children, the older people, and the group as a whole.

3.5. Thyroid Nodularity in the Utah Children

Nuclear testing in Nevada in the early 1950's produced measurable levels of fallout in populated areas, particularly in southwestern Utah (Washington County) and in adjacent areas in southeastern Nevada (Lincoln County). Potential whole-body doses were, of course, much lower than those experienced in the Marshallese situation, and there was apparently little concern about ingestion of short-lived fission products like the radioiodines. Then, in 1963 the possibility of thyroid injury to infants and young children was raised in testimony before the Joint Committee on Atomic Energy; this triggered large-scale, comprehensive investigations, involving (1) the Bureau of Radiological Health of the U. S. Public Health Service and (2) the Utah Division of Health. The results of these investigations are reported by Weiss, Rallison, London and Thompson (1971); all findings presented in this subsection are from that paper.

It is generally agreed that, in a U. S. situation in which food production and consumption practices continue normally in the presence of low-level fallout from weapon testing, the major intake threat is from I-131 in milk; inhalation of radioiodine(s) is a negligibly small contributor to the total thyroid dose. Therefore, in the Utah situation, accurate measurement of I-131 levels in milk and of milk-consumption patterns is the key to accurate thyroid dosimetry. Unfortunately, no measurements of I-131 concentrations in milk in Utah were made until 1957. Thus, estimates of the children's thyroid doses are somewhat speculative, involving

assumptions in each step of the explosion/deposition/forage/milk-production/milk-consumption chain. Six separate estimates for the period 1952-1955 were:

- a. All Utah children - 5 to 50 rad;
50 rad average
- b. Southwestern Utah children - > 100 rad average;
120 rad average
- c. St. George, Utah (Shot Harry of May 1953) - 68 rad;
700 rad

The follow-up for thyroid nodularity involved several thousand children in Utah and Nevada (some found to have been living in non-fallout areas in the period of interest), and a roughly equivalent number of children in Graham County, Arizona, chosen as the control area. For each geographical area investigated, the sample was over 85% of the school-enrolled children. Examinations were comprehensive; elements of "blind" design minimized subjectivity in the examining and screening physicians. In addition to the two (effective) control populations, another form of control was provided by comparison of nodularity incidence in the fallout-area children with reported expected (normal) incidence of nodularity in U. S. children.

Weiss, et al. conclude, on the basis of their three-year cumulative study (1965-68) that thyroid nodularity was found with equal frequency among children "potentially exposed" (1.3% incidence) and those "not exposed" (1.4% incidence) to fallout radiation in Southwestern Utah; the incidence in both Utah (incl. Nevada) groups was, however higher than that in the Arizona control group (0.9% incidence). For the Washington County, Utah group alone (1000 children), the incidence rate in the 1965-66 study was 1.1%, which falls within the range of 0.4 - 1.7% for this age group that would be calculated

(per Hempelmann's estimates, quoted in Weiss, et al.) for "spontaneous" or nonradiation-induced nodularity. Based on Hempelmann's (1968) risk parameters of 38 - 52 cases per 10^6 persons per rad per year (penetrating radiation), and an assumed thyroid dose of 50 rad for the Washington County group, Weiss, et al. calculate an expected (excess) incidence of nodularity of 2.3 - 3.1%. Since I-131 dose is much less effective than x-radiation in producing nodularity (see Appendix F, Section F.3) one might more accurately calculate an expected incidence of only 0.2 - 0.3% from 50 rad in the Utah situation. This would be almost impossible to detect with statistical confidence for a population of 1000, particularly in the presence of a normal background* of the same or greater size.

While the study of Weiss, et al., has an important conclusion, namely "Within the limitations of the study....there is no evidence that children of Southwestern Utah and adjacent areas of Nevada near the test site have received enough radiation from radioiodine to produce significant thyroid disease.", the results add nothing to the input required for the present evaluation except, perhaps, to confirm qualitatively (1) the assumption that inhalation of the short-lived, higher-energy radioiodines contributes a very small part of the total thyroid dose in a U. S. environment; and (2) some of the risk concepts discussed earlier and in Appendix F.

3.6. Thyroid Pathology in Hiroshima/Nagasaki Survivors

There is an extensive literature, published by the Atomic Bomb Casualty Commission (ABCC), on the medical history of the Japanese atomic-bomb survivors. This literature includes many papers on thyroid problems; for example, Wood, et al. (1968).

* That is, spontaneous incidence.

Since the Hiroshima and Nagasaki detonations were air bursts, the radiation source was initial neutron/gamma (penetrating) radiation. Therefore, in terms of input for the present evaluation, the Japanese experience would, at best, confirm existing risk/rad data from medical use of x-rays. Doses to individual survivors, however, are not well characterized; they are usually reported as falling within broad ranges, since they have been reconstructed on the basis of reported distance from ground zero, shielding provided by building components, position of individual at exposure, etc., not all of which are known precisely. Wood, et al. report incidence of thyroid carcinoma as a function of age at exposure, sex, and distance from ground zero; they also have estimates of thyroid dose for those developing those carcinomas. While they find that the distance effect (dose) is statistically significant for females, the much smaller incidence for males provides too little data for a test of the effect. No estimate is made of risk/rad parameters. While it is known that the incidence of non-malignant thyroid abnormalities in the Japanese is about a factor-of-ten greater than that of thyroid carcinomas, the author has not been able to determine whether risk/rad estimates have been made for the larger group.

4-1

SECTION IV

POTENTIAL FOR INHALATION OF RADIOIODINE IN SHELTER

4.1. Introduction

If there is a radioiodine-inhalation problem in the civil-defense context, the source of the radioiodine vapor would have to be volatilization of radioiodine(s) from the fallout particles deposited outdoors, trapped in the conventional filters of shelter ventilation systems, or somehow entering the shelter proper.* One estimation approach is to start with particles trapped in the shelter ventilation system, since all radioiodine volatilized from such particles will be taken up by the air available to the shelter occupants. In one sense, the inhalation threat from that source might be thought of as a measure of the upper limit of the radioiodine threat from particles outside the shelter, since volatilized iodine outside the shelter would have a chance to disperse upward due to vertical turbulence in the atmosphere near the ground surface.**

* Arriving particles in high-level local-fallout fields are much too large to be inhaled themselves, or to pass through conventional air filters or long and angled ducting systems. Clark and Cobbin (1963) have shown that the minimum particle size in ground-deposited fallout is greater than 30 μm , for all contours out (down) to "standard" exposure rate of 1 R/hr at 1 hr., for all yields between 1 KT and 50 MT (100% fission). It follows that, even for people in the open, direct inhalation of fallout particles in fields of significant intensity is impossible, since particles larger than 10 μm cannot reach the lung (Kochendorfer and Ulberg, 1967). A second-order effect, namely impaction of larger particles in the nose and mouth, followed by swallowing, is possible for people in the open or in particle-contaminated enclosed spaces. However, in the shelter context, most particles larger than 30 μm would be rejected at the intake, stopped by conventional filters (Strope, 1959) or deposited in corners, recesses and plenums of duct systems.

** Note, however, that (as shown in Appendix D) the "outside" threat turns out to be orders of magnitude greater than that from trapped particles. This is because the relatively rapid fall of arriving particles permits only limited quantities to be swept into the intake.

The rate of arrival of fallout must be specified if one is to estimate uptake during the fallout period; in particular, estimation of the amount of radioiodine in particles entering a shelter ventilation system requires specification of the radioiodine volume concentration for the arriving fallout in the air at the ventilation intake (for example, in curies of total particle radioiodine per cubic foot of air), as a function of time. With a worst-case assumption that all particle sizes present are swept into the intake, one need then specify only a ventilation rate (for example in cfm, or in cfm per person) to determine ingress of particle radioiodine into the ventilation system, again on either a total or a per-person basis. Volatilization of radioiodines would then depend primarily on subsequent flow of intake air over the particles.

Appendix D is devoted largely to the development and application of the methodology used for estimation of the volume concentration of particle radioiodine in air at the shelter intake during fallout arrival. One combination of weapon yield and downwind shelter location (a worst-case situation) is then selected, and the ingress of particle radioiodine (per person) into the ventilation system is calculated.* For purposes of subsequent comparison with the threat from gaseous radioiodine originating outside the shelter, the particle iodine ingress is then expressed in terms of equivalent square feet of outside fallout deposit. Finally, on the basis of simplified one-dimensional turbulent-diffusion considerations, an estimate (again in terms of equivalent square feet of outside fallout deposit) of gaseous

* The implications of this ingress for human uptake and thyroid exposure dose, under various assumptions as to iodine volatilization rate, are discussed in the present Section.

radioiodine entry into the ventilation intake is made.*

The main areas of uncertainty in the inhalation-threat estimation are: (1) the radioiodine loading of arriving fallout particles, in terms of total activity, composition (with respect to mass chain), and location within the particles; and (2) the evaporation tendency of radioiodine species from arrival onward. Radioiodine loading is controlled primarily by fractionation during fallout formation and transport; it is discussed in Appendices B and C.** Evaporation tendency depends on radial distribution of precursors within fallout particles; oxidation state of resulting iodine; adsorptive capacity (chemical properties) of particle matrix; temperature; and air flow over the particles.

There is no body of information permitting one to predict evaporation rates, even if all these factors have been quantified. For one radioiodine species deposited (say as iodine) on the smooth surface of a chemically simple or inert matrix material, evaporation rates are not high. For example, recent experimental work (Norman and Winchell, 1970) on tracer I-131 surface-deposited on glass beads showed that the rate of evaporation of iodine from the surface of

* This is an upper-limit calculation, on the assumption of instantaneous volatilization of all the iodine at the moment of fallout cessation, for comparison with the threat from ingress-particle radioiodine. Implications of more-likely volatilization rates are considered in the present Section.

** For close-in fallout from a low-yield surface-burst (Freiling and Crocker, 1968), depletion of I-131 due to fractionation may be as much as 75% ($r_{131,95} = 0.25$) on a gross-sample basis (although some samples have been found to be enriched in I-131). For the same material, depletion in particles larger than 44 μm may range from 10% to 85%, while that in particles smaller than 44 μm may range from -50% (enrichment) to about 50%. On a gross-sample basis, Te-132/I-132 is depleted to about the same extent as I-131 (r number a factor of about 1.1 lower); the other iodines would be expected to be depleted more, the depletion increasing monotonically with mass number to well over 90%, on a gross-sample basis, for I-135.

such beads (room temperature, no air flow) is constant with time, averaging about 1% per week. This low a rate for surface-deposited iodine would imply that volatilization would not produce a significant inhalation threat in the civil-defense context, since in actual siliceous fallout particles, much of the iodine precursor activity would have penetrated beneath the particle surface during particle formation; after solidification, such particles would be relatively impermeable to diffusion to the surface at room temperature.

Additional work by Norman and Winchell (1970a) supports this contention. Over a 2-hr period, the extent of evaporation of I-131 volume-distributed throughout 1.0- μm TeO_2 particles was measured at several temperatures, the lowest being 110°C. Even at this elevated temperature, the loss "rate" was very low. Cumulative loss varied as $t^{1/2}$, so that loss rate was a decreasing function of time; this relationship indicated that diffusion in the solid was the controlling mechanism. A measured loss of about $7 \times 10^{-3}\%$ in the first two hours implied a loss of about 0.1% in the first week, and 1% per week thereafter.

On the other hand, it is known that iodine stability is quite sensitive to the chemical nature of the environment; losses, even from dry preparations, are well known occurrences to radiochemists.* In early work (Miller, et al., 1953) on the development of fallout simulants for ocean-surface bursts, in which contaminants laid down on test surfaces were allowed to "age" for varying lengths of time before decontamination, it was found that I-131, deposited in the iodide form in the

* Who, for example, had to be concerned about contamination of counting-chamber surfaces that had not come in contact with the sample itself.

presence of carrier iodide, evaporated from the dried sea-salt and hydrous-oxide deposit with a "half-life" of about one week (room temperature, no air flow), after correction for radiological decay. No other radioelement tested exhibited any measurable loss, much less 50% in one week.

While prediction of the volatilization rate of iodine in particles is not possible yet from first principles, one would hope at least to determine an order-of-magnitude estimate from nuclear-weapon-test measurements. Unfortunately, very few attempts have been made to measure directly the extent or rate of particle-iodine volatilization in weapon tests. The limited available data are presented and evaluated in Appendix B, in which the conclusion is reached that dry siliceous fallout particles release radioiodine to the air at a rate of about 0.00025% per day. The conclusion that such particles release radioiodine at a negligible rate is supported by some semi-quantitative measurements made at Operation PLUMBBOB at the Nevada Test Site in 1957 (Strope, 1959). In Shots Diablo and Shasta, ventilation air for a manned underground shelter was filtered by an M6 collective protector consisting of a conventional particle filter backed up by a charcoal filter. The system operated for the first 2 days on Diablo, and for the first 24 hours or so on Shasta, after which the filter components were returned to the Naval Radiological Defense Laboratory for analysis. Because the back-up charcoal filter for each shot was found to have no measurable radioactivity (stated to be at background level, and also as less than 1/1000 of the activity on the main filter), the reported conclusion was that the main filter was an absolute (particle) filter. However, the additional conclusion can be drawn that the radioiodine release rate from the trapped particles on the main filter was negligible, i.e., certainly less than 0.5% per day,^{*} and very probably much less than that figure.

* I-133 plus I-131 represent about 10% of the total fission-product exposure rate at about D+2.

Other reported attempts to establish the volatility of fallout radioiodine(s) are not helpful in the present context (extent of volatilization from particles of diameter of the order of 50 μ m or greater); either (1) the measurements were indirect, in that another loss mechanism could have been acting, or (2) the source material was not representative of the total fallout mixture at the location or, in general, of the particle-size distribution of interest in "local" fallout from surface bursts. Martin (1963) (Supplementary Reference List), for example, used several approaches at Project SEDAN. In the first, he collected plant samples at a series of times post-detonation at each of several stations, measured specific activity of I-131 in each sample, and plotted a curve thereof vs. time post-detonation, for each station. The effective half-life thus determined was only 5.5 d, rather than the 8.04 d half-life of I-131. Martin recognized that "the difference may have been due to the mechanical removal of particles from vegetation and/or to the vaporization of I¹³¹ from particles retained by vegetation". He conceded also that "perhaps a part of the I¹³¹ released from the Sedan crater was released in a volatile form and part was released in a non-volatile form". Since SEDAN was an (underground) cratering shot, it is possible that much of the volatile fraction of the radionuclide mixture remained in the low-level cloud, rather than being carried aloft as in true surface bursts (and thus lost to the locally deposited fallout from the surface-burst situation)*.

In Martin's second approach, he collected several plant samples on D+7, divided them into aliquots, and then over a 10 d period stored 2 aliquots of each sample under 6N NaOH while exposing another 2 aliquots of each sample to room-temperature air. Overall, there was an apparent loss of I-131 from the dry

* However, Lane's air-sampler measurements at SEDAN (see Appendix B) indicated insignificant quantities of volatile iodine (I-133) at his one (off-hot-line) station.

samples, though the statistics are not thoroughly convincing. The present author believes that the samples as collected were not representative. As fallout was depositing, the smaller particles would have been less likely to bounce off plant surfaces to the ground, and would have been retained preferentially by (hairy) leaf surfaces; also, originally volatile I-131 could have been adsorbed on plant surfaces. Six days of exposure to wind, sun and dew* could have further altered the form and location of the retained I-131. Thus the behavior of that I-131 in the subsequent laboratory setting would not have been representative of the volatilization potential from the larger fallout particles of interest in the present evaluation.

In his third approach, using additional plant material that had been collected on D+7, Martin set aside four bags of material on D+33. Then, at intervals of about 5 days, he removed 5 g subsamples from each bag and analyzed them for I-131. During the first 15 days of the experiment, samples were removed from the top of the material in the bags, without other disturbance of the contents. During that period, the apparent loss rate of I-131 was slightly higher than would be produced by the 8.04 d radiological half life alone. Later samples, each taken after thorough mixing of the contents of each bag, gave an apparent loss rate that was very close to the radioactive-decay rate. Martin recognized that this third approach gave no evidence of volatilization; he concluded that the apparent losses during the first part of the experiment were due to "the settling of fallout particles toward the bottoms

* In Appendix B, it is demonstrated that a large fraction of the radioiodine in even the larger particles of interest in the civil-defense context is susceptible to (preferential) leaching by water; in dissolved form the iodide is immediately available for oxidation and volatilization. The latter phenomenon is documented in, for example, Yuill, et al. (1970).

of the bags containing the ground plant material". In any event, for our purposes the material used in this third approach would be subject to the same reservations as that used in the second approach.

Direct measurements on the physical and chemical states of fallout radioiodine are exemplified by the work of Perkins (1963) (Supplementary Reference List), which was, however, concerned entirely with world-wide (delayed) fallout. As emphasized earlier, volatility of iodine in such fallout is not relevant in the context of the civil-defense problem, since worldwide fallout consists of: (1) predominantly sub-micron-sized particles produced directly in the fireball/condensation stage; (2) originally volatile radionuclides later associated with very fine atmospheric dust during cloud transport; and (3) volatile radionuclides scavenged by raindrops, rain being the principal mechanism for the return of worldwide fallout to the earth's surface. Thus, it is not surprising that Perkins found that the gaseous fraction of radioiodine in worldwide fallout varies from 10 to 90 percent.

Similarly, measurements of the physical and chemical states of radioiodine in air downwind from nuclear reactors and nuclear-fuel-processing plants are not applicable to the present evaluation, since the iodine is released as vapor or associated with very small particles (Holland, 1963 and Perkins, 1963) (both Supplementary Reference List). It is worthwhile to note that both Holland and Perkins quote Lane's earlier conclusion about the volatility of radioiodine from close-in SEDAN fallout, which has been demonstrated here in Appendix B to be unsupportable.

Evidence of apparent volatility of radioiodine in close-in fallout clouds that is based on direct measurements of radioiodine uptake by experimental animals exposed only in an

"inhalation" mode is suspect because of the existence of the impaction/swallowing mode for the larger particles that cannot be inhaled directly, and the observation (for example by Engel, et al., 1971) that the measured uptake of radioiodine by cows exposed directly to the airborne debris from venting underground nuclear explosions must be due at least partly to the known propensity of these animals to continually lick their muzzles.

In the documentation of the Marshallese experience in Section 3.3 above, the distinctive properties of coral-type fallout particles with respect to iodine volatilization were discussed in general terms. In particular, it was pointed out that when such particles fell into the shallow-water areas around the small atoll islands, the high (measured) water-solubility of iodine in the particles could lead ultimately to significant levels of radioiodine vapor over the islands. Norman (1972)* has recently started a series of measurements of the volatility of fission-recoil-loaded radioiodines in coral-type matrices. The first measurements were on a fused CaO matrix (particle-size range 105-149 μ m). Preliminary results indicate initial release rates of the order of (centering around) 1% per hour with dry or H₂O-saturated room-temperature air. There is a slight enhancement of release rate when the air is saturated with water vapor. These rates are orders of magnitude higher than those for glass or TeO₂ matrices (Norman and Winchell, 1970 and 1970a), quoted earlier in this Section. While the pure coral-type particle certainly does not represent the particle of primary concern in the civil-defense framework, the most-recent results are still of some interest, since calcium oxide and related compounds will be present in fallout from surface bursts: (1) on limestone-containing soils; and (2) in cities with high densities of concrete in streets and in structural slabs.

* Supplementary reference list.

4.2. Iodine Inhalation from Particles Trapped in Ventilation Systems

With respect to the trapped-particle threat, the phenomenological basis for the evaluation has been stated in Section 4.1 above and presented in detail in Appendix D, Section D.1 (supported by Appendix C); detailed discussion of the human-uptake- and thyroid-dosimetry-model inputs will be found in Appendix E. Further aspects of the human-intake problem will be considered later in this Section.

The evaluation is performed on the basis of an example problem that is very much a worst-case situation; it will be seen that the trapped-particle threat is minimal even under worst-case assumptions. However, the discussion of this Section provides a basis for the semi-quantitative evaluation of the threat of shelteree inhalation of radioiodine released in vapor form from fallout outside the shelter; that evaluation is presented in Section 4.3.

Specifications, assumptions and calculated phenomenological results for the example problem (Section D.1) are as follows:

Specifications

Weapon Yield:	10 MT, 50% fission
Effective Wind Speed:	15 MPH
Shelter Location:	58.5 mi downwind on the hot-line. This is the location of the highest exposure rate in the fallout field.
Shelter Ventilation Rate:	5 cfm per person.*

* The quantity of radioiodine inhaled (from trapped particles) is independent of the ventilation rate (first-order approximation), so long as the ventilation rate is the same during fallout arrival as it is during subsequent volatilization and inhalation of iodine (see Section D.1).

Assumptions*

Thermal-neutron fission of U-235.
 No induced activities (transuranics or soil products).
 No loss (in particular, of radioiodine) by fractionation; similarly, no fractionation with particle size.
 Transport/arrival model of Clark and Cobbin (1963).
 Decay law for air-ionization rate of total fallout mixture is $t^{-1.2}$.
 Radioiodine abundance, decay, and relationship (activity) to total R/hr as given by Kochendorfer (1969).
 All particle sizes present in fallout are swept into the shelter ventilation system, according to their volume concentration in the arriving fallout.
 Activity/particle-size distribution is log-normal.

Calculated Results

Standard Exposure Rate:	6,050. R/hr at 1 hr.**
Fallout-Particle Size Range at Shelter Location:	108.6 - 240.7 μm (1%-99% cumulative-activity range).
Fallout Interval:	2.25 h (t_a) - 5.50 h (t_c).
Exposure Rate (Hypothetical) (decay-corrected to t_a):	2,300 R/hr***
Total Ground Deposit of Radioiodine (Hypothetical) (decay-corrected to t_a):	1.61 Ci/ft ² ***
Average Particle Fall Rate During Arrival at Ground Surface:	3.9 ft/sec.
Trapped Radioiodine (on particles) per Person:	33.0 mCi (decay-corrected to t_a).****

* While these, and other assumptions to be discussed, may appear arbitrary, the author is convinced that the tremendous uncertainty as to the extent and rate of iodine volatilization and the wide variability in dispersion of iodine vapor, once formed from ground-deposited fallout particles, justify an eclectic approach to the assembling and application of input data.

** True air ionization rate under the hypothetical assumption that all the fallout is down by H+1.

*** Under the hypothetical assumption that all the fallout is down by t_a , which is the time of arrival of the first fallout.

**** 11.2 mCi, decay corrected to t_c , which is the time of fallout cessation.

The extension of the phenomenological results to human uptake and thyroid doses is based on the model and the calculations of Mechali, et al. (1966). Their basic uptake model for iodine is discussed here in Appendix E, Section E.5. Additional details of their application of the model to inhalation of the mixture of radioiodines follow.

The basic 3-compartment uptake/degradation/retention model shown in Figure E.1 (Appendix E) pertains to the behavior of stable iodine, starting with a quantity of newly introduced (fresh) stable iodine, in iodide form, uniformly dispersed throughout the bloodstream (Pool I, Figure E.1). All rate processes involved are taken as first-order. When inhalation is the mode of intake, the uptake model must be expanded to account for the extent and rate of initial introduction of iodide into the blood.

For computation of thyroid dose from a radioactive iodine isotope, the decay rate must be incorporated in the model, to take into account potential dose lost during thyroid uptake, and to quantify dose absorbed by the thyroid during the retention period. The first step in the dose computation, calculation of iodine-isotope activity in the thyroid as a function of time, requires incorporation of all the first-order processes, including radioactive decay, in the original differential equation(s) of the model. Then, for computation of the thyroid dose from inhalation of the mixture of radioiodines in fallout, one must repeat the above calculation for each iodine isotope, taking into account the relative abundance of each such isotope (in volatile form)* at the assumed time, post-detonation, of

* If an iodine precursor, such as tellurium, is assumed to be volatile, then its abundance also must be used as a starting point, and the calculation must take into account the rate at which its daughter iodine is produced by decay and then is taken up by the thyroid (tellurium being known to have a short residence time in the body and no preferential uptake by the thyroid). Inhalable (volatile or small-particle) tellurium may be a significant factor in reactor releases, but is not likely to be important in the civil-defense (fallout) context.

inhalation.

For their actual computations, Mechali, et al. simplify the 5-rate-process model of Figure E.1 to one involving only 3 rate constants (see discussion in Section E.5). They present a stepwise treatment of the development of the equations used to compute radioiodine activity in the thyroid and resultant dose, including both direct (iodine) and indirect (tellurium) intake as starting points. However, they do not give a complete mathematical derivation; rather, they indicate, at each stage, the basic assumptions used and the resulting equations for application.*

The context for the calculations of Mechali, et al. is a criticality accident in a Pu-239-fueled reactor. Fission is treated as instantaneous. Inhalation of the vapor and the small particles in the resulting cloud is considered to be non-selective; that is, the inhaled mixture of fission products is unfractionated. The discussion in Appendix C in this report (See also Table C.1) shows that, in the civil-defense (fallout) context, calculations of thyroid dose based on the radioiodine products of the fission of Pu-239 (as opposed to U-235 and/or U-238) introduce errors that are insignificant in the light of the overwhelming uncertainties regarding volatilization rates and extents. In the present work, the calculated thyroid doses of Mechali, et al. were used directly (modified for the conditions of the example problem). The basis for the modifi-

* A more complete mathematical treatment, including a detailed derivation for the tellurium-intake problem, can be found in Stewart and Simpson (1965). That treatment also shows calculation of intermediate results for each radioiodine of interest, including: (1) fraction reaching thyroid; (2) effective elimination constant; and (3) fraction of total iodine activity in cloud (reactor release) as a function of time. Adams and Bonnell (1962) examine the same 3-compartment biological mechanism in even more detail, using 5 biological linear rate processes, and also evaluating the implications of some possibly non-linear rate processes.

cation, and the implications of the assumptions of the Mechali, et al. treatment, are discussed in the following paragraphs.

Mechali, et al. calculate (ultimate) thyroid doses resulting from the instantaneous inhalation of 1 mCi of fission products (total mixture) at each of a series of times post-fission ranging from 5 minutes to 6 months.* The calculated doses are for the 20 g adult thyroid, under the assumption of total absorption of radioiodine beta-ray energy and zero absorption of gamma-ray energy within the thyroid. This dosimetry is entirely consistent with the discussion and the equations of Section E.6 (Appendix E) of this report. Thus Mechali et al's. "rem" are equivalent to "rad", as used here. The overall calculation starts with the tabulation of independent yields of the radioiodines and their individual precursors in mass chains 130 through 136.** Then, the activity (μCi) of each inhaled radioiodine at time t is calculated and tabulated, the basis being the inhalation of 1 mCi of fission products at that time. Inhalation-intake assumptions are:

25% of the "inhaled" intake reaches and is processed by the lungs, with zero residence time there and zero loss of that component between lung and bloodstream.

50% of the "inhaled" intake deposits in the upper respiratory path, passing secondarily into the digestive tract. Residence time in the bronchi is about 2 hr (linear rate process). The subject is assumed to be fasting, so there is zero residence time in the stomach. Iodine is absorbed from the intestinal tract in about 20 min; for convenience, the residence time here is taken to be zero, with little error.

25% of the "inhaled" intake is immediately exhaled.

* Clearly, the dose is a function of a complex mixture of variables, starting with the proportion of each iodine isotope in the total fission-product mixture at the time, t , of inhalation. It turns out that on the (arbitrary) basis of 1 mCi FP inhaled at time t , the calculated thyroid dose peaks for a \underline{t} of 14 days (approx.). This time has no particular significance on a threat basis, however.

** Mass chains 130 and 136 are ignored in the present evaluation, for reasons given in Appendix C.

All tellurium precursors are considered to have a radiological half-life of zero; that is, all radionuclides of interest either arrive as Te or I or are rapidly converted to Te. If this assumption is reasonable in the reactor-release context, it is even more so in the fallout situation under consideration. Mechali, et al. present an additional series of assumptions about the course of tellurium through the system, with implications for thyroid dose from the appropriate iodine daughters; these will not be discussed here, because in the fallout context (high-yield weapons) only Te-132 is important, and the resulting I-132 produces a maximum (for inhalation at 3-4 days) of only about 20% of the total thyroid dose. The significance of the mass-132 chain will be discussed further later in this Section.

The principal results of Mechali, et al. are presented in their Table 4 (not reproduced here in its entirety^{*}). Tabulated therein are the ultimate adult-thyroid doses from the total mixture of radioiodines for each of a series of times (in the range 5 m to 180 d) of instantaneous inhalation of 1 mCi of the total-fission-product mixture.^{**} Since the basis of the calculation is 1 mCi of fission products as of the time of inhalation, and since the ratio of total radioiodine activity to total fission-product activity (as well as the makeup of the radioiodine activity itself) is changing with time, the tabulated thyroid doses per FP mCi change rapidly with time of inhalation, rising from only 0.3 rem for inhalation at 5 min to a peak of 151. rem for inhalation at 14 d, and then dropping more slowly to 0 rem for inhalation at 6 mo. As mentioned earlier, this variability has no significance in terms of

* **Appropriate extracts are given later in Table 4.3 below.**

** Table 4 of Mechali, et al. contains also a breakdown, for each time of inhalation, of the percent contribution of each iodine isotope to the total thyroid dose. This will be discussed further later in this Section.

relative threat: 1 mCi of total fission products 6 months post-detonation represents a much larger fraction of the total weapon output than does the same measure 5 minutes post-detonation. The point is that the doses of Nechali, et al. can be converted directly to doses for the example problem of interest simply by multiplication by the appropriate factors based on decay rates for total-fission-product activity and for total-radioiodine activity.

The pertinent data, based on the unfractionated products of the thermal-neutron fission of U-235, are shown in Table 4.1.

t, hr.	CI F.P. (total)*	CI Iodine (total)**
2.25 (t_a)	1.55×10^8	2.5×10^7
4.0***	8.5×10^7	1.35 "
5.5 (t_c)	6.1 "	8.5×10^6
12.	2.8 "	4.0 "
24.	1.3 "	2.0 "
36.	7.1×10^6	1.3 "
48.	5.4 "	9.0×10^5
72.	3.4 "	5.2 "
96.	2.4 "	3.0 "
120.	1.9 "	2.8 "
144.	1.55 "	2.1 "
168.	1.35 "	1.7 "

- * Figure 2, Kochendorfer (1969).
- ** Figure 1, Kochendorfer (1969).
- *** Midpoint of arrival interval (approx.)

As indicated in Appendix C, the error introduced by the use of U-235 thermal-neutron fission (to modify doses calculated for Pu-239 fission) is insignificant in the operational context.

The calculations of Appendix D for the example problem (see Subsection D.1. (XIX)) led to a figure of 33.0 mCi (corrected to t_a) for the total trapped radioiodine (on particles) per person. Thus, by applying appropriate ratios in the iodine column of Table 4.1, we can calculate the actual levels of total trapped radioiodine per person for all other times. Then, by applying the F.P./Iodine (row) ratios of Table 4.1 to these actual radioiodine activities, we can calculate the actual levels of total trapped fission products per person, for all times of interest. The results are given in Table 4.2.

t, hr.	mCi F.P. (total)	mCi Iodine (total)
2.25 (t_a)	205. (hypothetical)**	33.0 (hypothetical)**
4.0***	112. (hypothetical)**	17.8 (hypothetical)**
5.5 (t_c)	81.	11.2
12.	37.	5.3
24.	17.	2.7
36.	9.4	1.70
48.	7.2	1.19
72.	4.5	0.69
96.	3.2	0.40
120.	2.5	0.37
144.	2.1	0.28
168.	1.80	0.23

* Shelter ventilation rate during fallout arrival (trapping) = 5 cfm/person.

** Actually zero at t_a , and a little over half the indicated value at the midpoint of the arrival interval.

*** Midpoint of arrival interval (approx.).

The tabulated (some are interpolated) thyroid doses of Mechali, et al. (their Table 4) from inhalation of 1 mCi of fission products at the times of interest (those given in our Tables 4.1 and 4.2 above) are shown in Table 4.3 following.

Table 4.3*	
Ultimate Thyroid Doses Following Inhalation of 1 mCi of Fission Products at Specified Times Post-Fission	
t, hr.	Thyroid Dose, rem
4.0**	19.5***
5.5 (t _c)	23.***
12.	37.
24.	57.
36.	73.***
48.	86.
72.	107.
96.	122.
120.	133.***
144.	141.***
168.	147.

The final step in the computation of thyroid doses for the example problem is the normalization of the dose values of Table 4.3 (for 1 mCi F.P.) to the actual available F.P. mCi based on Table 4.2. This produces the entries in the "Adult Thyroid Dose" column of Table 4.4 following.****

* Extracted from Table 4 of Mechali, et al. (1966).

** Midpoint of fallout arrival period for example problem.

*** Interpolated Value.

**** The dose entries reflect the assumption of a 0.5 cfm breathing rate, with a 5.0 cfm/person shelter-ventilation rate. Thus, "available" mCi (F.P. or iodine) are only one-tenth the values in Table 4.2. Note again that the final dose figures are independent of the assumed shelter ventilation rate, so long as it is the same during fallout arrival as it is during subsequent inhalation of volatilized iodine.

Table 4.4

**INHALATION OF IODINE FROM DUCT-TRAPPED PARTICLES.
ADULT THYROID DOSES AND RADIOISOTOPE CONTRIBUTORS**

Yield 10 MT, 50% Fission
Downwind Distance 58.5 miles

Time t (hr)	Adult Thyroid Dose* (rad)	% Contribution to Dose				
		I-131 (8 d.)	I-132 (78. \rightarrow 2.3 hr)	I-133 (20.8 hr)	I-134 (53 min.)	I-135 (6.7 hr)
~4.0**	~110.**	19.	11.	47.	2.	21.
5.5	185.	21.	12.	49.	0.3	18.
12	137.	25.	14.	49.	-	12.
24	97.	34.	17.	44.	-	5.
36	68.	41.	19.	37.	-	3.
48	62.	48.	21.	30.	-	1.
72	48.	60.	22.	18.	-	-
96	39.	68.	22.	10.	-	-
120	33.	72.	21.	7.	-	-
144	29.5	76.	19.	5.	-	-
168	26.5	81.	18.	1.	-	-

Assumptions: (1) No fractionation.

(2) Inhalation of Te-132.

(3) Instantaneous release of 100% of duct-trapped iodine at time t (i.e. none before, none after).

* Multiply adult dose by 3.3 to obtain infant-thyroid doses.

** Midpoint of fallout arrival interval. Dose (entry) computed for $1/2$ the total ultimately trapped iodine. Total dose then given by $110 + 185/2 \sim 200$ rad.

Note that Mechali et al's. "rem" are equivalent to "rad", as used here. The last five columns of the Table show the percent contributions of the individual radioiodines to the total dose at each time (adding to 100% in each row). These figures are taken directly from Mechali et al's. Table 4, except for interpolation at $t = 4.0, 5.5, 36., 120.$ and 144 hr, as in Table 4.3 above.

Each entry in the "Adult Thyroid Dose" column of Table 4.4 signifies the (ultimate) dose accrued, given that there was instantaneous volatilization of all trapped radioiodine at that specified time. The entry for $t = 4$ hr reflects that fact that only about half the fallout has arrived by the mid-point of the fallout-arrival interval (and it is assumed to volatilize instantaneously at that time). If the other half is assumed to volatilize at fallout cessation ($t_c = 5.5$ hr), then the total dose is about 200 rad, as indicated in the second footnote of the Table. Young children have about 1/3 the respiration rate of adults, but 1/10 the thyroid size (mass). This combination yields the noted factor of 3.3 for conversion of adult-thyroid dose to infant-thyroid dose.*

The assumption of instantaneous release (volatilization) creates a worst-case picture, especially for the early times (first entries in the Table). For continuous releases, adult doses can be estimated by an intuitive approach. For example, if the half-life for volatilization is, say, 1 week (release of ca. 10% per day)** then the half that is volatilized after $t = 168$ hr will contribute something significantly less than 26.5/2 rad to the total, and the half that is volatilized

* Grossman (1970) uses a factor of 3.0, based on the following values:

Adult-thyroid mass = 20g; infant-thyroid mass = 2g
 Adult breathing rate = 20m³/day (ca 0.5 cfm);
 Infant breathing rate = 6m³/day.

** The highest literature estimates of volatilization rate (shown in Appendix B and earlier in this Section to be unportable) give volatilization half-lives of the order of one week.

before $t = 168$ hr will contribute something like, say $62/2$ rad.* Thus the adult-thyroid dose would be something under 40 rad, the child-thyroid dose something under 135 rad. There is every reason to believe (Appendix B) that iodine volatilization from the trapped fallout particles of interest will be very much (orders of magnitude) slower than has been assumed in this approximate calculation. If the volatilization rate were truly only 0.00025% per day, then the infant-thyroid dose in the example problem would drop to less than 0.005 rad.

It is clear from the above and from the discussion following that the trapped-particle threat is minimal even under worst-case assumptions. It has been demonstrated (for fallout from a single megaton-range surface burst) that the maximum possible child-thyroid dose from fallout particles trapped in a shelter ventilation system is less than 135 rad (about 1/8 that experienced by the Rongelap children) and that even this figure is unlikely to be approached in any conceivable situation.** The other worst-case aspects of the situation as evaluated (besides the volatilization situation) are:

a. The shelter is located at the hottest point in the fallout field from a 10 MT 50%-fission surface burst, where the standard exposure rate is 6,050 R/hr at 1 hr. At

* Taking the 48-hr entry as an average value, to reflect the rapid dropoff in the dose entries with time, plus the exponential nature of the volatilization (implicit in the half-life treatment).

** The author is aware of only one phenomenon (other than fallout from more than one burst) that can act to raise the calculated dose. Clark, Nichols and Cartan (1970) have measured the permeation of I-131-tagged iodine (originating in vapor form) through human epidermis in vitro, and have calculated (based on highly variable experimental data) that the resulting thyroid dose, for the uncovered human body, is about 50% of that from inhalation in the same vapor environment. The potential 50% increase in calculated thyroid dose is insignificant in the light of the arguments following.

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such a location, even a shelter P.F. of 40 will permit a whole-body dose of about 150 rad, which brings that shelter population close to the early-sickness range, and within a factor-of-four of the dose for 50% early lethality. Thus, the planner is not likely to be concerned with the ultimate threat from inhaled radioiodine at locations where: (1) with P.F. 40 the standard exposure rate is much above the 6,000 R/hr-at-1-hr level (from overlapping fallout patterns from multiple bursts);* or (2) the P.F. is much below 40 in the presence of the 6,000 R/hr-at-1-hr level.

b. The treatment of Appendix D assumes 100% efficiency for trapping of all particle sizes, according to their concentration per unit volume in the arriving fallout. The particle sizes of the example problem (100 - 250 μm) should be severely discriminated against by standard ventilation-system intakes, particularly mushroom and gooseneck types, in which intake is in the upward direction.

c. The Mechali, et al. treatment assumes that, in addition to iodine, the tellurium isotopes are inhaled.** For the fallout-arrival times of interest in the example problem, only the mass-132 chain is overestimated significantly by this assumption (see chain parameters in Appendix C). If only the iodine is available in vapor form, thyroid doses should be somewhat less than 80-90% of those shown in Table 4.4; this overestimate is apparently insignificant in the face of the other overestimates. However, see paragraph e below.

* As pointed out in Appendix D (Section D.2.), the relative importance of the trapped-particle threat (that is, relative to the threat from outside sources of radioiodine vapor) is enhanced at greater distances downwind along the hot-line. In other words, on a per-R/hr-at-1-hr basis the mCi of trapped radioiodine are greater at greater distances downwind. Thus, it is possible, in the overlapping patterns from several bursts, to have a thyroid dose higher than that noted above (i.e., higher level of trapped radioiodine) correspond to the same effective standard exposure of the order of 6,000 R/hr at 1 hr. However, it is not very likely that this will happen to a significant extent, since most of the overlapping is likely to occur from crosswind overlaying of offset parallel patterns, rather than from downwind overlaying of collinear patterns.

** In the context of the present evaluation, only iodine is assumed to be capable of volatilization from fallout particles.

d. The treatment of Appendix D and the calculations of Mechali, et al. assume no loss of the radioiodines or their precursors in fallout-particle formation; that is, no fractionation losses. As discussed in Subsection 4.1 above and in Appendix B, Section B.2.2., fractionation can lead to losses in the range 60-75% (I-131) to 90-98% (I-135)* in radioiodine loading of gross samples of particles from low-yield surface bursts; as pointed out in Appendix C, radioiodine depletion in fallout from high-yield surface bursts is probably even more severe. Thus, thyroid doses from the individual radioiodines have probably been overestimated by factors of more than 3 (I-131) to 20 (I-135). The implications of this depletion on total calculated thyroid dose depend on the assumptions about volatilization rate; for instantaneous volatilization at, say, 24 hr after burst, fractionation would reduce the dose by a factor of about 5; for slow volatilization, by a factor of about 3.

e. The Mechali, et al. treatment, as exemplified by the form of presentation of the dose data in their Table 4 (extracted in Table 4.4 here), assumes implicitly that the calculated doses from the different radioiodines are equally effective in producing damage to the thyroid. The work of Klassovskii and his colleagues, cited extensively in Appendix F, Section F.3, indicates strongly that I-131, because of the low average energies of its two principal beta rays, is only about one-tenth as efficient as the other radioiodines (themselves equivalent to x-rays) in producing thyroid damage. Since the dose/effect literature for thyroid damage, particularly that concerned with neoplasm formation, is based on x-ray dose, the logical way to sum the doses for the radioiodine mixture is to use the doses for I-132 through I-135 as calculated, and to add to their sum one-tenth of the calculated dose from I-131. Examination of Table 4.4 will show that this correction will have only a small effect (<20% reduction in effective dose) where the intake is very early (rapid volatilization or early ingestion, as at Rongelap), but will reduce the calculated total dose profoundly where the intake is late (slow volatilization or late ingestion, as in the CONUS civil-defense context). For example, with the data of Table 4.4 and the assumption of instantaneous volatilization at $t = 168$ hr, the calculated (effective) dose is reduced by a factor of about 4 even if one accepts the total intake of Te-132, and by a factor of about 10 if one rules out that intake, a more reasonable decision (paragraph c above).

* That is, retentions (as fractions of theoretical loading) in the range (approximate) $1/3$ (for I-131) to $1/20$ (for I-135).

In summary, it is difficult to see how there can be any situation in which volatile radioiodine from shelter-trapped fallout particles can produce a thyroid dose, even in infants, that requires consideration in civil-defense planning. Any one of a number of factors (limited volatilization rate; preferential production of early injury or lethality; discrimination against shelter intake of the pertinent particle sizes; fractionation losses of radioiodine (and precursors) during particle formation; and relative ineffectiveness of I-131 in producing thyroid damage) can reduce the maximum possible effective thyroid doses to (almost) inconsequential levels. Limited volatilization alone has an overwhelming effect. When all likely reductions are taken into account, the trapped-particle threat disappears.

4.3. Inhalation of Iodine Released by Particles Outside the Shelter

While it has been demonstrated that the trapped-particle threat is minimal, and most likely inconsequential, it has been indicated (in Subsection 4.1, and demonstrated in Appendix D, Section D.3) that the "outside" threat (the threat of inhalation by shelter inhabitants of radioiodine released in vapor form from fallout particles outside the shelter) turns out to be orders of magnitude greater, and therefore bears examination. Also, it has been stated (Subsection 4.1) that the calculation of the trapped-particle threat provides a basis for the semi-quantitative evaluation of the "outside" threat. It is the purpose of this Subsection to review the common ground between the two types of threat, to summarize the method of extending the calculational model to the "outside" threat, and to examine (starting with the results and

conclusions of the foregoing Subsection) the nature and magnitude of that threat in several potential situations.

The basis for comparison is the equivalent ground-deposition area "sampled" per person. In Appendix D, Section D.2, the trapped-particle radioiodine (per person) of the example problem is shown to be equivalent to that deposited on only 0.02 ft^2 (ca. $19. \text{ cm}^2$) of (outside) ground surface.* This fairly inefficient ventilation-system sampling of particles is the result of the relatively rapid average terminal fall rate of fallout particles past the shelter intake (3.9 ft/sec, or 2.7 mph). For purposes of comparison, let us suppose that volatilization from outside particles is instantaneous at the time of cessation of fallout.** (this assumption corresponds to the trapped-particle entry for $t = 5.5 \text{ hr}$ in Table 4.4 above). Then, in a reasonably quiescent air atmosphere outside the shelter, the iodine vapor produced from ground-deposited fallout will be much more efficiently sampled by the ventilation system than were the particles during fallout. That is, outside iodine vapor, once produced, will not be moving upward past the intake at a net average "velocity" as high as 3.9 ft/sec (2.7 mph), except under extremely gusty conditions. If it were conceptually possible to estimate an average net upward "velocity" for ground-level-generated

* Trapped-particle radioiodine activity (decay-corrected to fallout-arrival time) was 33. mCi/person; ground-deposited radioiodine activity, A (same basis), was 1.61 Ci/ft^2 (ca. 1.7 mCi/cm^2).

** Admittedly, this is very unlikely, especially for dry fallout, as discussed in Appendix B and earlier in this Section. Nevertheless, as long as volatilization rates, if appreciable enough to be of concern, are the same for trapped- and ground-deposited-particle radioiodine, the following argument should hold.

radioiodine vapor under normal atmospheric turbulent-diffusion conditions, then the potential ingress of outside vapor (on an equivalent-deposit-area basis) could be measured by that "velocity". In particular, under such circumstances the importance of the outside-vapor threat relative to that posed by trapped particles would simply be the ratio of the average terminal velocity during fallout to that (smaller) upward "velocity". However, net "velocity" is not a common measure in turbulent-diffusion theory (it is used here only to illustrate the potential magnitude of the "outside" threat). A more realistic way to measure the equivalent-deposit-area for ingress of outside vapor is presented in Appendix D, Section D.3, and reviewed in the following paragraphs.

With instantaneous volatilization of iodine from ground-deposited particles assumed, the resulting radioiodine vapor is allowed to (turbulently) diffuse upward past the shelter-ventilator intake, according to a simple partial-differential one-dimensional-diffusion equation (Equation D.1, Appendix D). A highly idealized situation is hypothesized. The ground deposit of particle radioiodine is assumed to be an infinite uniform field, so that (1) the effect of surface winds after deposition and volatilization is merely* to replace that vapor which moves downwind with an equivalent quantity from upwind sources; and (2) the effect of lateral turbulent diffusion is similarly nullified. The basic partial differential equation is solved in the traditional way, on the assumption of separability of variables, leading to a general (one-term) solution, which turns out to require a cosine distribution of initial concentration, $R(0,z)$, vs. altitude, z , over the initial

* Except for the creation of upward turbulent diffusion of iodine vapor, measured by a vertical turbulent-diffusion coefficient, to be discussed.

distribution space, prior to diffusion dilution,* and a similar cosine distribution (of exponentially diminishing amplitude) over the same space as time goes on (Equation D.4, Appendix D), with all concentrations reaching zero at infinite time (except where the effects of temperature inversions are being evaluated).

The total activity (C_i) of radioiodine (decay-corrected to time of fallout arrival, as for trapped-particle radioiodine) in the initial-distribution column of cross-section 1 cm^2 is simply A , the ground-deposited radioiodine activity, in (decay-corrected) C_i/cm^2 . This provides the initial conditions for the radioiodine activity concentrations in the initial distribution space, already denoted $R(0, z)$, in (decay-corrected) C_i/cm^3 . With the ventilation-system intake located at some point in the initial-distribution space (usually at ground level, for convenience and for worst-case estimating), and with a ventilation rate, G , of 5 cfm (ca. $2200. \text{ cm}^3/\text{sec}$)** per person, as used in the trapped-particle-threat evaluation, one now can compute Q , the total (decay-corrected) radioiodine vapor activity sampled to infinite time (or to 5 diffusion half-lives, in practical terms) by direct integration of $R(t, z)$ *** from $t = 0$ to $t = \infty$.

* The peak is at the ground surface; the concentration drops to zero at the top of the initial distribution space.

** The author apologizes for the inconsistency in units. He has tried to use, in each input area of the calculations, the units generally accepted in that area, and to indicate both metric and English values for the important results.

*** Usually $R(t, 0)$.

In the sequence of calculations, the deposited radioiodine activity, A (Ci/cm^2), is left in symbolic form, so that Q is expressed directly in equivalent ground-deposition area, and can thus be compared directly with the Q for trapped-particle radioiodine. From the point of view of inhalation, the direct comparison is valid as long as the shelter ventilation rate is the same (say 5 cfm per person) for both radioiodine sources; the inhalation rate (0.5 cfm per adult, ca. 0.15-0.17 cfm per infant) must of course be applied in calculation of the ultimate thyroid uptake and dose.

The turbulent-diffusion computation and all the results to be discussed are based on the assumption of instantaneous volatilization of radioiodine from ground-deposited fallout particles, as emphasized earlier. Intuitive (approximate) extension of these results to the slow-volatilization situation (analogous to the (intuitive) extension discussed for the trapped-particle threat in Subsection 4.2) is not straightforward, since in effect two exponential decays (volatilization and turbulent diffusion) are occurring, the first providing a distributed source for the second. Thus, for example, if volatilization is very slow compared to upward turbulent diffusion, upward dissipation in the atmosphere will drastically reduce the radioiodine activity "sampled" by the ventilation intake. In addition, any factor, such as slow volatilization, that lengthens the effective time over which turbulent diffusion operates will void the bases for assumption of an infinite horizontal source, required for the one-dimensional diffusion treatment.

Sample calculations of the effect of upward turbulent diffusion are made (Appendix D) for two situations: (1) upward diffusion in a normal atmosphere, with no lid to the rise of radioiodine vapor; and (2) upward diffusion during a

temperature inversion, when there is a lid to net upward motion, trapping the radioiodine vapor for periods of the order of days, and thus providing a continuous source of radioiodine vapor for the shelter ventilator intake; the latter is very much a worst-case situation.

4.3.1. The Normal Atmosphere, with No Lid to Vapor Rise

All the calculations for the first situation are based on a value of the vertical turbulent-diffusion coefficient, K_z , that represents approximately the "normal" atmosphere, the most-stable and therefore the least-dispersing situation existing in the absence of inversions (i.e., a worst-case condition for the non-inversion situation). For such an atmosphere, Richardson's earlier definitions (quoted in Meteorology and Atomic Energy (Slade) (1968)) imply a K_z of $800 \text{ cm}^2/\text{sec}^*$ (at the appropriate scale of turbulence for these calculations).

While Q , the shelter intake of radioiodine activity per person, is the major variable of interest, it is important to know also the half-life for upward diffusion for each situation examined, since the latter determines (after instantaneous volatilization) the effective time (ca 5 diffusion half-lives) for ultimate upward dissipation of all the radioiodine vapor in the vicinity of the shelter ventilation intake.

* The most-stable situation considered by Pasquill (quoted in the same source) is a night-time condition, with less than 3/8 cloudiness and a wind speed of the order of 2m(eter)/sec (ca 4.5 mph) or less. Pasquill's definitions imply a K_z of $530 \text{ cm}^2/\text{sec}$ for this situation. It turns out that Q is²inversely proportional to K_z , everything else being equal, so use of Pasquill's number would raise the quoted Q 's by about 50%. It should be noted that Pasquill's K_z (for this so-called Condition "F") refers to what he calls a "moderately stable" situation; this is more turbulent than a true (stagnant-air) inversion.

Typical calculational results (see Appendix D, Section D.3) for the normal-atmosphere (non-inversion) situation follow ($K_2 = 800 \text{ cm}^2/\text{sec}$, $G = 5 \text{ cfm}$ ($2200 \text{ cm}^3/\text{sec}$) per person, sampling at ground surface, simple cosine distribution; unless otherwise specified):

Case I. Initial distribution space (height), z_i
10 ft (1007 cm)

Results:

Diffusion half-life: 35 sec

Shelter radiiodine-vapor intake, O_i
550A, in Ci/person (decay-corrected to
fallout-arrival time)

That is, the shelter ventilation system takes in, per person, outside radiiodine vapor equivalent to the total (decay-corrected) radiiodine in 550 cm^2 of ground deposit; i.e., in ca. 0.6 ft^2 of ground deposit. This is 30 times the (per-person) trapped-particle radiiodine noted earlier in Subsection 4.3 (0.02 ft^2 equivalent) for the first example problem.

Case II. Initial distribution space: As in Case I
Sampling at 5 ft height, rather than at
ground surface.

Results:

Diffusion half-life: As in Case I

Shelter intake, O_i : 400A Ci/person

Case III. Initial distribution space, a : 30 ft (300 π cm)

Results:

Diffusion half-life: 5.3 min (315 sec)*

Shelter intake, Q : 1650A Ci/person**

Case IV. Initial distribution space: As in Case I
Diffusion coefficient, K_z : 530 cm²/sec
(per Pasquill)

Results:

Diffusion half-life: 53 sec***

Shelter intake, Q : 815A Ci/person****

* Per Equation D.6, the half-life is proportional to a^2 , everything else being equal.

** Per Equation D.7, the intake, Q , is proportional to a , everything else being equal. What this means is that, with everything else equal, the shelter intake of radioiodine vapor (for a given total ground deposit of fallout, per unit area) is greater the flatter the initial vertical distribution of vapor after the instantaneous volatilization. Thus, by assuming a fairly extensive initial mixing (even to 10 ft (Case I)), we may be overestimating the threat from intake of outside vapor. The crux of the matter is how close to the point of volatilization the turbulent-diffusion phenomena take over.

*** Per Equation D.6, the half-life is inversely proportional to K_z , everything else being equal.

**** Per Equation D.7, the intake, Q , is inversely proportional to K_z , everything else being equal.

Case V. Initial distribution space: As in Case I
 Initial distribution: Cosine plus sine
 (more uniform)
 Sampling at 5 ft height (peak concentration)
 rather than at ground surface.

Results:

Diffusion half-life: 35 sec (as in Case I)
 (Eq. D.8)

Shelter intake, Q: 400A Ci/person
 (Eq. D.9) (as in Case II)

For the turbulent-diffusion situations thus far considered, which represent the ordinarily most dangerous situations (outside of temperature inversions) from the point of view of shelter intake of radioiodine vapor in infinite fields, the threat (with Case III excluded for reasons there given) appears to be a factor of perhaps 10 to 30 times that from trapped particles (in the fallout and shelter situation of the example problem). However, even the factor-of-30 increase does not appear to change the conclusions of Sub-section 4.2 above, particularly when the major pertinent mitigating factors there cited (low volatilization rate from dry fallout particles; radioiodine depletion in arriving fallout, due to fractionation during formation; and diminished effectiveness of I-131 dose to thyroid in producing pathology) are taken into account. For example, if the volatilization rate of iodine from dry fallout particles is truly only 0.00025% per day, the best available figure, then the factor-of-30 increase would change the less-than-0.005 rad infant thyroid dose in the trapped-particle situation (based on data of Table 4.4) to the order of much less than 0.15 rad, still an inconsequential dose. If the volatilization rate were of the order of 10% per day (volatilization half-life ca. 1 week),

then an upper limit for the ultimate infant-thyroid dose from outside radioiodine vapor would be 30 times the figure of (less than) 135 rad calculated in Subsection 4.2 above, or (less than) about 4000 rad, mostly from the less effective I-131.* Fractionation depletion would reduce this to the order of about 1000 rad (again mostly from relatively ineffective I-131, so that the effective dose would be something over 100 rad). More importantly, with this slow a volatilization, the bases for the turbulent-diffusion calculation will collapse, as discussed earlier in Subsection 4.3; the effect is to reduce the calculated thyroid doses very drastically, due to extremely rapid vertical and horizontal dissipation of the vapor as produced, relative to the volatilization rate. The reader should recall also that the doses given are for the worst possible location in the fallout pattern.

The outside-vapor threat can be enhanced if fallout particles deposit in nearby bodies of water or even in puddles, or if rain occurs after deposition. As demonstrated in Appendix B, a large fraction of the radioiodine in even the larger siliceous particles of interest is susceptible to (preferential) leaching by water; in dissolved form the iodide is immediately available for oxidation and volatilization. The latter phenomenon is documented in, for example, Yuill, et al. (1970).** Leach rates from purely siliceous particles are slow (half-lives of the order of days); thus, the conclusions in the preceding paragraph with respect to slow volatilization (1-week half-life) are not likely to change very much, even if siliceous particles should enter water bodies. However, if

* While there is absolutely no justification for assuming that instantaneous volatilization at t_c (fallout-cessation time) is possible, the calculated appropriate (unmitigated) infant-thyroid dose of 18,000 rad ($185 \times 3.3 \times 30$) is noted here for the sake of completeness.

** In the fallout situation, where the radioiodine will be essentially carrier-free (except for background levels of natural (stable) iodine), the driving force for volatilization may be lower than in the reported experiments, because the solubility (as iodine) will not be exceeded.

the fallout particles contain calcium oxide and related compounds, as a result of surface bursts: (1) on limestone-containing soils; and (2) in cities with high densities of concrete in streets and in structural slabs; then the more-rapid solubility of iodine from such particles could lead to significant doses from ingress of outside radioiodine vapor in the stable atmospheres considered in this Subsection. The problem can become even more important in temperature inversions, as discussed in the next Subsection.

In the normal-atmosphere context, calculational results should not be pushed too hard, because of uncertainties as to vertical turbulent-diffusion coefficients, effects of nearby structures on vapor dissipation, diurnal variability, etc., as discussed in Appendix D, Subsection D.3.1 and elsewhere in this report.

4.3.2. Temperature Inversions

The calculational basis for the evaluation of ingress of outside radioiodine vapor in a temperature-inversion situation is presented in Appendix D, Subsection D.3.2: Three time regimes are required: the first, as in the normal-atmosphere situation, is the exponential dropoff within the shallow initial-distribution layer, essentially complete after about 5 diffusion half-lives. At the end of this first regime, the iodine concentration in air at the shelter ventilation intake is not zero, however, but rather the average concentration with the total iodine distributed uniformly throughout the (thicker) inversion layer.* The sample calculation is based

* That is, $A \text{ Ci}$ (from the ground-deposition density, $A \text{ Ci/ft}^2$) are distributed uniformly through a column, of cross section 1 ft^2 , whose length is the thickness of the inversion layer. Note again that instantaneous volatilization has been assumed, for purposes of comparison.

on a 10-ft-thick initial distribution, as before; the inversion layer is taken to be 500 ft thick.

The second regime, lasting for the life of the inversion (taken to be 24 hr in the sample calculation), is one in which the iodine concentration in the sampled air is constant, at the average (uniform) figure existing at the end of the first regime. The third regime starts with the dissipation of the inversion; it is treated as a straightforward upward turbulent diffusion like that of the first regime, except that the starting average concentration is that of the second regime (adjusted for a cosine-plus-sine representation of the starting uniform distribution), and the starting-layer thickness is that of the inversion itself. All other independent variables are as in the basic calculation of the normal-atmosphere situation, except that the K_z for the first regime is taken to be a factor-of-five lower than that for the "normal" situation considered earlier, to reflect the fact that turbulence at ground level will be somewhat less during the inversion than it is in a normal atmosphere. The results of the calculation are as follows:

<u>Regime I:</u>	Diffusion half-life:	165 sec
	Shelter intake, Q_I :	2750A Ci/person

Thus, during Regime I alone (because of the factor-of-five reduction in K_z) the shelter ventilation system takes in, per person, outside radioiodine vapor equivalent to the total (decay-corrected) radioiodine in 2750 cm² (3.0 ft²) of ground deposit. This is 150 times the (per-person) trapped-particle radioiodine noted early in Subsection 4.3 (0.02 ft² equivalent) for the first example problem. Note that the "infinity" (5 diffusion half-lives) for Regime I for this situation is about 15 min.

Regime II: The second regime lasts for the remaining 23.75 hours of the inversion duration. The shelter intake of radioiodine vapor is simply the product of the ventilation rate and the (constant) concentration existing at the end of Regime I. Because of the assumed uniformity of concentration during the inversion, the intake at ground level would be the same as that anywhere else in the inversion.

Shelter Intake, Q_{II} : 12,500A Ci/person

Thus, during the inversion the shelter ventilators take in (per person) outside radioiodine vapor equivalent to the total (decay-corrected) radioiodine in 12,500 cm² of ground deposit; i.e., in ca. 14 ft² of ground deposit. This intake is about 700 times the (per-person) trapped-particle radioiodine noted early in Subsection 4.3 (0.02 ft² equivalent) for the first example problem. The calculation shows that Regime II intake is almost 5 times that of Regime I if the inversion lasts as long as 24 hr. Intake in Regime II is, of course, proportional to the length (duration) of the inversion (and inversely proportional to the inversion height) so that for every 5.3 hrs that the inversion persists, there is an intake equivalent to that of Regime I.

Regime III: Diffusion half-life: 24 hr
Shelter intake, Q_{III} : 13,750A Ci/person

The large per-person intake (equivalent to the ground deposition on 13,750 cm² (ca. 15 ft²), or about 750 times the (per-person) trapped-particle radioiodine intake) is probably a gross over-estimate, for several reasons. A less-stable situation (stronger winds) causing the breakup of the inversion would imply a value of K_z higher than the 800 cm²/sec used, hence a shorter diffusion half-life and a lower intake during Regime III. The 24-hr diffusion half-life calculated implies an "infinite" dispersion time of about 5 days. Even if the atmosphere causing the inversion to break up were not very turbulent (whereas it is more likely to be gusty), this calculated dispersion time is so long that it is quite unreasonable to continue to invoke the assumption, basic to all these calculations, that horizontal dispersion is not operative (because of the

semi-infinite deposit field). Experience indicates that, once conditions leading to an inversion disappear, the breakup requires hours, rather than days (except, perhaps, in confined situations such as narrow valleys).

In summary, the presence of a temperature inversion during the volatilization of radioiodines from deposited fallout particles can markedly increase the shelter intake of radioiodine vapor. That intake is most sensitive (directly) to the duration of the inversion and (inversely) to the thickness of the inversion layer. However, because of the reduced turbulence there is a significant enhancement of intake even during the first stage of vertical dispersion, independent of inversion duration and thickness. There are limits to the effect of an extended inversion period, in that the finite limits of the fallout field lead to an enhancement of the effect of horizontal dispersion, thus dissipating the iodine vapor more rapidly, as time goes on. The effect is more drastic than a straightforward inverse- r^2 reduction of concentration, since the turbulent-diffusion coefficients for horizontal dispersion are about 4 times as large as those for vertical dispersion.

As calculated, the total (three-regime) shelter intake of radioiodine vapor, per person, during and immediately following a temperature inversion of duration 24 hr and thickness 500 ft is about 850 to 1550 times the (per-person) trapped-particle radioiodine noted early in Subsection 4.3 for the first example problem, depending on the importance attached to the third regime. For purposes of thyroid-dose calculation, let us accept half the calculated intake for Regime III, leading to an enhancement factor of, say, 1200. Then the infant-thyroid doses can be estimated as in Subsection 4.3.1., based again upon the values in Table 4.4.

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With a volatilization rate (iodine from dry fallout particles) of only 0.00025% per day, the best available figure, the factor-of-1200 increase would change the less-than-0.005 rad infant thyroid dose in the trapped-particle situation to the order of much less than 6 rad, which would still be a small fraction of the whole-body dose likely to be accrued in the example situation.

If the volatilization rate were of the order of 10% per day (volatilization half-life ca. 1 week), then an upper limit for the ultimate infant-thyroid dose from outside radioiodine vapor would be 1200 times the figure of (less than) 135 rad calculated in Sub-section 4.2 above, or (less than) about 160,000 rad, mostly from the less-effective I-131*. Obviously this upper limit is so high that even the mitigating factors discussed earlier cannot remove the threat. Fractionation depletion would reduce the upper limit to about 40,000 rad (again, mostly from relatively ineffective I-131), but at best this would still leave an effective dose of over 4,000 rad, which would destroy or at least seriously damage every thyroid so exposed. It is true that this slow a volatilization would collapse the bases for the turbulent-diffusion calculation, in that there would be extremely rapid vertical and horizontal dissipation of the vapor as produced, relative to the volatilization rate, so that the calculated thyroid doses should be drastically reduced. Thus, the above final effective dose of something over 4000 rad is very much an upper limit (with a one-day inversion). Nevertheless, the calculated doses are high enough so that the threat cannot be ignored, if one accepts the possibility of a volatilization rate implying a half-life of one week.**

* For the sake of completeness, we note that, while there is absolutely no justification for assuming that instantaneous volatilization at t_c (fallout-cessation time) is possible, the calculated appropriate (unmitigated) infant-thyroid dose is about 730,000 rad ($185 \times 3.3 \times 1200$).

** The calculated doses become more realistic in, for example: (1) confined situations, such as narrow valleys; or (2) situations where temperature inversions last longer than one day; or (3) situations where inversion layers are less than 500 ft thick.

As in the concluding remarks in Subsection 4.3.1., we note that, while an iodine volatilization half-life of the order of one week does not appear to be reasonable for dry siliceous particles, an effective volatilization rate of this magnitude is possible if fallout particles come in contact with bodies of water, and even faster ultimate release to the atmosphere may occur if particles thus leached contain calcium oxide and related compounds in significant percentages.

4.4. Summary

On the basis of the best available information, it appears that in the nuclear-attack context the only situation in which inhalation of radioiodine can create a threat that must be countered is one in which: (1) a long-lasting temperature inversion accompanies or immediately follows the arrival of high levels of local fallout; and (2) there are significantly large areas of shallow water nearby, in which fallout particles (particularly if they contain significant percentages of calcium oxide and related compounds, deriving from limestone or concrete at the burst point) can deposit (heavy rains accompanying or immediately following fallout arrival would enhance iodine volatilization; however they are not likely to be associated closely in time with temperature inversions).

Clearly, the important variables* in this specific kind of threat situation are almost impossible to parameterize for planning purposes; thus, it is not feasible to calculate, for a given assumed attack, the number of individuals who will be (ultimately) injured if not protected (at the time of fallout arrival) against the effects of intake of radioiodine. Nevertheless, the threat of such injury appears serious enough, and the cost of the appropriate countermeasure (administration of blocking iodide) low enough, so that it is worth planning such a countermeasure.

* These include: (1) particle composition (calcium content and fractionation depletion); (2) iodine volatilization rates from dry particles; (3) iodine leach rates into water; (4) effects of water depths on ultimate release rates; (5) local area covered by different ranges of water depth; (6) temperature-inversion probabilities, heights and durations; etc. The composite of these variables would have to be related, of course, to the assembly of variables used in standard evaluation of fallout consequences (fallout levels, shelter protection factors, deaths due to other effects of nuclear weapons, etc.).

SECTION V

BLOCKING AS A COUNTERMEASURE

5.1. Introduction

The basis for the use of blocking as a countermeasure to the uptake of radioiodine(s) by the thyroid is implicit in the mechanisms discussed in Appendix E. In this Section, "blocking" will refer specifically to the oral administration of stable (non-radioactive) iodine, in iodide form, immediately prior to or during an expected intake of radioiodines, or as soon thereafter as possible.*

* Any substance that interferes with the iodine-uptake and/or -conversion process in the thyroid can be considered a blocking agent. The so-called antithyroid drugs, such as: (1) thiourea and thiouracil and their derivatives, which inhibit the oxidation step in the organification of iodide in the thyroid follicles; and (2) KCNS and $KClO_4$, which inhibit the selective absorption of plasma iodide by the thyroid; fit this definition. So does thyroid hormone itself, whose continuing administration obviates the need for further hormone production by the thyroid, and thus reduces the uptake and conversion of inhaled or ingested iodine. All of these are eliminated from further consideration here because of one or more of the following considerations: (1) expense and/or limited availability; (2) uncertainty as to long-term storage stability; (3) possible danger in administration to large populations; (4) later operation in the uptake cycle than for iodide, leading to potentially higher ionizing-radiation dosage from retained radioiodine. In effect, there is no reason why other blocking agents should generally be preferable to iodide itself, and many reasons that argue against their use. Radioiodines firmly incorporated in the thyroid cannot be effectively removed by any of the inorganic or organic substances mentioned. However, thyroid-stimulating hormone (TSH), administered i.v., can accelerate the release of such radioiodines. It would have its major effectiveness only against longer-lived I-131. Its safety in long-term administration to large normal populations is untested.

Iodide as a blocking agent in principle operates mainly by means of a straightforward isotope-dilution mechanism. The experiments of Vought and London (1967) indicated that in adult humans there is an obligatory excretion of (at least) about 60 μg of (stable) iodine per day, independent of the iodine intake (this implies an obligatory intake of the same quantity, for balance)*; with intakes much higher than 60 $\mu\text{g}/\text{day}$ the fraction of intake excreted approached unity. Similarly, it is known (Pochin, 1965) that the output rate of thyroxine from the normal thyroid is constant, independent of the dietary input rate of iodine, within very wide limits. Thus, in the absence of continuing buildup of iodine levels in the thyroid, it is clear that the daily uptake of iodine by the gland is relatively constant, despite variations in the amount of circulating iodide. (However, on a one-time administration basis, it is possible that some fraction of an excess intake of stable iodide will be taken up by the thyroid). The results of Schneider (1964) and of Rosenberg, LaRoche and Ehlert (1966) indicated that iodine was processed by the thyroid on a "last come, first served" basis; that is, new iodine did not mix homogeneously with the old stores in the thyroid, and the freshest organified iodine left the thyroid earliest.

Thus, if an unavoidable one-time intake of radioactive iodine (essentially carrier-free) is immediately accompanied by administration of a massive (say order of hundreds of milligrams) dosage of non-radioactive iodide,** the dilution effect and the overloading of the thyroid-uptake mechanism will mean

* Wolff (1969) states that the normal thyroid releases about 65 μg of iodine daily and that, since renal clearance is about twice that of the thyroid, the required daily intake is about 200 μg . Others generally report the required intake at about 1/8 mg, or about 125 μg .

** It is to be emphasized that the accepted normal thyroid uptakes of the order of 20-40% of (ingested) intake are associated with the normal daily stable-iodine intakes of the order of tens to low hundreds of micrograms.

in principle that most of the total-iodine intake (and the same (high) fraction of the radioactive-iodine intake) will not be taken up by the thyroid, but will be retained in the circulation and rapidly excreted. Ionizing radiation doses to the blood and to other organs irradiated during circulation of the radioiodine are inconsequential.

If radioiodine is ingested or inhaled over a long period, then the blocking agent must be administered essentially over the whole period to provide complete protection. While smaller blocking-agent doses will probably provide good protection after the administration of a large initial dose, the continuing administration, even of the smaller doses, may lead to side effects in some individuals (see detailed discussion in Subsection 5.3 below). It is known that high doses of inorganic iodine compounds taken for extended periods will provoke iodism, chronic iodide poisoning, which is manifested by: increased respiratory-tract secretions; gastrointestinal symptoms such as vomiting and diarrhea; fever; skin lesions; mental disturbances; and occasional inflammatory reactions involving the salivary glands, pharynx, and larynx (the last being a magnification of the expectorant effect of iodide, commonly used in medical practice). Other possible effects include hypothyroidism, goiter, or even hyperthyroidism, in rare individuals. None of these conditions is believed to be irreparable. The consequences to the fetus of excess iodine intake in the mother represent a special case.

The blocking effect of stable iodide is an important consideration in the development and application of medical diagnostic techniques involving radioisotopes. Inadvertent intake of large quantities of stable iodide would, of course, compromise the results of thyroid-uptake (function) and thyroid-scanning procedures based on I-131. On the other hand, there are other kinds of function and scanning procedures making use of radio-

iodine in which thyroid uptake of radioiodine would be an undesired effect. For these, administration of thyroid-blocking dosages of stable iodide is a standard procedure.*

Thus, despite the known potential side effects from the administration of the quantities of iodide required for blocking, the inherent safety of the technique (at least for a one-time administration) is illustrated by its widespread use in medical diagnostic practice. Rodden, Suta and Weisbecker (1969, 1970) conducted a survey of the use of radionuclides in medicine, based on 1966 as the reporting year.** In that year, I-131-labeled albumin was used in lung scans on some 23,000 patients; iodide was used as a blocking agent in 83% of those procedures. I-125/I-131-labeled fats were used in fat-absorption tests on about 8,000 patients; iodide as a blocking agent was used on 67% of those tests. Similarly, I-125/I-131-labeled albumin was used in placenta scans on some 4,000 patients, with iodide as a blocking agent on 90% of the procedures. Finally, Tc-99m-labeled pertechnetate was used in brain scans on about 64,000 patients, with iodide used as a blocking agent (to screen unstudied parts of the brain from Tc uptake) in 65% of the tests. For each procedure, many patients received more than one test in the reporting year.

As a result of these and other indications of the general safety of the administration of blocking iodide, the procedure has been recommended by scientists and civil authorities for

* Also, for some procedures involving other radioelements, stable iodide is used in a blocking mode.

** The very widespread use of radionuclide procedures in general is illustrated by the 1966 figures for the most important of them: (1) thyroid function (uptake), with I-131-labeled NaI - 300,000 patients; (2) thyroid scan, also with I-131-labeled NaI - 154,000 patients.

use in the event of reactor accidents (such as a Windscale-type release) and in the nuclear-attack/civil-defense context. For example, Lengemann and Thompson (1963) (Supplementary Reference List) discuss the approach in their review article, stating:

"This procedure would have particular merit for individuals who may become exposed as a result of an occupational incident. Most of the efforts in this area have investigated the use of stable iodine as the thyroid blocking agent because it is usually well tolerated, large amounts do not materially affect renal clearance of I-131 and it can act as a diluent of I-131 in tissues that do not have an iodide-trapping mechanism."

United Kingdom planning against the iodine-inhalation threat in a nuclear-power-station accident is illustrated by Holbrook (1965):*

"..... In certain cases, it may be possible to postpone a decision on evacuation until the effects of administering potassium iodide tablets to members of the general public have been assessed.... The tablets are quite safe and have no side effects and their use has been approved by competent medical authorities. The decision to issue these tablets rests with the Emergency Controller and the police will be responsible for their issue to the local population. Stocks of these tablets will be held at the District Survey Laboratory, together with appropriate instructions regarding correct dose."

In the nuclear-explosion context, deGroot and Stanbury (1969) recommend the administration of potassium iodide, in a dosage

* See also Adams and Bonnell (1962), who provide a more detailed rationale, in the same context (U.K. accidents).

of 10mg/day for persons over 10 years (including pregnant women),* the prophylaxis to be instituted:

".....on indication of possible fallout contamination, and continued until environmental contamination has been judged safe by appropriate Civil Defense authorities."

They, too, cite the safety of the procedure for most of the population, while recognizing the potential dangers for some individuals:

"Present widespread usage of similar quantities of KI in cough syrups, asthmatic preparations, and anti-septics indicates it is rarely harmful, although it must be noted that no published experience with administration of this dose of KI to large population groups is available. Injections of iodinated oil (0.1 - 0.5 gm iodine) have been given to several thousand individuals for control of endemic goiter without adverse effects.Persons taking prophylactic KI should be advised to consult a physician if they develop thyroid enlargement, nervousness and weight loss, or other unexplained symptoms while on the medication."

The remainder of this Section is devoted to: (1) a review of the effectiveness of blocking, as related to the dosage of stable iodide (Subsection 5.2); (2) a more detailed examination of potential side effects of the administration of stable iodide (Subsection 5.3); (3) a brief discussion of methods of administration of iodide, with associated production and application logistics and estimated costs (Subsection 5.4); and (4) a summary of the blocking approach (Subsection 5.5).

* Recommended dosages for younger persons are: 5 mg/day for ages 1 - 10, and 2.5 mg/day for infants. Production would be as scored KI tablets of 5 mg mass, or as KI solution, 50 mg/ml (one drop = 2.5 mg).

5.2. Effectiveness of Blocking

Blocking effectiveness should be viewed in the perspective of the normal behavior of the thyroid with respect to the uptake of iodine. As discussed in Appendix E, the normal required daily intake of iodine is of the order of 1/8 mg, and about 20% of this intake is taken up by the thyroid of the average normal adult or child, this fractional uptake being essentially complete within 24 hr.*

A brief review of the literature on blocking through 1963 has been reported in Lengemann and Thompson (1963) (Supplementary Reference List). The important conclusions are summarized in this paragraph: Experiments on rats had shown that administration of a large amount of iodide as early as 12 hr before or as late as 3 hr after intake of radioiodine suppressed I-131 uptake to 1/3 or less of the normal uptake, the best results being obtained when the blocking agent was given 3 hr prior to the radioiodine. In tests on humans, simultaneous oral administration of 100 mg of stable iodide reduced the I-131 uptake to 10% of that of the control group;** if the administration of the blocking agent was delayed

* Uptakes in normal (euthyroid) adults have been reported to range from 6% to 45%, those for euthyroid children covering an equally wide, but higher, range, with some euthyroid children taking up substantially more than 50% of the intake. For most evaluations of the consequences of ingestion of radioiodine, the 20% figure is used for both children and adults.

** The 10% figure (based on the detailed measurements reported by Adams and Bonnell (1962)) was valid for both normal and hyperthyroid subjects. The figure applies to the uptake 24 hrs after I-131 ingestion (and later). When blocking iodide is administered simultaneously with I-131, thyroid content of I-131 (at least for the hyperthyroid subjects -- no histories were shown for the normal subjects) actually peaks at about 2-4 hrs, at a level perhaps 25-50% of that for the controls at the same time. Therefore, while the infinity dose from I-131 would be close to 10% of that for the control group in this situation, the doses from the shorter-lived radioiodines would be greater than 10% of those for the unprotected population.

for 4 hr, the relative uptake rose to 50%. If exposure of a large population to radioiodine is expected to be continuous over a long period, requiring inhibition of thyroid uptake for a long time, there may be some danger in prolonged administration of the high doses* recommended for the one-time intake. It would appear that the smallest dose possible is preferred. In a long-term study (Lengemann and Thompson are here quoting Saxena, et al. (1962)) it was found that a dose of 3-4 mg of iodide for adults and 1-2 mg for children was the minimal effective dose that could suppress thyroid uptake. At these levels, suppression was found to begin almost immediately after ingestion, with perhaps a 50% reduction in I-131 uptake by 24 hr. With continued intake of iodide, a gradual decrease in I-131 uptake occurred, until a minimal value of about 5% of intake (i.e., about a fourfold reduction in the normal (control) uptake of 20% of intake) was reached at 4-6 weeks. It was felt that these (low) levels of administered iodide could be sustained for long periods without ill effects.**

More detailed information on blocking can be found in the literature, particularly in publications since that of Lengemann and Thompson (1963). The points to be considered in this brief survey include: (1) relationship between blocking dosage and resulting uptake, with emphasis on minimum dosages

* For example, the dose recommended by Pochin and Barnaby (1962), which was 200 mg of iodine (as KI); those authors considered this dose safe for one-time administration, on the grounds that ".... it is of conventional size in normal medical use, comparable or larger doses commonly being given several times daily for long periods in expectorant cough medicines, and rarely causing toxic effects".

** Some implications of the results of Saxena, et al., will be discussed later in this Subsection.

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required; (2) duration of protection, possibly as function of blocking dosage; and (3) effects of mode of administration.

Pochin (1965) points out that most normal-uptake measurements are based on intake as iodide; results can probably be taken as valid for any soluble inorganic forms (or even simple organic forms), these being converted to iodide before passage through the thyroid or else accepted by the thyroid as received. This generality (interchangeability) applies to the concept of uptake half-life (that is, a half-life for removal of circulating "iodide" by the thyroid) of about 6-8 hr (see Appendix E for further discussion)*. Such a half-life implies that at least 90% of the ultimate thyroid burden from a single normal intake is accumulated within the first 24 hr.

One of the important results stated by Pochin is that a small increase in stable-iodide intake has a very small effect on uptake. For example, if the normal daily intake of $1/8$ mg** is trebled (and this new daily dose maintained), there is a delay of 1-2 weeks before a new, lower uptake percentage is attained. On the other hand, as Pochin states, careful measurements have shown that with a massive dosage of 200 mg of iodide*** uptake from a one-time intake of radioiodine stops (cumulative uptake stops rising) within 15 minutes of the administration of the blocking dose. Thus, even if the blocking procedure is delayed by 6-8 hr, there is still a saving of one-half

* Ng (James, 1964) used a half-life of about 5 hr in his calculations (See Appendix A, Subsection A.3).

** Iodized salt contains 0.01% KI, which is about 75% I⁻ by weight. If the normal intake of iodine comes entirely from iodized salt, one would have to consume ca. 1.7 g (ca. 0.06 oz.) of iodized salt daily to remain in balance.

*** The quantity found in about 6 lbs. of iodized salt.

the potential ionizing-radiation dose from I-131 (but less protection from the shorter-lived radioiodines). The best protection requires concurrent or advance administration of the blocking iodide. A large dosage (say 200 mg) of blocking iodide is effective for about 24 hr; that is, no resumption of uptake has been noticed in the first 24 hr after administration (the rebound is about 50% in 3 days). This behavior provides additional protection where the intake of radioiodine is not instantaneous, but rather occurs over, say, a 24 hr period. Longer-term delivery of radioiodine to the system would require repeated administration of the blocking agent. Finally, Pochin confirms the statement (See Subsection 5.1) that radioiodine already deposited (and "fixed") in the thyroid can be removed only by administration of thyroid-stimulating hormone (TSH) (he says intramuscularly), a procedure that he admits has appreciable potential hazards for a large population .

Cuddihy (1966) investigated thyroidal uptake of I-131 in seven "adolescents" (actually down to age 7) and four adults. Over a 14-day ingestion period (equal daily intakes of the I-131), the percent uptake, with no blocking agent administered (in fact, with the diet strictly controlled to omit all known major sources of iodine (iodized salt, sea food, iodine-containing medicine)), was identical for both groups of subjects, averaging about 23%*. The turnover rate was slightly higher in the "adolescent" group. A group of four individuals was continued on I-131 intake for another 14 days, with low levels of blocking agent administered daily. Two 8-9 year olds received 1.8 mg of I⁻ daily; their uptake dropped to an average of about 14% (about 60% of normal

* Cuddihy points out that European uptakes are generally closer to 50%. This is ascribed to the lesser quantities of iodide in the European diet (leading to a long-term adaptation by means of higher uptake).

uptake). Two adults aged 22 and 23 received an average of 4.1 mg of I^- daily; their uptake dropped to an average of about 9.4% (about 35% of normal uptake). Cuddihy extrapolated these results to imply that a pharmacological dosage of 10 mg of iodide would drop the uptake to about 4% (about 17% of normal uptake); this extrapolation depended partly on the quoted results (plotted by Cuddihy) of other authors*. Cuddihy states, "It appears that the maximum blocking effect can be obtained with about 10 mg of stable iodine. Any iodine in excess of this amount will probably not lead to any further substantial reduction in the percent of uptake." This statement, while reasonable in the face of the apparent leveling off of uptake between about 15 and 80 mg of blocking iodide (Cuddihy's plot of his own data plus that of Hamilton and the partial results of Adams and Bonnell), ignores the other (cited here) results of Adams and Bonnell (1962) which show only about a 10%-of-normal uptake with a blocking dosage of 100 mg of iodide, and also seems to sidestep the potential of the combination of: (1) isotope dilution; and (2) the limited capacity of the thyroid; for reducing the uptake, without limit, as the blocking dosage is increased.

Ramsden, et al. (1967) performed a very detailed blocking study on nine adult volunteers who had been on a low-iodide diet, such that their normal uptake was about 40%. Thyroid-uptake and urinary-excretion patterns were followed closely; measured values were compared with predictions of a four-compartment model, in which rate constants could be varied to simulate complex behavior, for example a change in the uptake

* J.G. Hamilton (Radiology 39, 541 (1942)) had measured an uptake of ca. 3.5% with a blocking dosage of ca. 15 mg; Adams and Bonnell (1962) had a point at ca. 3.0% uptake with a blocking dosage of ca. 75 mg.

constant, k_1 , as the thyroid becomes blocked. Two versions of the model were applied: (1) a straightforward dilution model, in which the uptake constant, k_1 , is a function of the daily dietary intake; and (2) the Adams and Bonnell (1962) model, in which k_1 is a function of the thyroid burden of iodine. In either version, k_1 drops as blocking is applied. It turned out that the latter model provided a better fit to the experimental data on the recovery (rebound) from blocking. Ramsden, et al. point out that under normal intake conditions, i.e., at equilibrium, the quantity of iodine transferred from the iodide space (plasma) to the thyroid is about 70 $\mu\text{g}/\text{day}$. At equilibrium, this figure must measure also, of course, the obligatory intake and the obligatory excretion, in agreement with the already cited figures of Vought and London (1967). Ramsden, et al. state further that the iodine capacity of the thyroid is quite limited, the normal burden of about 8.0 mg of iodine being raised to a maximum of only about 8.4 mg after achievement of the totally blocked state. During recovery from blocking, the transfer rate of iodine from the iodide space to the thyroid is fairly constant, ca. 3 $\mu\text{g}/\text{hr}$, or ca. 70 $\mu\text{g}/\text{day}$.

Ramsden, et al. considered one of the principal results of their investigation to be that the minimum amount of stable iodine needed to block thyroid uptake completely was about 30-40 mg. Supporting evidence was that, at this level of intake, thyroid uptake ceased within about 30 minutes of the administration of the blocking dose. This observation was based primarily on a series of measurements (Group B) in which a blocking dose of 100 mg of I^- was administered shortly (1.5-5.5 hr) after intake of radioiodine, and the resulting 30-hr uptake was compared with the known uptake (at the time of administration of the blocking agent) in the unblocked situation. From a radiation-dosimetric point of view, a more significant measure of required blocking dosage may come from another series of measurements (Group A), in which the blocking dose was administered 24 hr

prior to the radioiodine, and I-131 uptake was then followed for about 24-48 hr. The interesting measure in the Group-A series is the percent uptake of I-131 by the thyroid, as a function of blocking dosage. The volunteer who received a blocking dosage of 37 mg of I^- had a 30-hr uptake of 6%. With a blocking dosage of 124 mg of I^- , the 30-hr uptake was 2%. Finally, with a blocking dosage of 247 mg of I^- , the 30 hr uptake was recorded as 0%*. This implies that with simultaneous (or slightly delayed) administration of the blocking agent, a dosage of 200-250 mg would certainly drop the radioiodine uptake to (effectively) 0%, but a dosage of only 30-40 mg might not.

The conclusions and recommendations of Ramsden, et al. are as follows:

For a massive instantaneous intake of radioiodine, protection would be provided by a first blocking dose of 100 mg of iodide, given as soon as possible (no later than 6 hr after exposure). Where exposure can be anticipated, more complete protection is provided by administration of blocking iodide 1 hour before exposure. In this acute-exposure situation, the one blocking dose is sufficient to stop further uptake by the thyroid. However, if any uptake has occurred, recycling and reabsorption of radioiodine (particularly the longer-lived I-131) is possible. Reabsorption can be minimized by continuing administration of lower dosages of blocking agent, say 30 mg daily for 8 days.

For chronic exposure to radioiodine, continuing blocking doses are required. Regimes that can keep uptake below 1% of normal are:

- a. 250 mg I^- every 2 days; total 10.0 g over 80 days.
- b. 100 mg I^- every day; total 8.0 g over 80 days.
- c. 35 mg I^- every 12 hr; total 5.6 g over 80 days.

* The uptake for this normal subject peaked (at about 3%) at about 3 hr after radioiodine intake. This history is similar to that noted earlier for the hyperthyroid subjects in the investigations of Adams and Bonnell (1962). Whether the effect is real or an artifact due to undercorrection for the extrathyroidal neck radioactivity (circulating I-131) is not known.

For avoidance of possible side effects, the last regime is recommended, in that it keeps total iodide administered to the lowest reasonable figure.

Where the expected exposure is from consumption of contaminated milk, addition of one standard 300 mg KI tablet to each gallon of local milk would provide blocking for a population drinking about 1 pint of milk per day. Heavy use of iodized table salt would provide additional (or supplementary) protection, where water supplies (in the shelter context) were not limited.

Blum and Eisenbud (1967) investigated thyroid uptake in a large population of healthy adult volunteers (37 men and 25 women, all between the ages of 21 and 72). On the basis of 110 control determinations on the subjects, it was found that the normal 24-hr I-131 uptake (no stable iodide added to a presumably normal diet) was $27.1 \pm 8.9\%$ (std. dev.). Blocking studies were performed on 24 men and 17 women in the group, with dosages ranging from 5 to 1000 mg of iodide (dissolved in cherry syrup), administration occurring either one hour prior to, simultaneously with, or at specified times following administration of the radioiodine.

The best statistics in the Blum and Eisenbud investigation are those for simultaneous administration of blocking iodide and radioiodine. Thirteen subjects thus given 100 mg of I^- had a 24-hr uptake of $0.6 \pm 0.5\%$; i.e., a reduction of $98 \pm 3\%$. Ten subjects thus given 200 mg of I^- had a 24-hr uptake of $0.3 \pm 0.3\%$; i.e., a reduction of $99 \pm 4\%$. One subject thus given 1000 mg of I^- had a 24-hr uptake of 0.2%. The authors conclude from this that dosages higher than 200 mg are of only marginal utility, and not worth the risk of side effects.

Where blocking dosages of 100 mg or more were administered as late as 3 hr after the radioiodine (12 subjects), uptake reduction was still 60% or more; even for dosages of only 25-50 mg, administered up to 3 hr after the radioiodine, all but one of six subjects experienced uptake reductions of 60%

or greater.

The protection provided by administration of blocking iodide long in advance of the radioiodine intake is illustrated by some single-individual results of Blum and Eisenbud:

"Age" of Blocking Agent, hr	Blocking Dosage, mg	Uptake, %	
		Normal (Without Blocking)	With Blocking
48.	1000.	27.	0.
	100.	28.	6.
	50.	31.	10.
	25.	24.	24.
72.	1000.	28.	10.
	100.	22.	16.
	50.	35.	14.
	25.	24.	25.

Therefore, at the higher dosages, substantial protection continued for a few days after administration of blocking agent; thus continuing intake of radioiodine over a period of a few days is countered, at least in part.

These authors point out that for each of the cited results (all modes and times of blocking) the suppressive effect is better than the recorded reduction, because part of the body-counter reading of retained I-131 actually comes from extrathyroidal iodine in the neck region, not totally corrected for.

On the basis of all their measurements, Blum and Eisenbud conclude that the prophylactic administration of 100 to 200 mg of KI in anticipation of (or simultaneously with) exposure of adult's to radioiodines will largely prevent uptake by the thyroid, thereby reducing the ionizing-radiation dose to that gland

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(from I-131, in any event) by more than 98%. They conclude further that the same amounts given at intervals after I-131 intake are progressively less effective, but still reduce uptake to less than half normal after a delay of three hours. Daily readministration of the blocking agent is stated to be necessary for prolonged protection against continuing intake of radioiodine. They make no independent recommendations for the protection of children, relying on the already cited recommendations of Saxena, et al. (1962). Unfortunately, those recommended dosages (order of 2 mg per day over a prolonged period) provide substantially less protection than a 98% reduction, particularly at the start of the long-term prophylaxis.

Tanaka, et al. (1968) independently performed a series of experiments similar to those of Blum and Eisentud (adult human volunteers, 100 mg NaI, blocking prophylaxis at various times with relation to radioiodine intake) and came to almost identical conclusions. In addition, they found that 30 mg of methimazole was about as effective as 100 mg of NaI, when administered at about the same time as a one-time intake of I-131. Furthermore, they found that, where radioiodine was already fixed in the thyroid, therapeutic administration of 30 mg of methimazole (by mouth) daily could accelerate the discharge of I-131 from the thyroid to a considerable extent, while $KClO_4$, $KSCN$, or NaI seemed to be a great deal less effective. From a practical point of view, the oral administration of methimazole would seem to be preferable to the injection of TSH.

Some comments on the implications of the literature results on blocking effectiveness are now in order. First, in view of the tremendous uncertainty as to the extent of intake of volatile radioiodine, as discussed in Section IV, it is not possible to quantify the value, for a shelter population, of a given level of blocking effectiveness (expressed, say, as percent

of normal (unblocked) uptake). That is, it is likely (for reasonable values of volatilization rate) that, for most shelterees, intake of volatile radioiodine will be inconsequential in any event. At the other end of the spectrum of possibilities (namely, rapid volatilization (water-exposed calcium-oxide-containing fallout particles) in an extended temperature inversion), the potential intake, and the resulting thyroid doses from ionizing radiation, may be massive indeed. Therefore, it is impossible to specify a desired level of blocking effectiveness. Intuitively, however, one feels that a 50% reduction in uptake, for example, would not be very valuable. Thus, the already cited daily blocking doses of Saxena, et al. (1962) (1-2 mg of KI for children, 3-4 mg for adults), which may have some utility against long-term, low-level intake, but which provide minimal protection against a one-time massive intake of radioiodine, are inadequate in the volatilized-iodine context in the shelter situation. It is felt that, on the other hand, a reduction of 98-99% or more in the thyroidal uptake could provide significant protection for that part of the shelter population, if any, that is endangered by inhalation of radioiodine. This argues for a one-time administration, even for children, of at least 200 mg of iodide (subject to the reservations with respect to side effects, discussed in Subsection 5.3 following), and for a long-term administration, if necessary, according (at least) to the recommended schedule of Ramsden, et al. (35 mg I^- every 12 hr; total 5.6 g over 80 days) as noted earlier in this Subsection.

It is unfortunate that blocking-effectiveness data for children are so limited, since, as often emphasized elsewhere in this report, the young child is the most vulnerable member of the shelter population, in that the ionizing-radiation dose to his thyroid is so much higher than the adult's for the same radioiodine intake and percent uptake. This forces the recommendation that the child receive at least the same blocking dosage as the adult. Another special situation is that of the fetus

whose mother has inhaled the radioiodine mixture (see discussion in Appendix E, Section E.4). The 6-month normal fetus, for example, is known to be thyrotoxic, in the sense that the fetal-thyroid uptake of iodine is almost 100% of that in the fetal circulation, which is about 1% of the mother's intake. Since the fetal thyroid is extremely small, even this 1% uptake (with respect to the mother's intake) leads to a fetal-thyroid ionizing-radiation dose that is greater than the mother's thyroid dose (Tanaka, et al., 1968). Furthermore, ionizing-radiation damage to the fetal thyroid may be disproportionately high because of the rapid growth of the thyroid during this period. Unfortunately (see discussion in Subsection 5.3 following), stable iodide administered to protect the mother (if blocking dosages are high and continue over a long period) is likely to produce very serious side effects in the fetus in an unknown (but possibly small) percent of situations.

5.3. Side Effects

As pointed out in the introduction to this Section, it is known that high dosages of inorganic iodine compounds taken by adults or children for extended periods will provoke chronic iodide poisoning, known as "iodism", which is manifested by: increased respiratory-tract secretions; gastrointestinal symptoms such as vomiting and diarrhea; fever; skin lesions (urticaria or ioderma); mental disturbances; and occasional inflammatory reactions (sialoadenitis) involving the salivary glands, pharynx and larynx, these inflammations being an intensification of the expectorant effect of iodide, commonly used in medical practice and in non-prescription medications. Other possible effects include hypothyroidism, goiter, or (in rare individuals) even hyperthyroidism. None of these conditions is believed to be

irreparable. However, the consequences to the fetus of excess iodine intake in the mother represent a special situation which is extremely serious, sometimes leading to early death of the newborn infant.

Lengemann and Thompson (1963) review the side effects of intake of preparations containing iodine or iodine-containing compounds; they point out that there had been no hesitation (prior to 1963, at least) in the use of such preparations in relatively large quantities for diagnosis, treatment or the relief of symptoms in such medical situations as X-ray visualization, disinfection, syphilis, hypertension, bronchiectasis, asthma, and ordinary coughs. While it was known that increased protein-bound iodine (PBI) and low thyroid iodine uptakes may be produced by such intakes, persisting for days or even years, there had been relatively few untoward effects noted among large groups thus treated. Lengemann and Thompson state that an "allergic response" to iodine (presumably the iodism syndrome) may develop "in a few rare individuals" after use of small amounts of the element or its compounds, and note further that myxedema (the hypothyroid syndrome) and goiter had been shown to be induced by prolonged intakes, usually over periods of years, of hundreds of mg of iodide daily. One of the major characteristics of iodide goiter (the review of Wolff (1969) is summarized later in this Subsection) is that the symptoms disappear, and the individual returns to the euthyroid (normal) state, soon after cessation of excess-iodine intake. The 1963 review article states further that if large quantities of iodide are given to pregnant women, goiter can be produced in the fetus; if the goitrous thyroid of the infant is large enough the infant may not be able to breathe at birth. If the goitrous infant survives the initial difficulties in breathing (and other possible pathology) then, with cessation of excess-iodine intake, the goiter will usually regress and normal thyroid function will ensue. Another danger area is indicated by Lengemann and Thompson's

statement that long-term administration of iodide should be avoided for individuals suffering from chronic renal (kidney) and cardiac failure, and in cases of pulmonary tuberculosis. For the healthy individual, even a one-time dosage (if large enough) can lead to aftereffects that are at least unpleasant, even if the intake does no apparent permanent damage. For example, in a 1959 study quoted by Lengemann and Thompson, 1.3 g of KI was administered orally to each of 14 adults. Within about 12 to 48 hours, 10 of these people reported a mild to moderate sore throat. A few exhibited rhinitis (inflammation of the nasal mucous membranes), nausea, headache, acne, myalgias (muscle pains) and parotitis (inflammation of the parotid glands, as in mumps -- in fact, this symptom is called "iodide mumps"). 3 individuals independently reported experiencing a metallic taste; nearly all confirmed this effect upon questioning. All in all, most of the symptoms were mild and subsided within 48 hr. While it would be expected that few individuals would exhibit such symptoms with an iodide dosage of only 200-300 mg, the possibility of such symptoms in some people should be prepared for in the shelter situation. A final point of interest in the 1963 review article is the discussion of the possible side effects of the administration of other blocking agents. It is pointed out that, at least for long-term administration, iodide is probably the best tolerated of the inorganic thyroid depressants (somewhat safer than ClO_4^- , and much safer than SCN^- and NO_3^-). The organic anti-thyroid compounds in common clinical use (whose main desired effect in the blocking context would be the mobilization and excretion of radioiodine already fixed in the thyroid) can produce drug fever, dermatitis, and reduction in numbers of white corpuscles (leukopenia) or other elements (granulocytopenia) in the blood. Propylthiouracil produces such toxic reactions in about 9% of patients thus treated for thyrotoxicosis (hyperthyroidism); methimazole and carbimazole are probably somewhat less toxic.

Blum and Eisenbud (1967), in their study of 62 healthy adults, administered at least 100 mg of iodide (either 100, 200 or 1,000 mg) to 36 subjects. Only two subjects, both receiving 1,000 mg, reported side effects, namely uncomfortable sensations at the angles of the jaw and headache for several hours after ingestion; however, they were not incapacitated. Two other subjects receiving 1,000 mg reported no effects. These authors, who strongly recommend the administration of 200-mg capsules of KI in areas of high risk of exposure to massive amounts of radiiodines, stress that a single ingestion in the suggested dosage range is largely harmless. They note that even for long-term use (presumably at lower dosages) untoward reactions are uncommon, but cite a small risk of hypersensitivity, goiter, iodbasedow,* hypothyroidism and ioderma. Again in the long-term-blocking context, they suggest caution in the treatment of pregnant women, and advise against such treatment for people with renal disease, cardiac failure and pulmonary tuberculosis.

Pochin (1965) cites the following possible side effects of iodide administration:

1. Occasional skin rashes in the more-sensitive subjects.
2. Occasional suppression (sometimes total) of thyroid activity (i.e., hypothyroidism), with long-continued administration at higher levels.
3. Swelling of salivary glands, occurring commonly with doses of several grams of iodide (given at one time).
4. Possible increase in amounts of sputum. This is unlikely to be important in children and infants, as opposed to the elderly and ill.

* Thyrotoxicosis (hyperthyroidism), accompanied by large nodular goiters.

Adams and Bonnell (1962) contains a long discussion of the toxic effects of elemental iodine and of iodide, concluding first that in iodide form the element is rarely fatal; in fact, the reported fatal reactions in generally healthy individuals from direct ingestion of both forms are extremely rare, and are the result of either: (1) an overwhelming intoxication following large doses of I_2^* ; or (2) bullous ioderma occasionally occurring in patients taking iodides over long periods. Acute poisoning (never fatal) from a single dose of iodide is rare; even after intravenous injections, reactions are seldom seen. There are no reported cases of iodism following the ingestion of single small doses of iodide in the range (300-600 mg) recommended by Adams and Bonnell for blocking. They suggest that the reported cases of poisoning almost invariably result from repeated iodide administration, particularly for the treatment of hyperthyroidism and syphilis, or in patients suffering from advanced renal failure or cardiac failure. One study cited by Adams and Bonnell showed only 54 toxic reactions in 1100 patients receiving oral potassium iodide therapy, none of the cases being serious, and all symptoms subsiding on withdrawal of the drug. Even for: (1) patients suffering from chronic renal and cardiac failure (who are known

* Per Adams and Bonnell, few patients who attempt suicide with iodine are successful. For example, at Boston City Hospital between 1915 and 1936 there were 0 fatalities out of 327 treated, following such an attempt. Recorded deaths (New York City Medical Examiner's Office, for example) all involve local corrosive action of I_2 on the GI tract (esophagus downward) following ingestion of tincture of iodine. Adams and Bonnell state that since Strong Iodine Solution (USP) (Lugol's solution) contains 5% w/v I_2 and 10% w/v KI (in H_2O), even one ounce (the fatal dose in one case) contains 1.6 g I_2 and 3.2 g KI; this confirms the conviction that I_2 is more toxic than I^- . The present author notes that if the fatal dose was actually 1 oz of tincture of iodine, I_2 is even more dangerous than stated, since the standard tincture contains only 2% w/v I_2 and 2.4% w/v NaI in equal parts of ethanol and water. Thus, a fatal dose could have been only 0.64 g I_2 (and 0.77 g NaI).

to be hypersensitive to iodides and iodine, and in some of whom there have been fatal reactions following prolonged iodide therapy); and (2) tuberculous patients, in whom it is known that iodides cause irritative reactions and may even activate a dormant lesion; Adams and Bonnell suggest that small single doses of iodide would not lead to any untoward effects. These authors discuss also the other inorganic blocking agents and the organic antithyroid drugs, and conclude that, because of the potential danger of the antithyroid drugs, especially for children, for whom there was no experience in their use, "the administration of inorganic iodide to unselected groups of the general public is therefore preferred to the antithyroid drugs." One exception to this recommendation is the nuclear-accident situation, where reactor personnel, for example, could have been exposed to high concentrations of radioiodines. With treatment possibly coming after massive exposure, Adams and Bonnell recommend administration of the anti-thyroid drug carbimazole (neo-mercazole), under the supervision of the nursing staff on-site. Such treatment appears to be unnecessary in the nuclear-attack shelter context, if shelterees take iodide before or at the moment of arrival of fallout.

Sensitivity to iodine-containing drugs was studied by Raj and Dubey (1969), as part of a larger study on allergic reactions to commonly used antibiotics and other drugs and sera. Sensitivity was assessed by intradermal (injection) skin tests (patch tests). The test population was a representative sample of the national demographic structure (India), except for a low proportion of age group 51 and over. Of 292 individuals examined for sensitivity to (unspecified) iodine compounds, 32 (or 10.9%) exhibited detectable sensitivity to those compounds. Of these sensitive individuals, 26 (or 81.2%) could recall a previous history of receiving a similar kind of injection (i.e., of an iodine-containing compound). In fact, only one of the 32

sensitive people was certain that he had had no such history. Similar results (effect of previous history) were obtained for sensitivity to penicillin and to anti-tetanus serum. This suggests that if people are to be given iodide in the shelter context, special attention should be paid to those who report a history of iodide intake in any form,^{*} in response to carefully planned questioning by the shelter staff.

Wolff (1969) discusses in detail the so-called "iodide goiter", which has the following properties:

1. It is caused by chronic ingestion of iodide itself, or of iodide-generating organic compounds, in amounts about ten or more times the daily requirements for thyroid-hormone biosynthesis.
2. It occurs in only a fraction of subjects so exposed; predisposing factors are uncertain.
3. It occurs at all ages; in the fetus (placental intake from the mother) it may lead to fatal respiratory obstruction.
4. Goiter or myxedema (hypothyroidism) may exist independently; however, they frequently occur together. One exception is a form endemic in Japan, apparently due to seaweed ingestion in large quantities in certain communities,** where euthyroid goiter is the rule. Wolff uses the term "iodide goiter" to include also the other exception, those situations where only myxedema occurs in response to excess-iodide intake.
5. Upon iodide withdrawal, goiter shrinkage and full recovery of normal thyroid function occur. With reintroduction of excess-iodide intake, the iodide goiter returns.

* As well as to those otherwise expected to be sensitive to iodide intake, by virtue of known medical conditions.

** In fishing communities on Hokkaido, daily intakes of up to 200 mg iodide are likely during seaweed-consumption periods. The incidence of the resulting "coast goiter" is 6-12%. About 1000 such cases had been seen by 1965.

6. Even though the causes of iodide goiter (in particular, the predisposing factors in the affected individuals) are not completely known, this entity is better understood than any of the other side effects (effects on the respiratory tract and on the nature of bronchial secretions, induction of or influence on skin lesions (iodism), and jodbasedow).

7. The basic mechanism is the inhibition of organic iodine formation in the thyroid, the so-called Wolff-Chaikoff effect. The resulting imbalance in the normal thyroid/pituitary feedback system, starting with low levels of thyroid hormone in the blood stream, stimulates an attempt by the thyroid cells to grow and proliferate, in an attempt to produce enough hormone to restore the control mechanism to normal. This concept is supported by the evidence that administration of T_4 or T_3 (thyroid hormone) will cause the goiter to shrink, even with excess-iodide intake continuing.

8. Most patients with iodide goiter had received large amounts of iodine compounds (from 18 mg to more than 1 gm per day) for prolonged periods. Many had received such quantities for five years or more before the goiter appeared. There are exceptions: in some cases, the actual goiter appeared only two months after onset of the intake. Generally, myxedema follows within one to two months after the goiter is noticed.

With specific reference to effects of excess-iodide intake in children, Saxena, et al. cite extensive clinical experience indicating that "toxic effects of iodide are not observed with doses of 100 mg of iodide per day given to children over a course of years. Iodide goiter has been observed.....to occur only following daily doses of several hundred milligrams of iodide administered for years....."

We turn now to the problem of iodide goiter of the newborn. Wolff presents a tabulation of incidents of this condition, including duration of iodine intake by the mother, thyroid condition of the mother, symptoms in the infant, and outcome. His 25 tabulated cases result from a search of the literature covering the period 1940 to 1968 (there had been no reported cases of iodide goiter of the newborn prior to 1940). Important conclusions are as follows:

1. Iodide is readily transferred across the placenta; however, the mode of transport in man is not known. Whatever the original form of the iodine in the mother, her normal processes would convert much if not most of it to iodide, thus making it available to the fetus.

2. All the mothers involved in Wolff's summary had chronic lung disease; all but two received inorganic iodine, mostly as KI or unspecified iodides.

3. For most of the mothers, the duration of iodide intake had ranged from 2 to 10 years. However, in one case the iodide had been used for only the last 3 months of the pregnancy (intake as Lugol's solution). Also a case has been reported in an infant born 4 years after the mother received iophenoxic acid. A search of the literature (present author) shows that daily intakes of KI by the mothers had generally been of the order of 500-1000 mg or more. (See, for example, Hassan, et al. (1968).)

4. Iodide goiter in the newborn is similar to that in adults, except that tracheal obstruction (due to the goiter) is a frequent problem* (15 of 25 infants); it has proved fatal in 6 of 25 infants. Only immediate surgery can save the infant in serious cases. Controlled respiration and antibiotics are indicated in any event. In another review cited by Wolff it was reported that of 22 newborns with iodide goiter, 8 died of obstruction.

5. Goiter need not be present in the mother (it was found definitely in only 11 of the 25 mothers), and hypothyroidism in the mother is rare (only 2 definite cases).

6. Hypothyroidism has been reported in infants born with iodide goiter, but it is difficult to diagnose in the newborn, so reliable estimates of its incidence cannot be made.

7. In those infants who survive birth, the goiter gradually disappears (since the source of excess iodide has been removed**). However, iodide in the mother is readily secreted into the milk (even more so when there is excess intake), so the mother's iodide intake must be watched carefully during breast feeding.

* Respiratory obstruction has been reported on occasion in adults with iodide goiters, but not as a major problem.

** Sporadic or congenital goiter in the newborn (due to causes other than excess-iodide intake in the mother) requires therapeutic treatment, in addition to that for the obstruction, if any.

Hassan, et al. (1968), cited by Wolff, present detailed descriptions of the condition and treatment of three infants, delivered by mothers (euthyroid and goiter-free) who had been on iodide therapy for long periods. The three infants were born with goiters and hypothyroidism, stated to be definitely non-congenital and non-endemic. None required surgery; however, all were treated with desiccated thyroid (a source of thyroid hormone) for several months, leading to complete recovery (and cessation of therapy). These authors argue that iodide goiter in the newborn implies a pre-disposition to the condition, since it occurs in the offspring of only a minority of mothers exposed to iodides over long periods. In fact, while on the same medication that produced the neonatal iodide goiters, the three mothers cited by Hassan, et al. gave birth also to euthyroid and non-goitrous offspring. Hassan, et al. argue also that, despite the general feeling that remission of symptoms occurs spontaneously, administration of thyroid hormone speeds the process, and is therefore the treatment of choice until recovery occurs.

Carswell, et al. (1970) report an additional 8 cases (in a period of 14 years in Glasgow) of neonatal goiter and hypothyroidism associated with maternal ingestion of iodide, taken as an expectorant and/or cough suppressant for asthma or bronchitis. There were 4 deaths, 2 due to unrelated causes; also, 2 of the survivors are mentally retarded, presumably because of fetal hypothyroidism. Thyroid-replacement therapy was given (started immediately after birth) to 2 of the 4 survivors, including one of those who proved to be mentally

* One, at 56 hr age, from pneumococcal pneumonia; the other, at 216 hr age, with no cause of death found post mortem.

retarded (IQ 64 at 4 yr age). Dawson (1970) reports an additional such death, also in Glasgow. Carswell, et al. recommend that iodide-containing preparations not be used during pregnancy, and that they no longer be available without prescription. This recommendation is supported by other medical authorities (Lancet Editors, 1970).

5.4. Methods of Administration and Associated Production and Application Logistics

Before we consider the details of administration of blocking agents, it is worthwhile to summarize the dosage and scheduling requirements. As emphasized in the concluding remarks in Subsection 5.2 above, the levels of intake of radioiodine via inhalation (and the resulting ionizing-radiation doses to the thyroid) are so uncertain in the shelter context that blocking-agent administration is probably justifiable only if uptake reductions of 90% or greater are feasible. As discussed in Subsection 5.2, the (continuing) administration of iodide at levels of a few mg per day does not reduce thyroid uptake rapidly (and certainly not immediately) to the desired 10% of normal or less; in fact, even at steady state (after 4-6 weeks, perhaps) the reduction in uptake may be only about 75% (Saxena, et al., 1962. See also Cuddihy, 1966). Only dosages of 100 mg or more, taken at one time, provide the 90%-or-greater reduction in the 24-hr uptake.

It is one-time administration* that we should be most

* Or, to be more practical, 1-to-say-5-time administration, over a period of several days, to prevent recycling within the body, and to cope with inhalation intake over a few days.

concerned with, since:

(1) As discussed in Section 4 above, slow release of siliceous-particle radioiodine by volatilization is unlikely to create a real inhalation threat in shelter. The major threat of volatilization, if there is any, will come in the special situations discussed in Subsection 4.3.2, namely the more rapid volatilization occurring when calcium-oxide-containing particles fall into water bodies or standing water, or are leached by rain (or, perhaps, moistened by heavy dew); and

(2) The major threat, in any event, comes from the short-lived radioiodines, which exert most of their influence over the first few days.

Where there may be a continuing radioiodine-intake threat (for example, with fallout-producing explosions spread over time, or with unavoidable intake of contaminated water or food (especially, milk)), early intake of several doses of iodide at the 100-mg level, followed by continuing intake at the levels recommended by de Groot and Stanbury (1969)* would be a minimally acceptable (with respect to effectiveness) and generally safe approach.

A much more effective, but still quite safe, approach to the continuing radioiodine threat is a modification of the recommended approach of Ramsden, et al. (1967), namely: 200 mg**

* Individuals over 10 years old (including pregnant women): 10 mg/day; ages 1-10: 5 mg/day; infants: 2.5 mg/day. Production would be as scored KI tablets of 5 mg mass, or as KI solution, 50 mg/ml (one drop = 2.5 mg). The lower dosage levels recommended by Saxena, et al. (1962) (3-4 mg/day for adults, 1-2 mg/day for children) would probably not provide enough protection.

** Per Blum and Eisenbud (1967) this alone provides a 99% reduction in the 24-hr uptake, if the administration of the blocking agent occurs at the time of, or slightly before, massive instantaneous intake of radioiodine. Adams and Bonnell (1962) recommend 2 100-mg dosages, 4 hrs apart (adults); and 2 60-mg doses, 4 hrs apart (children under 4). Their suggested mode of administration is tablets taken by mouth, dissolved in milk or water. They point out that commonly prescribed dosages of KI for respiratory disorders are: 500 mg daily for adults, and 200 mg daily for children.

of iodide taken on warning of fallout arrival, or as soon thereafter as possible, and from the 2nd day onward, a dosage of 35 mg every 12 hr, for a total of about 5.6 g over 80 days (if protection for that long a period is required)*. This regime would keep uptake below 1% of normal, and would thus make sense in the context of the uncertainty as to intake, particularly from inhalation. Also, per Ramsden, et al., where the expected exposure is from consumption of contaminated milk, addition of one standard 300 mg KI tablet to each gallon of local milk (instead of the use of the individual 35 mg dosages) would provide blocking (99% protection) for a population drinking an average of about 1 pint of milk per day.

This suggested regime appears to be required for children as well as adults: in fact, it is more important to provide blocking protection for the children, because of their tenfold-higher ionizing radiation dose to the thyroid for the same intake of radioiodine. As suggested in Subsection 5.3 above, there is no reason to believe the regime is unsafe for children. The nursing infant should receive its blocking dose directly, since blocking in the mother reduces uptake of radioiodine by her thyroid but probably increases secretion of radioiodine in her milk.

It has been stated earlier in this Section that pregnant women taking substantial quantities of iodide (ca 500-1000 mg KI) daily over periods of the order of years for the relief

* For convenience in production, storage and administration, the continuing dosage might better be 100 mg every day, starting on the second day, for a total dosage of 8.1 g in 80 days. This is the second preference of Ramsden, et al., from the point of view of the total intake of iodide.

of symptoms in asthmatic and other respiratory disorders sometimes deliver infants with obstructing goiters and hypothyroidism (the iodide goiter syndrome) and that the resulting infant-fatality rate is extremely high. Only a minority of women on such (very high) dosages deliver such infants; in fact, the mother of such an infant often has delivered, or later delivers, several normal infants while on the same medication. There is no evidence that iodide intake by the pregnant woman at the levels recommended here (averaging only 100 mg/day for at most a few months) will produce iodide goiter in the fetus. Furthermore, since without administration of blocking agent to the mother the 6-month fetus, for example, would receive a thyroid ionizing-radiation dose greater than that of the unprotected mother, possibly with pathology disproportionately high for that ionizing-radiation dose (because of the rapid ongoing development of the fetal thyroid), it is judged that pregnant women should receive the same blocking doses as the rest of the population, particularly the initial 200 mg dosage. Physicians should be prepared for possible serious complications in some infants born after prolonged administration of blocking agent to pregnant women during the shelter period and subsequently.

The most convenient form of potassium iodide for production and storage (in tightly sealed containers) is as tablets; the following estimates of costs (Blake, 1971) are based on 200 mg tablets, which are the proper size for one-time protection against a massive instantaneous intake of radioiodine. For administration to infants and young children (or for those with difficulty in swallowing pills), the tablets can be easily dissolved, as needed, in a measured quantity of water, and an appropriate aliquot (fraction) administered in the diet.* If the

* The very-long-term storage stability of iodides in solution is an unknown quantity. Lugol's solution, $I_2 + KI$ in water, is no longer used as an expectorant in the United States; however, it had a reasonable shelf life when stored in tightly sealed brown glass bottles (under $38^\circ C$), as did tincture of iodine (an ethanolic solution of $I_2 + KI$). Aqueous solutions of iodide alone are stable under the same conditions. It is logistically simpler, however, to concentrate on the tablet form for the present application.

tablets are scored, they can be split at the proper time to provide the 100-mg dosages for continuing daily administration. A better approach is to produce only 100-mg tablets, with 2 taken by each shelterree for the initial dosage. The cost-estimate range given below is wide enough to cover both modes of production.

Per Dr. Blake, tableting would provide a storage-stable form of KI and would be a great deal less expensive than encapsulation. Proper storage would be important; i.e., in tightly sealed containers to exclude moisture. Dr. Blake indicated that there should be no difficulty in swallowing a KI tablet, and no associated unpleasant effects. He tried a KI tablet* and reported that it tasted salty, but not overpoweringly so, and had no after-taste. The experience was something like taking the conventional salt tablet. There appears to be no need to mask the taste of the iodide tablet by "coating" or "press coating", which would increase the cost significantly. In Dr. Blake's opinion, administration in milk or orange juice would be the best route for infants; again, it is suggested here that some such mode might be required for young children who cannot swallow pills.

Dr. Blake's Laboratory purchases pharmaceutical-grade "raw-material" KI powder in 25-lb containers at about \$3.50 per lb (October, 1971 prices). Thus, one can obtain about 2000 200-mg doses for about \$3.50. On this basis, Dr. Blake estimates the cost of 1000 200-mg tablets (produced in very large

* Per Dr. F. H. Meyers (Meyers, 1971), tabletted NaI and KI are still used sometimes as expectorants in the treatment of asthma. The standard dose is in the order of 300 mg or more (coated tablets). Thus it is possible that some individuals taking 100 mg at a time would exhibit cold-like symptoms.

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quantities) would be in the range \$1.50 to \$2.00*. This takes into account price reductions on raw material in quantity, which would tend to compensate for the cost of tableting and of packaging (presumably in large sealed brown-glass bottles, or possibly in sealed metal containers). Presumably 2000 100-mg tablets (produced in very large quantities) would also cost \$1.50 to \$2.00.

We may now estimate total costs for blocking protection of a population of 200 million people, for each of several assumed levels of protection:

1. One-time protection; that is, one administration of 200 mg. - This requires 200 million 200-mg tablets (or 400 million 100-mg tablets). Estimated cost: \$300 - \$400K.

2. One-week protection; that is, a 200-mg tablet (or 2 100-mg tablets) on the first day, followed by daily administration of 100 mg for the next 4 days, or perhaps by 400 mg spread out over 6-7 days - This requires the equivalent of 600 million 200-mg tablets (or 1.2 billion 100-mg tablets). Estimated cost: \$900 - \$1200K.

3. Eighty-day protection (ten half-lives of I-131)** that is, a 200-mg tablet (or 2 100-mg tablets) on the first day, followed by daily administration of 100 mg for the next 79 days. - This requires the equivalent of 8.1 billion 200-mg tablets (or 16.2 billion 100-mg tablets). Estimated cost: \$12.2 - \$16.2 M.

* More refined cost estimates would require the efforts of pharmaceutical manufacturers engaged in tableting operations; those estimates would depend on the buyer's specifications as to purity, stability, quantities, packaging, etc.

** Modified recommendation of Ramsden, et al. (1967), for protection against intake by ingestion, particularly of milk.

5.5. Summary

Methods and costs of administration of blocking agents depend on the expected circumstances in which they would be required, the extent of protection desired, and the possible side effects of administration of iodide, in quantities of hundreds of milligrams to grams, to large populations. Because the uncertainties as to potential intake of radioiodines are so great, it appears that once the decision to stockpile blocking agents is made, it is reasonable to plan for sufficient quantities to provide a reduction of at least 99% in the uptake of iodine by the thyroid. This means an initial administration of 200 mg of KI per person, followed by daily intake of 100 mg of KI for as long as protection is required.

The gravest threat, if it exists at all, is the inhalation of the total mixture of radioiodines at very early times (the first few days after the attack). For a shelter population breathing filtered (or at least ducted) air, this can occur at significant levels only if there is extensive and rapid volatilization of iodine from fallout particles deposited outside the shelter and there is a prolonged (order of perhaps days) temperature inversion. While rapid-enough volatilization is extremely unlikely for totally siliceous fallout particles, it may occur with particles containing calcium oxide or related compounds (resulting from surface explosions over limestone or in central sections of cities, where there are high densities of concrete), particularly where fallout particles in the vicinity of the shelter deposit in bodies of water or in standing water (or snow), or are wetted extensively by heavy rainfall.* In view of the great uncertainty as to the extent

* It is recognized that heavy rainfall is generally incompatible with the required temperature inversion. However, heavy dew may moisten particles sufficiently to accelerate volatilization.

of this potentially major early threat, it seems worthwhile to be prepared for it. Of all the possible unavoidable radioiodine intake modes, it is the most conveniently and cheaply countered, since it requires at most the administration of a total of about 600 mg of KI per person over the course of the first week, (2 100-mg tablets on, or shortly before, arrival of fallout, the remainder over the next 4-6 days; for those who cannot swallow pills, the tablets can be dissolved in fluids), at a cost (for 200 million people) of at most \$1.2 million.*

Intake of the mixture of radioiodines by a quasi-inhalation (impaction) route is possible: (1) for people forced to work outdoors (presumably for very short periods) at early times, either during fallout arrival or afterwards (in the latter case, only if fallout particles are resuspended in the air by winds or by human activities); or (2) where significant quantities of fallout enter shelter spaces, for example through broken or open windows or by being tracked in. In either such situation, the controlling factor will most likely be the whole-body ionizing-radiation dose (including its impact on the thyroid, which cannot be countered by blocking). The later such intakes occur, the less dangerous they are, since the long-lived I-131 is only about one-tenth as effective as the other radioiodines, rad for rad, in producing thyroid damage.

It may seem that another direct inhalation threat would come from the (delayed) arrival of world-wide fallout from perhaps thousands of megatons of fission spread over the North

* If one could be certain that the inhalation threat existed for only one day (most importantly the day of fallout arrival), only one administration of 200 mg of KI per person would be required, at a total cost of at most \$400K. Even here, there might be some compromise in protection because of recycling of radioiodine within the body; however the 99% reduction in uptake would still hold, even with the one-time administration.

American and Eurasian land masses. Here, even air bursts contribute to the threat. Dry particles in such fallout are small enough to be inhaled directly; iodine in rainfall is susceptible to volatilization after arrival. Delayed tropospheric fallout may be almost entirely deposited after only one pass around the world (say within 2-4 weeks). Stratospheric fallout has a retention half-life long enough that most of the iodine will have decayed before deposition. Generally, the radioiodine will be almost entirely I-131 by the time of arrival, providing some safety. In addition, extrapolation of the Tripler and Walter Reed I-131 urine burdens (ca 0.3 nCi) (See Appendix A, Table A.3) from a few tens of megatons upward to the nuclear-attack assumptions of a few thousand megatons still leaves levels of intake of I-131 of only a few tens of nano Curies, not enough to be of major concern.

Intake of radioiodine (almost entirely I-131) by the forage/cow/milk route has not been quantified in this report, for reasons stated in Section II. It is being investigated as part of a study on criteria for use of food in the post-attack situation. It is assumed for the moment, however, that with adequate stockpiling of powdered milk (and/or with stored forage), consumption of contaminated fresh milk can generally be held off until such milk is safe to drink (say between 5 and 10 half-lives of I-131, or between 40 and 80 days, preferably closer to the latter). Where consumption of contaminated fresh milk at early times is unavoidable (and this assumes that cows will have survived the whole-body and other doses implicit in their grazing on contaminated fields), then protection can be provided by administration of 100 mg of KI (one capsule) daily as long as the contaminated milk is being consumed, or, per the suggestion of Ramsden, et al. (1967), by addition of 300 mg of KI to each gallon of local milk (with daily consumption

of 1 pint of milk per person assumed)*.

If blocking iodide is to be stockpiled, in any event, against the (possible, but not very likely) massive early inhalation threat, then it would seem worthwhile to be prepared to counter the other, less important or more easily controlled, threats (delayed fallout, quasi-inhalation (impaction), milk intake) by the stockpiling of additional iodide. Eighty-day protection against any or all of these would require 8.1 g of iodide per person, which for 200 million people would cost at most \$16.2 million.

Possible side effects of blocking-iodide administration must be taken into account; however, the important consideration is the possible consequences if blocking is not used. For a given radioiodine environment, that component of the population that is at the greatest risk is the infant or very young child (and perhaps the fetus in utero), because for a given intake of radioiodine, the small size of the young thyroid implies a disproportionately high ionizing-radiation dose. Furthermore, the young thyroid is growing rapidly, so that a given thyroid dose may be disproportionately effective in (ultimately) producing a pathological state. Children should therefore receive the same dosage of iodide as adults; there is no reason to believe they are more vulnerable to side effects. Adolescents and pregnant women are in a metabolically stressful state, so that a given ionizing-radiation dose to their thyroids may also produce a disproportionately high effect. The general adult population is least at risk; also, because of the long induction period for the onset of ionizing-radiation-induced thyroid pathology (except where doses are so high as to produce early ablation),

* To maintain the assurance of reduction of radioiodine intake by 99% would require the addition of 800 mg of KI per gallon at this consumption rate.

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the oldest members of the population may be at the least risk.

For all members of the population, the administration of a one-time blocking dosage of 200 mg of KI appears to be quite safe; even administration of a total of 600 mg over the course of 5-7 days appears safe for the vast majority of the population. Administration of 100 mg daily for a period of say 2 months should be safe for most of the population; for those for whom it may be risky, decisions would depend on circumstances.

For several of the known occasional side effects of prolonged administration of high levels of iodide (iodism, iodide goiter) the incidence of effects is statistical, not deterministic; that is, predisposition to the effects is probably involved, but the nature of the predisposition is not known.* If an unknown 10% (or less) of a population may exhibit such side effects, it is undoubtedly better to protect 100% of the population against radioiodine uptake, whose consequences are usually irreversible, and take the risk of side effects in the unknown 10%, particularly since most of the side effects are reversible, disappearing on cessation of excess-iodide intake. Minor effects, such as the possible appearance of cold-like symptoms (sniffling, eye-watering) in some individuals taking a 100 mg dose of iodide, should be expected, but are tolerable.

Long-term administration of iodide is contra-indicated for individuals suffering from chronic kidney and/or cardiac failure, and in cases of pulmonary tuberculosis. Medical judgment on the spot would be required to decide on blocking

* However, people with strong allergic reactions in general are more likely to show allergy to iodide; those known to have been sensitized to iodide are most likely to react.

administration to such individuals; however, it is only realistic to point out that such individuals would be at great risk in any event in the shelter and the postattack environment.

The most dangerous side effect for healthy members of the population is the possibility of iodide goiter in infants born to mothers who have ingested large quantities of (stable) iodide for long periods. The rate of incidence of this condition, given a population of mothers so exposed, is unknown, but probably small; however, where it does occur, the risk of fatal respiratory obstruction in the newborn is extremely high. Again, with a known (high) risk of radioiodine uptake by the fetal thyroid, it would always be wiser (statistically) to administer blocking iodide to the pregnant woman. Unfortunately, in the shelter situation the decisionmakers would be in the dark about the actual long-term radioiodine threat; thus, in some circumstances the "cure" might be worse than the "disease". However, the neonatal iodide-goiter syndrome is very unlikely to develop after only a one-time, or even a one-week, administration of iodide to the mother. Therefore, the best prophylaxis might be to protect the pregnant woman (and thereby the fetus) against massive early intake of radioiodine, by one-week administration of iodide, and then take special pains to protect the mother against subsequent ingestion of radioiodine, so as to obviate the need for further administration of blocking agent to her.*

* This same philosophy can be applied to the entire shelter population: that is, in the face of uncertainty, protect everyone by first-week administration of blocking agent; continue administration as needed, depending on habits and subsequent ingestion history, and on increasing knowledge (perhaps measurement, by surviving laboratories) of the radioiodine threat.

SECTION VI

SUMMARY AND CONCLUSIONS

In the years 1963 through 1969, severe thyroid pathology, requiring surgical removal in most cases, developed in 17 of the 19 Rongelap children exposed to the highest levels of fallout experienced in the populated areas of the Marshall Islands following the CASTLE BRAVO nuclear explosion of March 1, 1954. Despite the facts that: (1) intake of fission products (in particular, of iodine-131) had been measured in the Rongelap population shortly after exposure; and (2) all the Marshallese exposed to significant levels of fallout had been carefully examined over the years since the event; the ultimate development of the thyroid pathology was totally unexpected. It seems clear that the cause of the thyroid abnormalities was beta irradiation of the thyroid by the mixture of radioiodines concentrated in that gland after ingestion and/or inhalation, and that the children would have had a much higher thyroid dose than adults taking in the same quantities of radioiodines, in view of the smaller size of the child thyroid.

The experience of the Marshallese children illustrates one combination of conditions leading to a serious radioiodine threat; by its very occurrence it suggests that worse radioiodine threats may occur in nuclear warfare. These could become important where people in shelter are well shielded from penetrating-radiation (whole-body) exposure, but vulnerable to inhalation of radioiodine vapors. Subsequent ingestion of large quantities of radioiodine in milk is another possible threat. The inhalation threat is considered to be the potentially-more-serious one, because if it develops at all, it will be at its most dangerous level on the first day of attack; this means that dealing with it will require planning and preparation, as

well as action during the probably-most-disorganized period of the attack situation. On the other hand, the milk-ingestion problem is to a large extent amenable to after-the-fact consideration and action.

The purpose of the effort reported here was therefore to evaluate the nature and extent, if any, of the threat of inhalation of radioiodines (in situations where other weapon effects would not control the outcome) and to evaluate and recommend reasonable countermeasures, where required. The first approach to the threat question was to examine what happened in the Marshall Islands. The native Marshallese lived under fairly primitive conditions in the context of protection from fallout; even if radioiodine did not vaporize from fallout particles, the Rongelap children could have received massive intakes of radioiodine by direct ingestion of fallout particles on food, and by quasi-inhalation by nasal impaction of larger particles* during play outdoors, followed by swallowing. However, a group of exposed American military personnel on Rongerik atoll, who also had high measured intakes of radioiodine, were protecting themselves as best they could from fallout contamination, so that direct ingestion, and even quasi-inhalation of fallout particles, were much less likely routes of entry of radioiodine. Therefore, a major part of this reported effort was to examine later-measured individual body burdens of longer-lived (non-iodine) radionuclides in the Rongerik men (the iodine-131 levels had been measured (early) only in one pooled sample for the entire Rongerik population; the same situation obtained for the Rongelap population also), and to attempt to correlate these body burdens with the reported movements and activities of the individual

* Particles small enough to be inhaled directly could not have existed in the arriving fallout. This is generally true in the nuclear-attack situation also.

Rongerik personnel in an effort to establish whether inhalation was the probable route of entry of radioiodine for them. An important consideration in extrapolating from the Marshall Islands situation to the nuclear-attack situation in the continental United States is the obvious difference between the coral-type fallout from Pacific Proving Ground tests and the expected predominantly siliceous particles from surface bursts in the continental United States.*

Since direct exposure to the intake of fallout particles per se is usually ruled out in the shelter situation (at least where ventilating air is ducted, and usually filtered against dust ingress), a major question is whether radioiodines in fallout will volatilize from fallout particles. Therefore, the weapons-effect literature was reviewed, and an evaluation of the possible extent of such volatilization was made. Then the inhalation problems (measured as the ultimate ionizing-radiation doses to the thyroid) in the shelter environment were characterized in terms of two potential sources of inhalable radioiodine: (1) gaseous radioiodines originating from particles trapped in the ventilating system; and (2) those originating from particles deposited outside the shelter.

The next part of the reported effort was an examination of possible countermeasures to radioiodine intake, whatever its mode or form (but with emphasis on the inhalation threat). Vapor filtration of air in shelters, not a standard practice, would be an expensive countermeasure, and has not been evaluated. Administration of stable iodide, as a so-called blocking agent, is fairly inexpensive, and has therefore been looked at carefully. Considered in this evaluation were: (1) blocking effectiveness, as related to dosage and scheduling of blocking agent; (2) potential side effects of the administration of blocking agent; (3) modes of administration; and (4) costs for protection of the total U.S. population, based on recommended

* Detonations on limestone or on urban targets may create fallout with coral-like or intermediate properties.

quantities and schedules.

Supporting evidence, examined in some detail in appendices to the report, includes, in addition to that already mentioned in this Summary:

Basic information on the nuclear and physicochemical properties of the fission-product radioiodines in fallout particles.

A methodology for the estimation of the extent of penetration of gaseous radioiodine in the shelter environment, and for the calculation of the resulting ionizing-radiation dose to the thyroid.

A summary of background information on normal-thyroid function, iodine-uptake modes and extent, and the models used to characterize the behavior of iodine in the thyroid and in the thyroid hormones.

A summary of background information on thyroid pathology, with specific reference to that induced by ionizing radiation. It includes estimates of risk per rad of thyroid exposure dose, for both benign and malignant tumors.

The important results and conclusions of this investigation are as follows:

The Marshall Island Experience.

1. The high levels of I-131 and of the non-volatile radionuclides found in the early pooled Rongerik urine samples resulted from massive intake, presumably a mixture of inhalation and ingestion, on shot day and on D+1, before the servicemen were evacuated from the Atoll.
2. The lower levels of the non-volatile radionuclides found later in individual Rongerik urine samples resulted from the same source, and were therefore valid input data for an attempted correlation of body burdens with individual location and behavior during the first two days.
3. No single factor (with respect to individual behavior or exposure) appeared to account for the variation in the measured body burdens of the non-volatile radionuclides in the Rongerik men. Fallout on Rongerik was so ubiquitous,

apparently, that it would have been almost impossible for the men to avoid ingestion (by nasal impaction and swallowing) of fallout particles, at least, to say nothing of true inhalation (the latter being a possibility if there had been volatile forms of radioiodine in the air, as a result of the peculiar properties (relatively high solubility) of coral-type fallout).

4. The generally accepted (literature) dosimetry for the Rongelap children, including the whole-body dose from penetrating radiation, as well as the thyroid dose from radioiodine uptake, is compatible with all the clinical measurements and symptoms, early and delayed; therefore, there is little to be gained by attempting to refine the dose calculations. If one accepts recent assertions in the Soviet literature that rad for rad a thyroid exposure dose from I-131 is only about 1/10th as effective as that from x-radiation (or from the more-energetic betas of I-132, I-133 and I-135, asserted to be equivalent to x-radiation, rad for rad), then the thyroid pathology in the Rongelap children is consistent with the calculated thyroid dose. This is true for both the statistical incidence of pathology and the severity in a given individual. Another important consequence of the Soviet assertion, if valid, is that any future massive uptake of I-131 only (in nuclear war) will be less important than has been assumed, following the development of the Marshallese pathology.

Volatility of Radioiodine in Fallout Particles.

1. From limited direct-measurement information available from nuclear tests in the SURNAM series, it is concluded that the radioiodine volatilization rate from (dry) siliceous fallout particles is of the order of 0.00025% per day.

2. Where siliceous fallout particles are exposed to water, radioiodine will be leached out of the particles at a much higher rate; the apparent half-life for leaching of the available radioiodine may be only about 1.5 day, but the total amount of leached radioiodine may be less than the total contained in the particle. Once dissolved in water, iodine (even as iodide, which can be oxidized) is immediately available for volatilization.

3. Where coral-type fallout particles are exposed to water, very rapid release of the radioiodine to the water is likely, so that the volatile-radioiodine threat may be considerable in such a situation. Direct volatilization of iodine from such particles, even when "dry", may be faster than in siliceous particles.

4. Nuclear weapons exploded at the surface over limestone or in central sections of cities, where there may be high concentrations of concrete, may produce fallout particles containing calcium oxide and related calcium compounds in concentrations high enough to produce more extensive and more rapid release of radioiodine to water* and, perhaps, more rapid direct volatilization from "dry" particles.

Potential for Inhalation of Radioiodine in Shelter.

1. Under worst-case assumptions, the inhalation threat from particles trapped in shelter ventilating systems is minimal, and in any event, is only a small fraction of the whole-body penetrating-radiation threat (even with a shelter protection factor of the order of 40).

2. On the basis of the best available information, it appears that the only situation in which inhalation of radioiodine by shelter populations can create a threat that must be countered is one in which: (a) a long-lasting temperature inversion accompanies or immediately follows the arrival of high levels of local fallout; and (b) there are significantly large areas of shallow water nearby, in which fallout particles (particularly if they contain significant percentages of calcium oxide and related compounds, deriving from limestone or concrete at the burst point) can deposit. Heavy rains accompanying or immediately following fallout arrival would enhance iodine volatilization; however, they are not likely to be associated closely in time with temperature inversions. The formation of dew on deposited fallout particles could lead to limited leaching of radioiodine, and therefore a possible volatilization threat in the temperature-inversion situation.

3. Clearly, the important variables in this specific kind of threat situation are almost impossible to parameterize for planning purposes; thus, it is not feasible to calculate, for a given assumed attack, the number of individuals who will be (ultimately) injured if not protected, at the time of fallout arrival, against the effects of intake of radioiodine. Nevertheless, the threat of such injury appears serious enough, and the cost of the appropriate countermeasure (administration of blocking iodide) low enough, that it is worth planning such a countermeasure.

* As compared to totally siliceous particles.

The Blocking Countermeasure.

1. Blocking with stable iodide works by a combination of isotope dilution and saturation of the thyroid against further uptake of iodine in any form.

2. In the shelter situation, under the tremendous uncertainties as to the quantitative measure of the inhalation threat, it appears that blocking will be useful only if it reduces the uptake of iodine to 1% of normal, or less. This implies the administration of 200 mg of potassium iodide just prior to, or at the start of, the arrival of fallout. Protection against continuing inhalation during the first week would be provided by the administration of an additional 400 mg of KI at the rate of 100 mg per day over the next 4 days, or spaced over the next 6 days. The cost of such protection (in the form of a stockpile of 1.2 billion 100-mg tablets, stored in the shelters) for 200 million people would be, at most, \$1.2 million, or less than a penny per person so protected.

3. If blocking iodide is to be stockpiled, in any event, against the (possible, but not very likely) massive early inhalation threat, then it would seem worthwhile to be prepared to counter the other, less-important or more-easily-controlled, threats (delayed fallout, quasi-inhalation during outdoor activities, contaminated-milk intake) by the stockpiling of additional iodide. 80-day protection against any or all of these would require (at 200 mg of KI on D-day, followed by 100 mg per day for the next 79 days) 8.1 g of KI per person, which for 200 million people would cost at most \$16.2 million, or about \$0.08 per person.

4. Possible side effects of prolonged administration of blocking iodide (iodine allergy, iodide goiter, etc.) must be taken into account. However, the important consideration is the possible outcome if blocking is not used. That component of the population that is at the greatest risk (to ionizing-radiation-produced thyroid pathology) is the infant or very young child (and perhaps the fetus in utero). The general adult population is least at risk.

5. For all members of the population, the administration of a one-time blocking dosage of 200 mg of KI appears to be quite safe; even administration of a total of 600 mg over the course of 5-7 days appears safe for the vast majority of the population. Administration of 100 mg daily for a period of say 2 months should be safe for most of the population; for those for whom it may be known to be risky, decisions would depend on circumstances.

6. Incidence of the major known side effects of prolonged administration of high levels of stable iodide is statistical, rather than deterministic; that is, predisposition to the effects is probably involved, but the nature of the predisposition is not known. If an unknown 10% (or fewer) of a population may exhibit such side effects, it is undoubtedly better to protect 100% of the population against a high risk of radioiodine uptake, whose consequences are usually irreversible, and take the risk of side effects in the unknown 10%, particularly since most of the side effects are reversible, disappearing on cessation of excess-iodide intake.

7. The most dangerous side effect for healthy members of the population is the possibility of iodide goiter in infants born to mothers who have ingested large quantities of stable iodide over long periods. The rate of incidence of this condition, given a population of pregnant women so exposed, is unknown, but probably small; however, where it does occur, the risk of fatal respiratory obstruction in the newborn is extremely high. Again, with a known (high) risk of radioiodine uptake by the fetal thyroid, it would always be wiser (statistically) to administer blocking iodide to the pregnant woman. Where the risk of radioiodine intake is not certain, the picture is not as clear. However, the neonatal iodide-goiter syndrome is very unlikely to develop after only a one-time, or even a one-week, administration of stable iodide to the pregnant woman. Therefore, the best prophylaxis might be to protect the pregnant woman (and thereby the fetus) against massive early intake of radioiodine, by one-week administration of iodide, and then take special pains to protect the mother against subsequent ingestion of radioiodine for the remainder of her pregnancy, so as to obviate the need for further administration of blocking agent to her.*

8. This same philosophy can be applied to the entire shelter population. That is, in the face of uncertainty at early times after attack, protect everyone by first-week administration of blocking agent; continue administration as needed, depending on habits and subsequent ingestion history, and on increasing knowledge (perhaps measurement) of the radioiodine threat.

* As a general rule, the nursing infant should receive blocking agent directly; if the mother is receiving blocking agent, her milk may contain more radioiodine than it would if she were not so protected.

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APPENDIX A

MODE OF INTAKE OF RADIOIODINE AND OTHER
RADIONUCLIDES IN AMERICAN MILITARY PERSONNEL ON RONGERIKA.1. Introduction

It has been emphasized in the text of this report that the measured extent of intake of radioiodine in the American military personnel on Rongerik in March of 1954 was anomalously high (Harris, P. S., 1954). It had been anticipated (stated in reports after the event) that their intake of radioactivity would be quite low, compared to that of the Marshallese on Rongelap, since: (1) the Americans had had radios that indicated the arrival of fallout; (2) they had covered up and had taken shelter in aluminum hutler buildings as soon as possible; (3) they had showered and changed clothing; and (4) their food and drink had been packaged or otherwise protected from contamination. On the other hand, the Marshallese on Rongelap had eaten food exposed directly to fallout (including fish drying on racks in the open), had drunk water from a cistern that collected rainwater that had fallen on an already contaminated roof, and had been exposed to fallout particles in the open for two days. (It was known that the Marshallese children playing in the sand had been literally rolling in the fallout). Nevertheless, the per-capita radioiodine burden in the Americans (measured in one pooled urine sample taken on D-17), bore about the same ratio to their whole-body exposure dose as did the per-capita radioiodine burden in the Marshallese (also one pooled sample, taken on the same day) to their whole-body dose. In other terms, the whole-body exposure dose for the Rongelapese was about 2.5

times that of the Americans, while the Rongelap per-capita radioiodine intake was about 3 times the American. This rough equivalence implied to the present author that either (1) the major route of intake of I-131 had been inhalation, or (2) the Rongerik servicemen had somehow been ingesting radioactivity to the same extent as the Rongelapese (or that both routes had been important factors in the Rongerik intake).

Information on the protection, movements and habits of the individual Rongerik Americans during and after the arrival of fallout is available (Sharp and Chapman, 1957). The pooling of their urine samples for determination of radioiodine precluded an attempt here to correlate individual radioiodine burdens with personal histories. However, Cohn, et al. (1957) had beta-ray counted individual urine samples, taken on D+44 from both the Rongerik Americans and the Rongelapese. Essentially, this counting determined the presence of non-volatile radionuclides (predominantly Sr-89, with smaller quantities of Zr/Nb-95 and Ba/La-140). Again, the intake ratio, on an average (per-capita) basis, was about three times as high for the Rongelapese as for the Rongerik Americans*. Since both groups lived in the same radiological environment after their evacuation to Kwajalein and up to the time of Cohn's sampling, the constancy of the intake ratio supports the contention that the measured activities resulted from intake (either ingestion or impaction/swallowing) during the first few days on Rongelap

* Also, Harris had determined Sr-89 and Ba-140 in the D+17 pooled samples. The average Rongerik Sr-89 level was about 1/5 that for Rongelap; for Ba-140, the corresponding ratio was about 1/1.3, with poorer counting statistics.

or Rongerik, rather than from inhalation or ingestion of delayed fallout.*

On that assumption, Cohn's data could be checked against the reported histories (shot day through D+1) for the individual Americans in an attempt to correlate body burdens of the longer-lived activities with those histories. If correlations were found, they might explain the source of the radioiodine also.

The next section of this Appendix presents an examination of eleven hypotheses as to the route of internal contamination of the Rongerik Americans, based on Cohn's data, as well as on data of a similar nature reported by Brennan (1954) (based on somewhat later sampling).

In addition to the longer-lived radionuclides, I-131 was found by Brennan in the urine of the Rongerik servicemen, in samples taken almost two months after Castle BRAVO. The final section of this Appendix presents an evaluation of the source of that radioiodine, the consequences of that evaluation with respect to the origin of the radioiodine found originally by Harris (1954), and the reason why Brennan's radioiodine data were not used in the tests of hypotheses.

* The large particles (order of 100 μm diameter) in the fallout on Rongerik and Rongelap could not have been inhaled directly (that is, have passed directly to the lung). However, some could have impacted in the nasal passage and then have been "swallowed", leading to the same uptake mode as in true ingestion. The much-smaller particles in delayed fallout (order of 1 μm diameter) could have been inhaled directly. In either situation, radionuclides of elements such as strontium, zirconium and niobium (unlike those of iodine) could not have existed as true vapors. Therefore, the only way they could have reached the urine if taken up by D+1 was via ingestion or impaction/swallowing.

A.2. Tests of Hypotheses on Ingestion of Non-Volatile Radio-Nuclides

The pertinent data of Cohn, et al. (1957) are individual body "burdens", in beta dis./min in 24-hr urine samples taken on D+44. The predominant activity was found to be Sr-89 (ca. 42%); lesser quantities of Zr/Nb-95, and Ba/La-140 were present also. Data were presented (by name of individual) for only 23 of the 28 Rongerik Americans. Ten of the 23 had reported levels of 0 dis./min.* That fact suggested that the histories of those ten might have a common element, or at least lead to an explanation of the source of the intake. A descriptive investigation of their histories and, by contrast, of those with measurable "burdens", gave no clue as to what might have caused or contributed to the intake. The next step was to postulate hypotheses as to the source(s) of intake, and for each hypothesis, calculate the average "burden" for all the men who could have been subjected to that source, and then compare the result to the average "burden" for all the others.

Each of the 11 hypotheses involves an element of exposure to fallout or else is based on another measurement that is correlated with exposure. The hypotheses are in the form: "Intake is caused by, or correlated with: 1. Being in the open.....". A listing, with comments, follows:

* Cohn did not state the size of the aliquot used, nor did he quantify the background level. Also, his counting efficiency was probably only about 5-10%. If the aliquot was small, and the gross count close to background, he may have arbitrarily called the net count zero; this would have scaled up to a total count (activity) of zero also. Therefore, the reported zero activity for the whole 24-hr sample for any one individual may be suspect. One would still, in any event, have confidence in the average count for the Rongerik Americans, since that figure, as discussed earlier, was about 1/3 that of the Rongelapese, and was therefore consistent with the iodine measurements of Harris (1954).

1. Being in the open at least 2 hr. on March 1 (during fallout).

The assumption here is that a man in the open during fallout is exposed to the impaction/swallowing route, and also to contamination of the whole body, leading to a subsequent ingestion threat.

2. Being in the open (or in non-Butler building*) most of March 1 (during fallout).

The assumption of hypothesis 1 applies here also.

3. Doing outdoor decontamination of any kind.

Even after the cessation of fallout, the process of decontamination may stir up the radioactive particles and thus expose the individual again. Hosing down of buildings is an example.

4. Not having a shower or full washup during the two days on Rongerik.

If whole-body external contamination implies a subsequent ingestion threat, then failure to remove the contamination early may maximize the threat.

* Butler buildings were 24' x 8' steel quonsets. They had screened windows, which could be closed, along the sides. Quarters for the Air Force men were Butler buildings, as were the mess hall, the rawinsonde building, the dispensary and supply building, and 4 smaller buildings housing the refrigerators. The 5 Army men were normally quartered in a tent, with no screening; tent flaps were always elevated, even on March 1-2. Other facilities in which men spent time during and after the fallout were: (1) an air-conditioned radio equipment van (air-conditioning turned off after fallout started); (2) a radio shack "about the size of a piano crate. Could not completely close door."; and (3) various tents, including those used for the water-distillation plant, the OIC's work area, supplies, latrines, and rawinsonde equipment.

5. Consumption of water and/or coffee.

If the water supply was contaminated,* this would be a major route of ingestion.

6. Consumption of solid food (either day).

Since fallout particles were known to have accumulated in visible layers on horizontal surfaces inside buildings (even the buttoned-down Butler buildings), food could have been contaminated during preparation or consumption.

7. Consumption of solid food (March 2).

This more restrictive hypothesis is included because all but 2 of the 28 men ate the evening meal on March 1. However, only 12 men ate solid food of Rongerik origin on March 2.

8. Having less-than-full body cover on March 1.

Again, the body-contamination/ingestion route.

9. Wearing no cap or T-shirt (or other shirt) on March 1.

Same route. This more restrictive hypothesis is included because all but 5 of the men were in Status "yes" on Hypothesis 8 (at least partially uncovered).

10. Having external contamination greater than 6 mr/hr (just before first shower after evacuation to Kwajalein).

Another attempted correlation with external contamination.

* The Rongerik report (Sharp and Chapman, 1957) states that the water distillation unit was housed in a tent, and that drinking water was stored at housing areas in covered 5-gallon cans. The report states elsewhere that the unit was a 300-gallon Badger type, the vapor-compression system being under a tarpaper roof with 2 sides of the shelter closed and 2 sides open. The report fails to say whether any of the water drunk, or used in cooking, was distilled after fallout had started. Also, there is no information on the water-intake point; that is, the depth at intake, and whether intake was on the lagoon or the ocean side. Sharp's recollection (1971) is that all drinking and cooking requirements on March 1 and 2 were met by previously produced water.

11. Having external contamination greater than 25 mr/hr (just before first shower after evacuation to Kwajalein).

A more restrictive hypothesis than 10, for comparison purposes.

The tests of the 11 hypotheses are presented in Table A.1. Numerical entries under Status "Yes" are measurements for those individuals who were exposed as stated in the hypothesis. Cohn's data are in the first two columns of entries; additional data of Brennan's (1954) are in columns 3 and 4. Brennan took individual 24-hr urine samples on about D+63, and measured (his Table 3, Appendix IV) total beta-plus-gamma counts per minute (thin-window G-M counter) in two fractions from wet-ashed urine specimens (one containing zirconium, niobium and rare earths, the other containing strontium and barium). The present author combined Brennan's count rates for the two fractions to produce beta-plus-gamma count rates for individual samples corresponding to those of Cohn, et al. Brennan's aliquots were never smaller than one-half the total 24-hr collection for the individual. His net count rates averaged about 30% of background, and his stated precision on the individual net count rates was about $\pm 20\%$. Thus, his data, although he considered them insignificant in terms of permissible body burdens, may be more reliable on an individual basis than those of Cohn, et al.* Nevertheless, both sets of data fail to support any of the 11 hypotheses strongly.

Table A.2. is a summary for all the hypotheses. Note that the direction of the correlation (anti-correlation) is

* Further evaluation of the significance of Brennan's beta-plus-gamma count rates will be presented in the next section of this Appendix.

TABLE A.1

TESTS OF HYPOTHESES ON ROUTES OF INTERNAL
CONTAMINATION OF RONGERIK SERVICEMEN

HYPOTHESIS 1. Being in the open for at least 2 hr on March 1 (during fallout) causes significant intake.			
Individual Activities* (Cohn, et al., 1957)		Individual Count Rates** (Brennan, 1954)	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 d/m	0 d/m	4.4 c/m	6.7 c/m
820	0	15.9	Incompl. meas.
78	248	Incompl. meas.##	9.2
No measurement#	0	Incompl. meas.	5.9
385	1260	6.1	8.3
0	No measurement	Incompl. meas.	Incompl. meas.
965	830	Incompl. meas.	12.1
438	No measurement	10.0	8.8
0	0	9.2	7.3
0	466	4.9	10.5
353	No measurement	7.0	3.8
750	0	9.4	3.4
187	0	11.7	8.8
323		7.9	
No measurement		16.5	
<hr/> 4299/13	<hr/> 2804/10	<hr/> 103.0/11	<hr/> 84.8/11
= 331 d/m	= 280 d/m	= 9.4 c/m	= 7.2 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

* Cohn's "individual activities" are individual body "burdens", in beta dis/min in a 24-hr urine sample taken on D+44. These activities are chiefly Sr-89 (ca. 42%), plus lesser quantities of Zr/Nb-95 and Ba/La-140.

** Brennan's "individual count rates" are individual body "burdens", in beta-plus-gamma counts/min (in a specified G-M counter geometry) in a 24-hr urine sample taken on about D+63. The same radionuclides are involved, except that Ba/La-140 are even less important than in Cohn's data.

Cohn et al. had no measurements for 5 of the 28 Rongerik servicemen.

Brennan had incomplete measurements for 6 of the 28, 2 of the 6 being missed in Cohn's measurements also. Thus only 19 of the men had measurements in both groups of data.

TABLE A.1 (continued)

HYPOTHESIS 2. Being in the open (or in non-Butler Bldg.) most of March 1 (during fallout) causes significant intake.			
Cohn		Brennan	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
820 d/m	0 d/m	15.9 c/m	6.7 c/m
78	0	Incompl. meas.	4.4
No measurement	0	Incompl. meas.	Incompl. meas.
965	248	Incompl. meas.	9.2
438	0	10.0	5.9
0	1260	3.4	8.3
187	No measurement	11.7	Incompl. meas.
	385		6.1
	0		Incompl. meas.
	830		12.1
	0		9.2
	No measurement		8.8
	0		7.3
	466		10.5
	0		4.9
	353		7.0
	No measurement		3.8
	750		9.4
	323		7.9
	No measurement		16.5
	0		8.8
<hr/>	<hr/>	<hr/>	<hr/>
2488/6	4615/17	41.0/4	146.8/18
= 415 d/m	= 272 d/m	= 10.2 c/m	= 8.2 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

TABLE A.1 (continued)

HYPOTHESIS 3. Doing outdoor decontamination of any kind causes significant intake.			
Cohn		Brennan	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 d/m	820 d/m	6.7 c/m	15.9 c/m
0	78	4.4	Incompl. meas.
248	0	9.2	Incompl. meas.
1260	0	8.3	5.9
385	No measurement	6.1	Incompl. meas.
0	No measurement	7.3	Incompl. meas.
0	0	4.9	Incompl. meas.
No measurement	965	3.8	Incompl. meas.
323	438	7.9	10.0
No measurement	830	16.5	12.1
	0		9.2
	No measurement		8.8
	466		10.5
	353		7.0
	0		3.4
	750		9.4
	187		11.7
	0		8.8
<hr/>	<hr/>	<hr/>	<hr/>
2216/8	4887/15	75.1/10.	112.7/12.
= 277 d/m	= 326 d/m	= 7.5 c/m	= 9.4 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

TABLE A.1 (continued)

HYPOTHESIS 4. Not having shower or full wash-up on Rongerik causes significant intake. (Status "Yes" means "Not having")			
Cohn		Brennan	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 d/m	248 d/m	6.7 c/m	9.2 c/m
0	0	4.4	5.9
820	No measurement	15.9	Incompl. meas.
78	385	Incompl. meas.	6.1
0	0	Incompl. meas.	Incompl. meas.
1260	965	8.3	Incompl. meas.
No measurement	438	Incompl. meas.	10.0
0	830	9.2	12.1
No measurement	0	8.8	7.3
466	0	10.5	4.9
323	353	7.9	7.0
No measurement	No measurement	16.5	3.8
0	0	8.8	3.4
	750		9.4
	187		11.7
<hr/>	<hr/>	<hr/>	<hr/>
2947/10	4156/13	97.0/10.	90.8/12.
= 295 d/m	= 320 d/m	= 9.7 c/m	= 7.6 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

TABLE A.1 (continued)

HYPOTHESIS 5. Consumption of water and/or coffee* causes significant intake.			
Cohn		Brennan	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 d/m	0 d/m	6.7 c/m	4.4 c/m
820	0	15.9	5.9
78	1260	Incompl. meas.	8.3
0	No measurement	Incompl. meas.	Incompl. meas.
248	No measurement	9.2	Incompl. meas.
385	0	6.1	Incompl. meas.
965	No measurement	Incompl. meas.	8.8
438	0	10.0	4.9
830	No measurement	12.1	3.8
0	187	9.2	11.7
0	323	7.3	7.9
466	No measurement	10.5	16.5
353		7.0	
0		3.4	
750		9.4	
0		8.8	
-----	-----	-----	-----
5333/16	1770/7	115.6/13	72.2/9
= 333 d/m	= 253 d/m	= 8.9 c/m	= 8.0 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

* Status "Yes" includes unspecified "liquids". Status "No" includes juices only (presumed to be canned), or "status of liquid consumption unspecified".

TABLE A.1 (continued)

HYPOTHESIS 6. Consumption of solid food (either day) causes significant intake.			
Cohn		Brennan	
Status "Yes"*	Status "No"	Status "Yes"*	Status "No"
0 d/m	1260 d/m (No info. on food)	6.7 c/m	8.3 (No info.)
0	830	4.4	12.1
820		15.9	
78		Incompl. meas.	
0		Incompl. meas.	
248		9.2	
0		5.9	
No measurement		Incompl. meas.	
No measurement		Incompl. meas.	
385		6.1	
0		Incompl. meas.	
965		Incompl. meas.	
438		10.0	
0		9.2	
No measurement		8.8	
0		7.3	
466		10.5	
0		4.9	
353		7.0	
No measurement		3.8	
0		3.4	
750		9.4	
187		11.7	
323		7.9	
No measurement		16.5	
0		8.8	
<hr/>	<hr/>	<hr/>	<hr/>
5013/21	2090/2	167.4/20.	20.4/2.
= 239 d/m	= 1045 d/m	= 8.4 c/m	= 10.2 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

* Everyone on Status "Yes" list ate evening meal on 1/1.

TABLE A.1 (continued)

HYPOTHESIS 7. Consumption of solid food on March 2 (breakfast or lunch) (not counting tea lunch from arriving aircraft) causes significant intake.			
Cohn		Spencer	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 g/n	0 g/n	4.4 g/n	6.7 g/n
820	78	15.9	Incompl. meas.
0	248	Incompl. meas.	9.2
No measurement	0	Incompl. meas.	5.9
0	1260 (No info. on food)	Incompl. meas.	6.3 (No info.)
945	No measurement	10.0	Incompl. meas.
438	395	6.8	6.5
No measurement	830	4.9	17.1
0	0	3.8	7.2
No measurement	0	3.4	20.4
0	446	11.7	7.0
187	153		9.8
	750		9.0
	323		14.5
	No measurement		8.8
	0		
<hr/>	<hr/>	<hr/>	<hr/>
2410/9	4692/14	62.9/8	121.9/14
= 268 g/n	= 335 g/n	= 7.9 g/n	= 8.7 g/n
Overall average = 309 g/n		Overall average = 8.5 g/n	

TABLE A.1 (continued)

HYPOTHESIS 8. Having less-than-full body cover on March 1* causes significant intake.			
Cohn		Brennan	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 d/m	0 d/m	6.7 c/m	Incompl. meas.
0 (Before 1630)	No measurement	4.4 (1630)	Incompl. meas.
820	830	15.9	12.1 c/m
78 (Before 1700)	0	Incompl. meas. (1700)	9.2
248	No measurement	9.2	8.8
0		5.9	
1260		8.3	
No measurement		Incompl. meas. (1700)	
385 (Before 1700)		6.1	
0		Incompl. meas.	
965		Incompl. meas.	
438		10.0	
0 (Through P.M. 3/2)		7.3 (P.M. 3/2)	
466		10.5	
0 (Before 1630)		4.9 (1630)	
353		7.0	
No measurement (Before 1700)		3.8 (1700)	
0		3.4	
750 (Before 1730)		9.4 (1730)	
187		11.7	
323		7.9	
No measurement		16.5	
0		8.8	
-----	-----	-----	-----
6273/20	830/3	157.7/19.	30.1/3.
= 313 d/m	= 277 d/m	= 8.3 c/m	= 10.0 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

* Everyone had full clothing coverage, including cap, after 1530 3/1, unless otherwise indicated in table.

TABLE A.1 (continued)

HYPOTHESIS 9. Wearing no cap or T-shirt (or other shirt) on March 1* causes significant intake.			
Cohn		Brennan	
Status "Yes"	Status "No" (Head and Upper Torso Covered)	Status "Yes"	Status "No"
0 d/m	0 d/m	6.7 c/m	Incompl. meas.
0 (Before 1630)	No measurement	4.4 (1630)	Incompl. meas.
820	0	15.9	Incompl. meas.
78 (Before 1700)	965	Incompl. meas. (1700)	Incompl. meas.
248	830	9.2	12.1 c/m
0	0	5.9	9.2
1260	No measurement	8.3	8.8
No measurement	353	Incompl. meas.	7.0
385 (Before 1700)	0	6.1 (1700)	8.8
438		10.0	
0 (Through P.M. 3/2)		7.3 (P.M. 3/2)	
466		10.5	
0 (Before 1630)		4.9 (1630)	
No measurement (Before 1700)		3.8 (1700)	
0		3.4	
750 (Before 1730)		9.4 (1730)	
187		11.7	
323		7.9	
No measurement		16.5	
<hr/> 4955/16	<hr/> 2148/7	<hr/> 141.9/17.	<hr/> 45.9/5
= 310 d/m	= 307 d/m	= 8.3 c/m	= 9.2 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

* Everyone had full clothing coverage, including cap, after 1530 3/1, unless otherwise indicated.

TABLE A.1 (continued)

HYPOTHESIS 10. Having external contamination > 6 mr/hr (before first shower on Kwajalein) is associated with significant intake.			
Cohn		Brennan	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 d/m	0 d/m	6.7 c/m	4.4 c/m
78	820	Incompl. meas.	15.9
No measurement	0	Incompl. meas.	Incompl. meas.
No measurement	248*	Incompl. meas.	9.2*
385	0*	6.1	5.9*
0	1260	Incompl. meas.	8.3
965	0	Incompl. meas.	9.2
438		10.0	
830		12.1	
No measurement		8.8	
0		7.3	
466		10.5	
0		4.9	
353		7.0	
No measurement		3.8	
0		3.4	
750		9.4	
187		11.7	
323		7.9	
No measurement		16.5	
0		8.8	
<hr/>	<hr/>	<hr/>	<hr/>
4775/16	2328/7	134.9/16.	52.9/6.
= 299 d/m	= 333 d/m	= 8.4 c/m	8.8 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

* But had had shower on Rongerik. If these are moved to "Yes" column, new averages (Cohn et al.) become "Yes": 279 and "No": 416; and (Brennan) "Yes": 8.3 and "No": 9.5.

TABLE A.1 (concluded)

HYPOTHESIS 11. Having external contamination > 25 mr/hr (before first shower on Kwajalein) is associated with significant intake.			
Cohn		Brennan	
Status "Yes"	Status "No"	Status "Yes"	Status "No"
0 d/m	0 d/m	6.7 c/m	4.4 c/m
78	820	Incompl. meas.	15.9
No measurement	0	Incompl. meas.	Incompl. meas.
965	248*	Incompl. meas.	9.2*
438	0*	10.0	5.9*
0	1260	7.3	8.3
466	No measurement	10.5	Incompl. meas.
0	385*	3.4	6.1*
187	0*	11.7	Incompl. meas.
No measurement	830*	16.5	12.1*
0	0	8.8	9.2
	No measurement		8.8
	0*		4.9*
	353*		7.0*
	No measurement		3.8*
	750*		9.4*
	323		7.9
<hr/>	<hr/>	<hr/>	<hr/>
2134/9	4969/14	71.9/8.	112.9/14.
= 237 d/m	= 355 d/m	= 9.4 c/m	= 8.1 c/m
Overall average = 309 d/m		Overall average = 8.5 c/m	

* But had had shower on Rongerik. If these are moved to "Yes" column, new averages (Cohn, et al.) become "Yes": 276 and "No": 401; and (Brennan) "Yes": 8.3 and "No": 9.1.

TABLE A.2

**SUMMARY OF TESTS OF HYPOTHESES ON ROUTES OF
INTERNAL CONTAMINATION OF RONGERIK SERVICEMEN**

HYPOTHESIS Intake is caused by, or correlated with:	AVG. ACTIVITY IN 24-HR URINE SAMPLE			
	Cohn, et al. (1957)		Brennan (1954)	
	Status "Yes"	Status "No"	Status "Yes"	Status "No"
1. Being in open at least 2 hr. on Mar. 1 (during fallout).	331 d/m	280 d/m	9.4 c/m	7.2 c/m
2. Being in open (or in non-Butler bldg.) most of Mar. 1 (during fallout).	415	272	10.2	8.2
3. Doing outdoor decontamination of any kind.	277	326	7.5	9.4
4. Not having shower or full washup on Rongerik.	295	320	9.7	7.6
5. Consumption of water and/or coffee.	333	253	8.9	8.0
6. Consumption of solid food (either day).	239	1045*	8.4	10.2*
7. Consumption of solid food (Mar. 2).	268	335	7.9	8.9
8. Having less-than-full body cover on Mar. 1.	313	277	8.3	8.5
9. Wearing no cap or shirt on Mar. 1.	310	307	8.3	9.2
10. Having external contamination > 6 mr/hr (before first shower on Kwajalein).	299	333	8.4	8.5
11. Having external contamination > 25 mr/hr (before first shower on Kwajalein).	237	355	9.4	8.1
	Overall Average = 309 d/m		Overall Average = 8.5 c/m	

* 2 individuals, one of whom may have eaten solid food.

the same for both sets of data in 7 of the 11 hypotheses. This is (weak) support for the contention that both investigators were at least measuring the same phenomenon. Also, a plot (not reproduced here) of Brennan's count rates against Cohn's activities (for the 19 men for whom both had complete data) shows a weak correlation between the individual measurements of the two investigators.

A.3. Radioiodine in Individual Rongerik Urine Samples Taken at Late Times

Toward the end of April, 1954 the Rongerik servicemen were moved from Kwajalein to Tripler Army Hospital, Honolulu, for further study before return to duty. It was at Tripler that the (Brennan) samples discussed in the previous section of this Appendix were taken.* During the same period (on or before May 5), 24-hr urine specimens were taken for radioiodine determination (Brennan, 1954); the levels found were about 10% of permissible concentrations, and decay and beta-absorption (aluminum-absorber) measurements confirmed that the recovered iodine was all I-131. In a supplementary report, Brennan (1954a) pointed out that all measurements made at Tripler on the Rongerik men were backed up by control measurements on local servicemen with no known exposure to radioactivity. In working up those data, Brennan was surprised to find that the controls had the same levels of urine iodine-131 as the Rongerik men.** This finding was later confirmed by

* The chemistry and counting were done later, at Walter Reed Army Hospital, Bethesda, Md.

** Rongerik: 219×10^{-6} μCi per average (individual) 24-hr sample (May 5), based on 7 samples.

Controls: 172×10^{-6} μCi per average (individual) 24-hr sample (May 5), based on 5 samples.

Later measurements were even closer together; in some, the control levels were higher than the Rongerik levels.

the pooling of several control samples at Tripler on May 9, and by the taking and pooling of more control samples (from a new population) at Walter Reed Army Hospital, Bethesda on May 12. According to Brennan, "these data indicate that the body burdens of I-131 are about the same for Hawaii and Washington, D. C. residents. This at least suggests a world wide equilibrium as far as I-131 is concerned." Every measurement pointed to the Bikini/Eniwetok test series (which had continued into about mid-May, 1954) as the source of the I-131. Pooled urine samples taken at Walter Reed in mid-June had almost negligible levels of that radionuclide. It is of interest that pooled tapwater samples taken in early- and mid-May at both Tripler and Walter Reed contained no I-131.

The fact that the relatively high levels of I-131 found in Tripler/Walter Reed urine specimens were clearly the result of delayed tropospheric fallout suggested the possibility* that the earliest I-131 measurements on the Rongerik urine specimens (sampling at Kwajalein on March 18) might have been misinterpreted. The argument went as follows: Harris (1954) had assumed that the I-131 excreted in the urine on D+17 (March 18) had all been ingested (or inhaled) on shot day.** It is known that a very small fraction of the iodine taken into the body on a given day will be excreted on any subsequent day, since: (1) only 20-30% of the stable-iodine intake is retained in the thyroid (70-80% is excreted in the urine on day 1); and (2) the rate constant for release of organic iodine from the thyroid to the bloodstream for metabolic control is

* J. C. Greene, OCD Postattack Research, personal communication, June 22, 1971.

** This assumption was the basis for his own calculations of thyroid dose in the Rongerik servicemen (as well as the Rongelap adults) and had also been accepted by James (1964) in his calculations of thyroid dose in the Rongelap children and adults.

low, as is the rate constant for turnover (release) of extrathyroidal iodine (thyroxine). Thus, the burden of radioiodine on the day of intake, which was the basis for the thyroid-dose calculations, had been calculated by back-extrapolation from a very small measured quantity (scaling up by 3 orders of magnitude). If the D+17 Rongerik urine radioiodine actually came from another source, then the true intake was grossly over-estimated.

On the basis of unspecified kinetics, Harris had stipulated that 0.1% of a first day intake of iodine (stable, or radioactive-decay-corrected) would be found in a 24-hr urine specimen taken between the 15th and 17th days thereafter. (With radioactive decay taken into consideration, about 0.025% of a first-day intake of I-131 would be found in a 24-hr specimen on about the 16th day.) Harris' number is consistent with an uptake (retained portion) of 20%, a biological half-life of about 60 days, and the assumption that, of the iodine released when extrathyroidal thyroxine is degraded, half appears in the urine. If the assumption is made that all the (degraded) iodine appears in the urine, then the 0.1% figure is consistent with a biological half-life of about 130 days.*

* Ng (see James, 1964) used a slightly different approach, based on the peak I-131 content of the thyroid in μCi , rather than the total intake of stable iodine. He used three first-order rate constants. That for uptake was 3.7 day^{-1} ; that for release of (thyroxine) iodine from the thyroid was either 4.85×10^{-3} or $17.15 \times 10^{-3} \text{ day}^{-1}$; that for turnover of extrathyroidal thyroxine was either 7.2×10^{-2} or $13.8 \times 10^{-2} \text{ day}^{-1}$. Finally, he assumed that, of the iodine released when extrathyroidal thyroxine is degraded, 60% was excreted in the urine. Depending on which combination of the rate constants he used, the percent of peak thyroid content of I-131 excreted in the urine on the 15th day after intake ranged from 0.05 to 0.21, with an average value of 0.12%. It can be seen that this number is consistent with Harris' 0.1% of stable intake, since radiological decay to the 15th day would provide a factor-of-3.4 reduction in Harris' number, while conversion of Ng's basis to total intake would provide a factor-of-5 reduction in his number (for 20% uptake). Thus Ng's and Harris' final percentages are closer together than 1/2 the range of uncertainty in Ng's second or third rate constants.

In Harris' work, the actual quantity of I-131 in the average 24-hr Rongerik urine specimen on the day of sampling (D+17) was only 4.0 nCi. This implied to Harris a peak thyroid burden (on shot day) of 3.5 μ Ci of I-131 (intake of 17.5 μ Ci); the latter, together with the associated quantities of the other radioiodines (assumed not to fractionate with respect to I-131), led to a calculated thyroid dose of 50 "rep" (Harris' calculated intake of radioiodine, in so-called I-131 equivalents, was 1.9 mCi)*. Thus, a measured urine I-131 level of the order of nanocuries implied an original intake, in I-131 equivalents, of the order of millicuries.

Therefore, in quantitative terms, the argument against Harris' interpretation would be that the measured Rongerik urine content of 4.0 nCi on D+17 might have actually represented the 80% rejection of an intake of 5.0 nCi on that day (at Kwajalein), a level that might have been compatible with a delayed-tropospheric-fallout source.** If accepted, this argument would then imply that the same quantity was taken in by the Rongelapese (at Kwajalein) on D+17, so that their ingestion/inhalation burden on shot day was only 2/3 of what was originally calculated. A similar argument would apply

* The corresponding numbers for the Rongelap adults were:

Urine on D+17:	13.1 nCi
Peak thyroid I-131:	11.2 μ Ci
Original radioiodine intake, in I-131 equivalents:	5.1 mCi
Calculated thyroid dose:	150 "rep". (James' (1964) calculated thyroid dose for the Rongelap infants was about 1,000 rad, the higher value arising from the smaller size of the infant thyroid.)

** Qualitatively, the same argument would be made for the non-volatile radionuclides (Sr-89, etc.) found in the urine of the Rongerik men by Harris.

also to the later urine levels of the non-volatile radio-nuclides (Sr-89, etc.) (Cohn, et al., 1957), if it could be shown that the measured urine levels on D+44 were consistent with rejection of a major fraction of the intake from delayed tropospheric fallout on that day (at Kwajalein).

Resolution of the uncertainty was attempted via three approaches, all based on measured (or calculated) average 24-hr urine-sample levels of I-131 in Rongerik servicemen:

1. Convert measured levels to 24-hr I-131 excretion, in nanocuries per individual, at time of sampling. Compare results for different times and locations of sampling, to see if all outputs were of about the same order of magnitude.

2. Accepting Harris' assumption of a massive intake of I-131 on shot day (17.5 μ Ci per individual on that day), calculate the expected urine output of I-131 from that source on the day of Brennan's sampling (D+63). Compare with Brennan's measured average value. If the calculated value turned out to be much higher than the measured value, this would cast great doubt on Harris' assumption. On the other hand, if the calculated value was much lower than the measured value, this would confirm the already accepted conclusion that Brennan's results were a measure of delayed-tropospheric-fallout arrival, but would neither confirm nor deny the validity of Harris' assumption. Finally, if the calculated value was about the same as the measured value, this would lead to confusion, since it would imply the validity of both Harris' assumption and Brennan's conclusion, taken separately, but would at the same time say that both could not simultaneously be valid.

3. Starting with known shot dates in the CASTLE series, and with accepted rates of (eastward) travel of upper

tropospheric debris, investigate whether the finding of I-131 in urine on each of the sampling dates is compatible with the arrival of delayed tropospheric fallout at the sampling point on the sampling date, preferably on the first pass around the globe.

The overall results of all three approaches are presented in Table A.3. Backup data for the third approach are presented in Table A.4.

With the first approach, it appears that all measured 24-hr I-131 outputs are of the (roughly) same order of magnitude. While the measured level in the Harris samples runs 10-20 times the levels in all the others, it is not entirely inconceivable that the Harris levels are the result of delayed fallout on D+17 rather than of a release from a massive intake on shot day. That is, the case cannot be proven either way on the basis of the first approach alone.

From the second approach, we see that the calculated output of I-131 on May 2 is lower by a factor of about 7 than the measured output. This result provides further confirmation for the conclusion that Brennan's samples resulted from the arrival of delayed fallout on May 2, but neither confirms nor denies the validity of Harris' assumption about the source of the I-131 in the March 18 samples.

Consideration of the results of the third approach will be deferred pending further discussion of that approach:

All 6 shots in the CASTLE series at the Pacific Proving Ground occurred between March 1 and May 14, 1954 (Effects of Nuclear Weapons, 1962). No United Kingdom nor Soviet Union shots occurred between October 1953 and October 1954, and no other powers had yet started testing in 1954. Therefore, the 6 CASTLE shots must be examined as the only potential sources of the I-131 found by Harris and by Brennan.

TABLE A.3

**I-131 OUTPUT OF AMERICAN MILITARY
PERSONNEL (RONGERIK) AND CONTROLS**

Measured and Calculated I-131 in Urine
(24-hour collection, activity per individual)

Description	Sampling Date (1954, local time)	Activity on Sampling Date, nano Ci
1. Harris (1954) measurements	March 18	4.0* (implies intake of 17.5 μ Ci on March 1)
2a. Brennan (1954) measurements (Tripler Army Hospital, Hono- lulu)	May 2**	0.29***
2b. Calculated activity in urine (if from March 1 intake of 17.5 μ Ci)	May 2 (hypothetical sampling)	0.041****
3. Brennan (1954) measurements on <u>controls</u> (Walter Reed Hospital, Bethesda)	May 12	0.34

Measured activities in groups 2 and 3 are consistent with arrival, on the day of sampling, of first-pass delayed tropospheric fall-out from Pacific Proving Ground megaton-range tests. On the other hand, the Harris-measured activity (group 1) cannot possibly be due to delayed fallout (the BRAVO cloud was half-way around the world on the day of sample collection, and no other shots had occurred yet). See text and Table A.4 for details.

* Rongelap samples contained 3.1 times this activity.

** Actual sampling date was not specified. Counting was done on May 5, but sampling could have occurred on any date between April 29 and May 5. Errors introduced by this uncertainty do not affect the basic arguments (the activity on sampling date must have been in the range 0.22-0.37 nCi)

*** Local (Tripler) controls had roughly the same levels (0.22 nCi May 2, 0.43 nCi May 9).

**** Based on Harris' working hypothesis that 0.1% of iodine intake appears in urine (per day) about 14 days after intake, which apparently follows from the assumptions: (1) thyroid uptake = 20% of intake; and (2) (biological) half-life of iodine in thyroid = 60 days, 1/2 of released iodine appearing in urine. The calculation here was therefore based on assumptions (1) and (2).

TABLE A.4

DELAYED TROPOSPHERIC FALLOUT AS POTENTIAL SOURCE OF RADIOIODINE INTAKE

Sampling Location	Shot No.	Shot Date (Bikini Time)	Sampling Date (Local Time)	Est. Arrival Date (Local Time)		
				1	2	3
Kwajalein	1	Mar. 1	Mar. 18	Mar. 1*	Mar. 31	Apr. 30
	1	Mar. 1	May 2** ± 3&&&, May 9&&&	Mar. 3	Apr. 2	May 2&&&
	2	Mar. 27	May 2** ± 3&&, May 9	Mar. 29	Apr. 28&&&	May 28
	3	Apr. 7	May 2** ± 3, May 9&&	Apr. 9	May 9&&	Jun. 8
	4	Apr. 26	May 2** ± 3&, May 9	Apr. 28&	May 28	Jun. 27
	5	May 5	May 2** ± 3, May 9&	May 7&	Jun. 6	-
Bethesda (Walter Reed)	6	May 14	May 2** ± 3, May 9	May 16	Jun. 15	-
	1	Mar. 1	May 12&&&, Jun. 15 (weak)	Mar. 10	Apr. 9	May 9&&&
	2	Mar. 27	May 12&&, Jun. 15&&&	Apr. 5	May 5&&	Jun. 4&&&
	3	Apr. 7	May 12&&, Jun. 15&&&	Apr. 16	May 16&&	Jun. 15&&&
	4	Apr. 26	May 12, Jun. 15	May 5	Jun. 4	-
	5	May 5	May 12&, Jun. 15&&	May 14&	Jun. 13&&	-
6	May 14	May 12, Jun. 15&&	May 23	Jun. 22&&	-	

* The cloud passed the longitude of Kwajalein on March 1, but far to the north of the Atoll. There could have been no local nor "delayed" fallout there on that date. Furthermore, of course, the servicemen were on Rongerik on March 1. Finally, the small particles could not have fallen out that soon except in rain.

** Counting definitely done on May 5, however.

& Possible coincidence between fallout deposition (first pass) and finding of I-131 in urine; i.e., possible explanation for source of intake of I-131 on day of sampling. Allow uncertainty of ± 1 day in first-pass front arrival at Honolulu, ± 3 days for Bethesda. Also, allow 2-4 days for cloud passage.

&& Same, for second-pass arrival. Differences up to ± 7-day accepted as matches.

&&& Same, for third-pass arrival.

According to Effects of Nuclear Weapons (1962) (ENW), delayed tropospheric fallout is important for up to several months after a megaton-range detonation. For the smaller particles originally reaching the upper troposphere, the half-residence time is 2-6 weeks. The chief mechanism for return of the radioactivity to the earth's surface is scavenging by rain and snow, but return in a dry form also occurs. Transport is by west-to-east winds; according to ENW, Pacific Proving Ground fallout takes perhaps 1 month to circumnavigate the globe, the bulk of it in a belt that "spreads to a width of about 30° of latitude", originally centered on about 10° N Lat. The 1-month circumnavigation implies an effective wind speed of about 35 mph, or 800 miles per day, for fallout originating near the equator.* An idealized version of the reduction in fallout with downwind distance shows that the decay-corrected activity (or long-lived activity), averaged over a belt ca. 2000 mi wide around the downwind axis, has a "half-distance" of about 6500 mi from ground zero. The drop-off for point locations along the downwind axis itself is much steeper, decay-corrected fallout levels in relative units being 40 at 1000 mi, 20 at 2500 mi, 10 at 4500 mi, 5 at 7000 mi, and 2.5 at 10,500 mi; i.e., implying "half-distances" of 1500 mi at 1750 mi, 2000 mi at 3500 mi, 2500 mi at 5750 mi, and 3500 mi at 8750 mi.

Distances parallel to lines of latitude are as follows:

Bikini to Kwajalein:	ca. 170 mi
Bikini to Honolulu:	ca. 2500 mi
Bikini to Bethesda:	ca. 8000 mi

First-pass times of arrival are therefore:

Kwajalein:	1/4 day**
Honolulu:	3 days
Bethesda:	10 days

with second-pass arrival, if any, delayed another 30 days for each location.

* Later data on arrival from Chinese detonations (and some Soviet Semipalatinsk shots) imply a circumnavigation time of only about 15 days for fallout originating at about 40° N Lat. (Machta, 1968 and Machta and Telegadas, 1970).

** Since the north/south distance between Bikini and Kwajalein is also about 200 mi, the cloud could not have dispersed enough to have covered Kwajalein on the first pass. Also, none of the CASTLE detonations produced a local fallout pattern whose hot line had a southerly (to-the-south) component.

Table A.4 is a presentation of possible arrival dates, at each of the three sampling locations, for delayed fallout from each of the CASTLE detonations that occurred before the (last) sampling at that location.* Estimated arrival times can be compared with sampling dates, also shown in the Table. Note that estimated arrival times pertain to the cloud front. Since the (delayed) cloud may be several thousand miles in diameter, cloud passage may take 3 days or more at a given sampling location.

Thus, absolute coincidence between arrival of front and sampling date is not required for a match. Only if the front could not possibly have arrived by the day of sampling is a match ruled out. For example, fallout from Shot 5 (May 4, Honolulu time) would have had to travel 2500 mi in 1 day to reach Honolulu by the first sample-counting date, May 5, a very unlikely event, but could easily have arrived there by May 9, the second sampling date. Possible matches become more likely for more-distant sampling points and for later circumnavigations, simply because of the increased uncertainty in front-arrival time and the longer time for cloud passage.

It can be seen from Table A.4 that measured I-131 urine outputs (in the Rongerik servicemen and the controls) at Tripler and Walter Reed Hospitals during the month of May are each consistent with deposition, on the day of sampling, of first-pass delayed tropospheric fallout from some CASTLE detonation. Second-pass deposition provides further possible matches.

* Second- and third-pass (circumnavigation) arrival are included, but it must be remembered that arrival times are much more hazy for the later times, and fallout levels much weaker, as a consequence of decay, dispersion and loss by deposition during transport.

The weaker I-131 levels found in June 15 samples at Walter Reed are compatible (on an arrival-time basis) with second-pass deposition, but cannot be associated with first-pass deposition from any shot. The low levels themselves confirm this conclusion.

Perhaps the most important implication of the Table is that the I-131 in the Harris samples (taken March 18 on Kwajalein) could not have resulted from delayed fallout, since the Shot-1 (BRAVO) cloud was halfway around the world on the date of sampling, and no other detonations had yet occurred.

A.4. Possible Implication of Delayed Fallout in Intake of Non-Volatile Radionuclides.

There is no reason per se why the non-volatile nuclides (Sr-89, etc.) found in urine could not have been inhaled (on micron and sub-micron particles) during the passage of the delayed cloud.* Unfortunately, Brennan's results, discussed earlier in this Appendix, did not include any control measurements for such nuclides. Therefore, the question of their source in the Rongerik servicemen is still open. However, Cohn's measurements (1957) can be checked on an arrival-time basis. He sampled on D+44 (April 14) on Kwajalein. As explained in Table A.4, first-pass deposition was never a possible explanation for intake on Kwajalein. However, second- or third-pass deposition is a possibility, which can be examined

* However, inhalation of the non-volatile nuclides would have been less likely if deposition had occurred in rainfall.

by means of a table similar to Table A.4:

SAMPLING LOCATION: Kwajalein				
SAMPLING DATE : April 14 (Bikini/Kwajalein Time)				
Shot No.	Shot Date (Bikini/ Kwajalein Time)	Approximate Arrival Date		
		Pass Number		
		1	2	3
1	March 1	(March 1)	March 31	April 30
2	March 27	(March 27)	April 26	May 26
3	April 7	(April 7)	May 7	June 6
4	(April 26)	--	--	--

There appears to be no second-pass fit for the D+44 samples, and third-pass arrival is too late, even for the first shot. Finally, Harris' findings of substantial quantities of Sr-89 and Ba-140 in the pooled Rongerik urine sample taken on D+17 (March 18) (discussed earlier in this Appendix), coupled with the finding here (conclusion of Section A.3) that delayed-fallout arrival could not have been a factor on that date, further support the conclusion that there had been a massive intake of non-volatile radionuclides in the Rongerik servicemen on shot day and D+1 for the BRAVO detonation, and that this same intake was the source of the non-volatiles found by Cohn on D+44, and used in the analysis here in Section A.2.

A.5. Summary

On the basis of all the evidence presented and discussed in this Appendix, it seems reasonable to conclude that:

1. The high levels of iodine-131 and of the non-volatile radionuclides found by Harris in pooled Rongerik urine samples taken on D+17 (Shot 1) resulted from massive intake, presumably a mixture of inhalation and ingestion, on shot day and on D+1, before the servicemen were evacuated to Kwajalein.

2. The lower levels of the non-volatile radionuclides found by Cohn in individual Rongerik urine samples taken on D+44 (Shot 1) resulted from the same source, and were therefore valid input data for an attempted correlation of "body burdens" with individual behavior during the first two days.

3. No single factor (with respect to individual behavior or exposure) appeared to account for the variation in the measured "body burdens" of the non-volatile radionuclides in the Rongerik servicemen. Fallout on Rongerik was so ubiquitous, apparently, that it would have been almost impossible for the men to avoid ingestion, to say nothing of inhalation (the latter being a possibility if there had been volatile forms of radioiodine in the air, as a result of the peculiar properties of coral-type fallout).

4. All of Brennan's measurements were consistent with intake (late) from delayed tropospheric fallout, but none of Brennan's results compromise the first 3 conclusions of this summary.

APPENDIX B

ASSESSMENT OF THE VOLATILITY OF
FISSION-PRODUCT IODINE IN FALLOUT PARTICLESB.1. Introduction

In the body of the report, it has been emphasized that the locations of interest in this work are the downwind high-exposure-rate areas in fallout fields from strategic nuclear weapons. In such areas, inhalation of radioiodine(s) is possible only if iodine volatilizes from deposited fallout particles. The well-studied initial cloud of radioiodine vapor, moving downwind near ground level, that is associated with an accidental reactor-release or with a venting deep-underground nuclear explosion simply does not exist in the civil-defense context.*

The purpose of this Appendix is, therefore, to present the results of a survey of the literature on volatility of fallout-particle radioiodine(s) and to provide a critical review of the limited pertinent information thus found.

The definitive publication on fallout and on fallout fields is the 5-volume DASA-1251 Series, "Local Fallout from

* During fallout formation in the fireball of the megaton-range surface burst, radioiodine produced in vapor form and untrapped by the soil matrix will become part of the delayed tropospheric or the stratospheric fallout. Most of the former will normally return to the earth's surface in greatly diluted quantities, after the loss by decay of most of the radioactive iodine isotopes. The latter, in even-more-diluted quantities, will return long after the decay of all the radioiodine of concern. Large quantities of tropospheric iodine vapor can be trapped in thundershowers, leading to an early return, but that is generally unpredictable.

Nuclear Test Detonations," based on an extensive survey of the U. S. weapons-test (WT and ITR) literature. Volumes III and IV (Part 1) are relevant to the radioiodine problem; they appear in the reference list for this report as Kawahara, et al. (1966) and O'Connor and Crocker (1968), respectively. DASA-1251 covers all U. S. nuclear weapons tests through (including) 1958. Nowhere in Volume III (physical and chemical properties of fallout) nor Volume IV, Part 1 (radiochemical and radiation characteristics of fallout) is there any information or discussion on volatility of iodine in fallout particles.

Because of the 1958-1961 test moratorium and the atmospheric-test-ban treaty of 1963, the only surface or near-surface tests after 1958 that could have produced the desired information on radioiodine volatility were those in the 1962 operations at the Nevada Test Site: i.e., the Sunbeam Series shots SMALL BOY, JOHNIE BOY and DANNY BOY; and the Ploughshare cratering shot SEDAN. W. B. Lane attempted to measure the extent of radioiodine volatility in fallout particles from SEDAN (Lane, 1964) and from SMALL BOY (La Riviere, et al., 1963 and Freiling, et al., 1964). The remainder of this Appendix will be devoted primarily to a presentation of Lane's approaches, interpretations and claimed results; a critical review thereof; and the author's assessment of the implications of Lane's work in the civil-defense context. Some indirect measurements of iodine volatility are discussed in Section IV.

B.2. Shot SEDAN

B.2.1. Introduction

SEDAN was the more thoroughly documented test, in that two experimental approaches to the iodine-volatility assessment were attempted, both at the same sampling/collecting station, and backup information on other properties of the fall-

out at that point in the field was also available. SEDAN fallout cannot be considered to represent true surface-burst fallout in all respects. In particular, material from a cratering burst* may be as much as a factor of 10^5 lower in specific activity than that from a surface burst of the same yield, since in the former a much larger mass of rock or soil mixes with the same quantity of fission products before the particles become airborne (Crocker, et al., 1966). Also, fractionation behavior and radial distribution of specific chains may not be the same as in surface-burst fallout.** Nevertheless, much useful information can be gleaned from the SEDAN data; in fact, because the SMALL BOY investigations on iodine were not as complete, it turns out that SEDAN provides the only directly useful information.

B.2.2. Iodine-Gas-Sampling Experiment

The best way to describe the experiment is to quote directly from Lane (1964) (unless it is stated otherwise, all references to SEDAN information will be based on that report):

"A gaseous iodine collection was made in the field. The devices employed two intakes; one intake sampled the airborne iodine, the other sampled the airborne iodine plus the iodine which was liberated from fallout deposited in a 1 foot diameter funnel. A funnel was connected to an Anderson Aerosol Sampler (a five stage impaction device with a millipore filter) so that any gaseous products evolved from the fallout would be carried through the system by the 3.8 liter/min air flow. In the first case the entrance to the Anderson Sampler was covered with a hood to exclude all particulate fallout, and an identical flow rate was used. A gasoline driven motor generator powered a vacuum pump which drew 3.8 liters of air per minute through each of two metering orifices. The orifices were connected to bubble columns containing 75 ml of 0.05M sodium thiosulfate solution. The bubblers were in turn connected to the Anderson Samplers. The millipore filters insured that no particulate matter reached the thiosulfate."

* SEDAN had a yield of 100 KT; it was fired at a depth of 635 ft.

**See Appendix C for fundamental information on chains.

The gas-sampling experiment was performed at Station 19, which was about 2 miles from ground zero (radial distance) and about 1 mile off the hot line*. About 4 hours before shot time, the equipment was put in place and the motor generator started. The gas sampler, together with three other collectors, was recovered from the station at H+31 hr.

"The radioiodine from the gas sampler in the field was determined by oxidizing the iodide in sodium thiosulfate solution with nitrous acid, extracting with carbon tetrachloride, reducing back into sodium thiosulfate and precipitating as silver iodide. The silver iodide precipitate was placed in a calibrated geometry and its gamma spectra measured with the 100-channel pulse height analyzer. Both samples showed only the 0.53 Mev peak characteristic of I-133 " at H+59 hr.

The chemical yield of the recovery procedure was presumably high (say greater than 70%) and the decontamination efficiency was clearly high. The principal gamma ray of iodine-133 (half-life 20.8 hr) is at 0.53 Mev, with a yield of 1 photon/dis. There are no other photons of importance.

The total debris on the millipore filter of the covered (hooded) sampler was 0.1 g, presumably in very small particles. The I-133 activity in the associated thiosulfate absorber (corrected to shot time, and under the assumption of 100% chemical yield) was 95 photons/sec, or 2.5 nCi. On the other hand, the millipore filter of the uncovered sampler held 95.8 g of particulate debris; the I-133 activity in the associated thiosulfate absorber, on the same basis as before, was 283 photons/sec, or 7.6 nCi.

* As delineated in Vuillemot (1963). By back-extrapolation from the (H+24)hr exposure rate at this point, as given in that report, it was found that the (H+1) hr exposure rate at Station 19 was of the order of 50 R/hr.

From these measurements, Lane concluded, "Airborne iodine fission products were found in the contaminated field downwind from ground zero." Partly on the basis of these measurements, he stated further, "It was found that iodine fission products volatilize and are released from particulate fallout." These statements are somewhat misleading, in that the quantities of iodine involved are insignificant in the hazard context. The following discussion will be an attempt to put this assessment into quantitative terms.

Lane recognized that, if the quantity of iodine vapor trapped by the thiosulfate absorber of the unhooded sampler (associated with 95.8 grams of collected fallout particles) was only about 3 times as much as that trapped by the absorber of the hooded sampler (associated with 0.1 grams of debris, or about 0.1% of the collected fallout in the unhooded sampler), then

(a) a substantial fraction of the gaseous iodine came from the field, rather than from the collected debris, and

(b) the release rate from collected particles was low. This combination implied to Lane that the fallout "cloud" passing over the station contained radioiodine in vapor form, in fact (Lane, November 1971) that the bulk of that radioiodine was independent-yield I-132, I-133, I-134 and I-135, all but I-133 then decaying to negligible activities by the time of counting.

Corrected to zero time, the I-133 activity released per gram by the collected particles was $(283 - 95) / (95.8)$, or 1.96 photons/sec per gm. This is based on the assumption that, in each thiosulfate absorber, 95 photons/sec came from "field" iodine vapor. The total I-133 loading of the collected particles, on the assumption of unfractionated*

* In his Abstract, Lane stated, "Gamma decay measurements showed no evidence of radionuclide fractionation in debris

(Footnote continued)

(Footnote continued)

from different locations, nor among different particle-size fractions. Pulse height distributions also indicated no significant fractionation of gamma emitting radionuclides." He also presented radiochemistry results for the major fission-product radionuclides, expressed in "f" numbers. For example, f_{131} represents the equivalent fissions in the sample, based on I-131. If Zr-95 is taken to represent the refractory nuclides, then $R_{131,95}$ ($= f_{131}/f_{95}$) is a measure of iodine fractionation (in the 131 chain). Similarly, $R_{132,95}$, based on Te-132 measurements, is a measure of iodine fractionation in the 132 chain. R values for several sieve fractions for each of several stations (including Station 19, where the gas-sampler was installed) are as follows:

<u>Station</u>	<u>Particle-size Range</u>	<u>$R_{131,95}$</u>	<u>$R_{132,95}$</u>
19	> 2830 μm	1.25	1.32
19	177 - 710 μm	0.69	0.48
19	< 44 μm	2.0	2.1
14	710 - 1410 μm	0.46	0.34
14	177 - 710 μm	0.14	0.32
14	< 44 μm	3.4	2.5
12	177 - 710 μm	0.80	0.42
12	< 44 μm	3.0	0.38

It appears that the smaller particles are somewhat enriched in the 131 and 132 chains (R values greater than 1.0), while the larger particles are depleted in these chains. This is to be expected on the basis of particle-formation thermodynamics and kinetics. The total sample at a station, having a weighted average of these R numbers, would probably appear to be unfractionated. This is supported by Lane's further finding, based on specific-activity distributions with respect to particle size, that the radionuclides are associated with the volume of the particle rather than with its surface area. Finally, the $R_{131,95}$ values are, more often than not, greater than the $R_{132,95}$ values. Samples from JOHNNIE BOY, another cratering shot, also showed $R_{131,95}$ values somewhat greater than the $R_{132,95}$ values (Clark, et al., 1963a). This would imply a somewhat greater escape of the 132-chain precursors during particle formation, a result which would be expected, based on the following analysis (Crocker, 1968): If particle condensation, per J. H. Norman, occurs as high as 2000°K, then the chain precursors Sn and Sb may be taken as volatile, but the even-earlier precursors Cd and In would still be taken as refractory. As one proceeds from the 131-chain through the 135-chain, independent yields shift to the later (daughter) members in the chain (Kochendorfer, 1969). That is, in the 131-chain, the peak independent yield

(Footnote continued)

(Footnote continued)

occurs between Sn and Sb and the independent yield of I is essentially zero; in the 132-chain between Sb and Te, but close to Sb; in the 133-chain about half-way between Sb and Te, with a substantial independent yield already for I; in the 134-chain between Te and I, but close to Te; and in the 135-chain between Te and I, but close to I, the independent yield of I-135 being almost 45% of the chain yield. As a result of these considerations, for surface-burst fallout the 131-chain is only about 60% volatile during particle formation; 132 ca. 70% volatile; and 133, 134 and 135 over 98% volatile. Fractionation data for the most volatile chains measured for SEDAN indicated that the actual extent of fractionation was nowhere near as great as would be predicted for such particle-formation volatilities.

(Footnote concluded)

debris, was estimated by Lane on the basis of: (1) 4π -ionization measurements of Station 19 debris; (2) predicted total ionization per fission, from Miller and Loeb (1958); and (3) expected activity of I-133 per fission, from Bolles and Ballou (1956). The debris was thus found to contain 1.1×10^{12} fissions/gm., leading to a zero-time I-133 specific "activity" of 6.6×10^5 photons/sec per gm. It follows that, in the 31-hr period of air flow through the collected particles, no more than $(1.96)/(6.6 \times 10^5)$, or 0.00030%, of the radioiodine actually volatilized. Taken by itself, that result would certainly **rule** out inhalation as a threat in the shelter situation (see argument in text).

There are some inconsistencies, which will be discussed further, in the data from the gas-sampler experiment. Nevertheless, one can put upper limits on the amounts of radioiodine that would have been inhaled in the Station 19 environment, regardless of how one interprets the data, so long as one accepts an efficiency of close to 100% in the trapping of iodine vapor and the recovery of iodine from the thiosulfate absorber.

The total I-133 collected in the unhooded sampler (environmental gaseous I-133 plus that, if any, from collected particles) was ca. 7.6 nCi (corrected to zero time).

If, contrary to the interpreted result of the experiment, this was all environmental gas, collected at a uniform rate (3.8 liter/min, or 0.134 ft³/min) over the 31 hours of collection, then it implies an average concentration in ambient air of the order of 0.03 nCi/ft³ (zero-time basis). Note that this is a very low concentration, in a civil-defense context.

The adult-human breathing rate is about 0.5 ft³/min, or ca. 14 liter/min. Thus an adult in that presumed environment would have inhaled, over the 31-hour period, (14./3.8)(7.6), or about 28 nCi (zero-time) of I-133. If the total 7.6 nCi in the experiment was actually collected during cloud passage, then an adult in that environment would have inhaled the same 28 nCi during that shorter period. This intake is a factor of the order of a thousand below that of the Rongelapese*, yet it (SEDAN) must include a substantial, if not dominant, contribution from radioiodine vapor in the passing cloud, a contribution impossible in the Rongelap situation.

The first potential inconsistency in the gas-sampler experiment results from the relative quantities of iodine vapor trapped by the two absorbers. Lane's calculation-by-difference implicitly assumed two simultaneous phenomena: (1) a relatively high contribution from release from the particles collected by the unhooded sampler; and (2) a relatively low contribution from environmental iodine vapor, including that released from fallout particles in the surrounding field. It has been emphasized in the text that, in the shelter-ventilation-system context, a relatively small contribution

* The Rongelapese had I-131 intakes of over 50 μ Ci. The (H+1) hr exposure rate at Rongelap was of the order of 100 R/hr, compared to a SEDAN Station 19 rate of about 50 R/hr.

is to be expected from particles trapped in intakes and ducting, even if the iodine release rate per gram of fallout is the same inside and outside the shelter. Therefore, other things being equal, it would be expected that both samplers would have roughly the same quantities of radioiodine trapped in the thiosulfate absorbers. The only apparent explanation for the factor-of-three ratio between the two samplers is that air flow over the collected particles substantially enhanced the release of iodine vapor, as compared to the presumably more quiescent conditions for particles reaching the ground and perhaps partially "buried" within the surface soil structure. In the latter situation, exchange of ionic radioactive species (such as iodide) with local soil is also a possibility.

A second inconsistency in the gas-sampler experiment results from the fact, already mentioned, that "both samples showed only the 0.53 Mev peak characteristic of I-133" when counted (pulse-height spectrometry) at H+59 hr. At that time, a significant contribution from the 0.364 Mev photon of I-131 (8 day) should have been seen.* Lane's first interpretation of this result was that the thiosulfate absorber trapped only early-arriving ("cloud") independent-yield radioiodine. Iodine-131, with essentially zero independent yield (actually about 1/20 of the I-133 independent yield), could come only via volatilization (as a daughter nuclide) from particles.**

* By H+59, I-135 (6.7 hr) and I-134 (53 min) would be gone, while I-132, the 2.3 hr daughter of 78 hr Te-132, would have decayed to insignificance (in the thiosulfate) in the 28 hours between sampler recovery and counting. However, the ratio of 0.364 Mev I-131 photons to 0.53 Mev I-133 photons would have been about 1:2.

** If the recovered iodine could have resulted only from independent-yield iodine, then the photon ratio (I-131:I-133) at time of count would have been about 1:45.

Unfortunately, this argument, if accepted, would rule out the apparent extent of volatilization (from collected particles) already discussed extensively in this section, and it would be even more damaging to the claimed results of the second "volatilization" experiment (performed at a later time post-detonation), to be discussed in the next section. Furthermore, if only the independent-yield I-133 in the passing cloud was collected, both samples should have recorded the same amount of activity.

A more reasonable explanation of the absence of I-131 in the pulse-height spectra is that the precursors in the 131-chain penetrated deeper into the particles than those of the 133-chain, so that the daughter I-131 was less available for subsequent release than the daughter I-133. This behavior would be consistent with the higher volatility, during particle formation, of the 133-chain (as predicted in the earlier discussion of fractionation).

B.2.3. "Volatilization" ("Air-Exposure") Experiment

Lane conducted a second experiment, designed to measure iodine loss by air exposure over a 10-day period. It was performed as follows:

"In a concurrent iodine study, ten 20 gram portions of fallout from station 19 were placed in petri dishes and exposed to normal air currents and sunlight. One of the 20 gram samples was added to a test tube containing 50 ml of 0.05 M sodium thiosulfate"..... at H+35 hr, "thus effectively trapping any iodine which would be released [thereafter] from the particles. Each day thereafter one of the portions was added to a thiosulfate solution. After ten days"..... on D+11 "the radioiodine was extracted from the mixture and measured in the following manner: Each test tube was measured initially in the 4- π ionization chamber. The slurry of thiosulfate solution and fallout was transferred

from the test tube to a bubble column. The iodine was oxidized with nitrous acid and the iodine transferred to another bubble column containing sodium thiosulfate by drawing a stream of air through the system. The iodine was extracted from the second thiosulfate solution by again oxidizing with nitrous acid and extracting into carbon tetrachloride. The carbon tetrachloride fractions were then measured in the 4- π ionization chamber to determine the radioiodine."

The stated approach of this experiment was determination of radioiodine remaining in fallout particles after varying periods of air exposure, the ultimate objective being to determine the rate of release of radioiodine from particles in the field. The proper technique, as Lane recognized, would have been application, immediately at the end of each air-exposure period, of standard radiochemical procedures (involving destructive analysis of the particles) to determine I-131 remaining in the particles. However, this was not feasible under the circumstances in the field, so the thiosulfate-trapping approach was used as an expedient.

The procedure used precluded the use of gamma spectrometry, since the slurry in the test tube (which had to be assayed before iodine separation) contained the complete fission-product mixture. Therefore, the purity of the iodine product was not determined. However, the chemical separation procedure used on iodide in the thiosulfate was essentially identical to that used in the earlier gas-sampler experiment, where the purity of the resulting iodine had been demonstrated by gamma spectrometry.

The main difficulty in interpreting the results of the experiment arises, as Lane recognized, from the fact that the iodine recovery (from immersed particles) by the thiosulfate-immersion technique was not known. Furthermore, as will be indicated, use of this technique introduced major questions about the validity of the experiment, which were not considered at the time. In fact, evidence will be pre-

sented that the claimed volatilization during air exposure did not occur, and that the apparent loss of radioiodine over time was really an increased leaching of radioiodine into the thiosulfate solution with higher leaching times (shorter air exposures).

Lane's results appeared in his Table 3.6, presented here in slightly modified form as Table B.1, and in his Figure 3.4, redrawn here as Figure B.1. He stated:

"Iodine loss by air exposure over a 10 day period is reported..... The progressive decrease in the observed iodine/total fraction with time indicates a loss of iodine during the period..... [Miller and Loeb (1958)] show that at all days [*] the radioisotopes of iodine contribute 25.0 percent of the gamma radiation (by 4- π ionization chamber) from normal U-235 thermal fission products. Dividing the observed iodine/total fraction by the expected fraction (0.25) yields the percentage of the theoretical iodine actually recovered[**]. The most apparent reason for the low initial percent.... [32.0 percent] was the inability of the analytical procedure to remove iodine which might have been trapped within the insoluble particles. In addition, the iodine may have been depleted from the outset due to fractionation in the fallout formation process."

Lane recorded no data for the so-called 1-day, 2-day and 10-day iodine samples, though he did for the corresponding total samples. The missing data might have provided key information for much of the evaluation that follows. In

* In particular, on D+11, the day on which all counting was done.

** This operation is misleading; most of the iodine ionization at 11 days is from I-132, which because of its short half life and of its being in secular equilibrium (in the particles) with its much-longer-lived parent, Te-132, will not be seen in the thiosulfate in significant quantities. This fact does not affect the arguments of the present author, however, since calculations can be made on the basis of the observed iodine/total ratio.

TABLE B.1

APPARENT LOSS OF IODINE FROM PARTICULATE DEBRIS BY AIR EXPOSURE

Sample	Duration of Air Exposure, Days	4-pi Activity at D+11		Iodine Total (Observed)	$\frac{(I/Total)_{obs.}}{(I/Total)^{*exp.}}$	Duration of Leach, Days
		Total ma	Iodine ma			
1	1	180×10^{-9}	-			9
2	2	185×10^{-9}	-			8
3	3	195×10^{-9}	15.6×10^{-9}	0.0800	32.0** %	7
4	4	170×10^{-9}	13.0×10^{-9}	0.0764	30.5	6
5	5	170×10^{-9}	13.2×10^{-9}	0.0776	31.1	5
6	6	165×10^{-9}	11.5×10^{-9}	0.0697	27.9	4
7	7	165×10^{-9}	10.5×10^{-9}	0.0636	25.4	3
8	8	160×10^{-9}	8.0×10^{-9}	0.0500	20.0	2
9	9	160×10^{-9}	6.4×10^{-9}	0.0400**	16.0	1
10	10	165×10^{-9}	-			0

* From Miller and Leeb (1958):

$$\text{At D+11, } \frac{I(\text{ma})}{\text{Total F.P. (ma)}} = 0.25 \text{ expected}$$

** Corrected for error in original table.

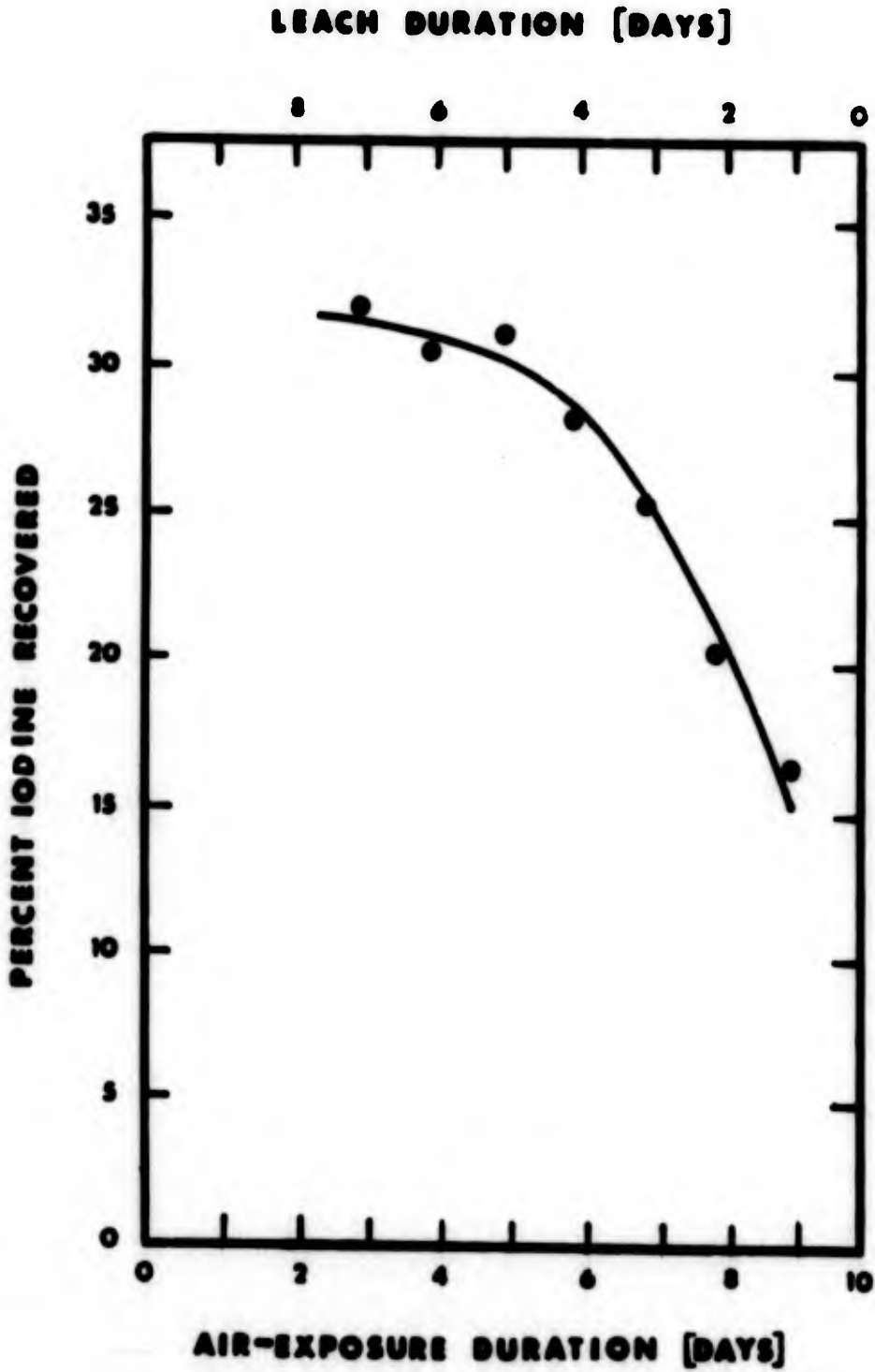


FIGURE B.1
APPARENT LOSS OF IODINE FROM PARTICULATE DEBRIS BY AIR EXPOSURE

the 3-day sample, 32.0% of the theoretical (available) iodine* ended in the thiosulfate. By the 9th day, only 16.0% of the total available iodine was recovered. Thus, if Lane's interpretation was correct, and volatilization was really being measured, then 16% of the total radioiodine was released between days 3 and 9 of the experiment (or between D+4 and D+10), implying an average volatilization rate of 2.7% per day. This rate is a factor of more than 10^4 higher than that represented by the vaporization of 0.00030% in 31 hours in the gas-sampler experiment. There is no apparent reason why this should occur, especially since both experiments involved fallout from the same station. The evidence indicates that the lower rate is the valid one.

It is understandable that "iodine trapped within the insoluble particles" would be very slow to be taken up by water or aqueous solutions (one would expect it to vaporize even more slowly); however the argument that "iodine may have been depleted from the outset due to fractionation in the fallout formation process" is in conflict with (1) the reported radiochemistry data that indicate no depletion of iodine in the fallout at the station under investigation; and (2) the stated conclusions that gamma decay measurements showed no evidence of radionuclide fractionation in debris from different locations, nor among different particle-size fractions, and that pulse-height distributions also indicated no significant fractionation of gamma-emitting radionuclides.

The most important consideration is that the experiment, as carried out, must have included some contribution from (variation in) leaching of iodine. In fact, it

* Since the counting was done on D+11, it is clear that the iodine involved was almost entirely I-131. The iodine members of the 133, 134 and 135 chains would have decayed away, and the 78 hr \rightarrow 2.3 hr Te-132 \rightarrow I-132 pair could have contributed no iodine in the experiment as performed.

is likely that it was leaching variation that was being measured, rather than volatilization. If the amount leached increased with duration of immersion, with an expected saturation effect, then this would account for the shape of the curve in Figure B.1, with leach (immersion) duration plotted from right to left on the upper abscissa scale*. That is, if leaching is the controlling phenomenon, then saturation occurs after about the fourth day of immersion; i.e., at initial immersion times earlier than day 6 of the experiment, or D+7. This behavior is reasonably consistent with the (limited) results of Lane's total-ionization (4π) leaching experiments**, in which the percent leached did not increase between day 3 and day 8 of immersion, these being the only times measured (Lane's Tables 3.7 and 3.8, not reproduced here). Since other supporting information results from the separate leaching experiments, they will be discussed here in some detail.

"The leaching of gamma emitting radionuclides from fallout particles by HCl solutions of pH 1.0 and 4.0, distilled water of pH 6.0 and NaOH solution of pH 10.0 was determined. Two grams of particles from the 42, 150, and 325 mesh and pan fractions were added to 25 ml of solution in 40 ml centrifuge tubes" on D+1*** "After 3

* On the other hand, the concave-downward shape of the curve is not consistent with the air-exposure explanation, since in that case it would imply a release rate that increases as time goes on.

** An independent set of experiments, not connected with the iodine investigation, but (fortunately) duplicating some of the conditions of that investigation.

*** This experiment was started on the same day as the "volatilization" experiment. The material used was also from Station 19; thus physicochemical and ionizing-radiation properties of the particles were identical in the two experiments. Note that the particle-size ranges involved in the leaching experiment were 350-710 μm , 104-124 μm , 44-62 μm , and 0-44 μm . On a weighted basis, this meant that the leaching experiment used 53% of the mass of the fallout mixture and 52% of the 100-hr activity, the total sample thus being representative with respect to specific activity.

days the tubes were centrifuged, the liquid decanted into clean tubes and the gamma activity of the solid and liquid fractions measured in the 4- π ionization chamber. A duplicate set of samples, "also started on D+1..... "was tested in 8 days."

A concurrent similarly performed experiment on exchange between fallout particles and (inactive) clay or adobe soil (in distilled water) was related, though not by specific intent, to the already mentioned problem of inhibition of vaporization of iodine from fallout particles "buried" within the surface soil structure in the field.

Lane's general conclusions for the leaching experiments were:

"The acidity or basicity of the solution apparently has little effect on the leaching result. Small particles with their much greater surface area do leach a larger fraction of the radionuclides than large particles." There is "little increased leaching in the additional five days of contact."

The present author has extended Lane's reported individual-sieve-fraction leaching percentages to expected percentages for the total fallout mixture at Station 19, by weighting the before/after "counts" for each sieve fraction by that fraction's mass percent in the mixture. The results are presented in Table B.2. The grand average of total (all-fission-product) leachability for all 8 samples (2 times, 4 pH's) is 10.8%. The apparent slight reduction in leachability from 3 days to 8 days was probably an artifact; almost half the leachate counts at 8 days were less than twice background.

TABLE B.2

LEACHING PERCENTAGE FOR TOTAL FALLOUT MIXTURE AT STATION 19

Solution	Percent Leached	
	3 days	8 days
pH 1.0	13.7 %	10.4 %
pH 4.0	14.6	8.7
pH 6.0	14.0	8.4
pH 10.0	7.3	8.7
Average	12.4 %	9.1 %

The fact that equilibrium leachability is of the order of 11% for the Station 19 fallout is quite significant in the context of the true mechanism in the "volatilization" experiment. The maximum "% iodine recovered" in the "volatilization" experiment (Table B.1 and Figure B.1) is 32% (for immersion time of 7 days). Since at all times in the interval 3 days to 10 days post-detonation the radiiodines in the gross-fission-product mixture contribute about 25% of the total 4π ionization (Lane's figure), this maximum "recovered" (iodine) ionization of 32% in the "volatilization" experiment represents 8% of the total-fission-product ionization in the sample (before immersion)*. Therefore, if during

* Or, note in Table B.1, in the entry for Sample 3 (7-day leach), that the observed ratio of recovered-iodine 4π ionization to total 4π ionization is 0.0800 (8%). Thus, the previously discussed ambiguity of the 32% figure (or the 25% basis figure), arising from the lack of a Te-132:I-132 contribution, does not affect the argument of the present author. That is, the observed 8% recovery has been first divided by 0.25 (by Lane) and then multiplied by 0.25 (by the present author).

the time period of interest iodine is the major contributor to the gamma-emitting soluble fraction of the total-fission-product mixture*, then the 8%-ionization figure in the "volatilization" experiment is consistent with the 11%-ionization figure (all leached radionuclides) in the leaching experiment, and thus could have represented leaching of iodine rather than volatilization. It is known that leaching of iodine is a much more rapid process than volatilization; the former requires only that the iodine be in the iodide (or any other soluble anionic) form, while the latter requires oxidation of the (thermodynamically preferred in particle formation) iodide to elemental iodine, followed by sublimation.** Furthermore, the leachant solution "attacks" the particle surface, and thus renders available more iodide than can be susceptible to true volatilization.

An additional piece of evidence for the leaching of iodine comes from Lane's gamma spectra of the liquid fractions in the 3-day total-mixture leach experiment (his Figure 3.6, not reproduced here). Though the spectra have not been unfolded, there appears to be a strong peak at about 0.37 Mev in both liquid fractions (pH 4 and pH 6), but a continuum in the same region in the corresponding solid fractions. The chief I-131 photon is at 0.364 Mev; the spectra were run on D+4, before I-131 would make its peak contribution to the total ionization.

* At 10 days post-detonation, I-131 alone constitutes between 5 and 10% of the total-fission-product ionization, depending on the fissionable element and the neutron spectrum.

** Any iodine in the iodate or periodate form would be stable (with respect to reduction to the elemental-iodine form) in the air environment.

JOHNIE BOY, like SEDAN a cratering shot, provided some supporting information (Clark, et al., 1963a, plus original data files) for the leach interpretation of the results of the SEDAN "volatilization" experiment. In the leaching experiment, performed exactly as in the SEDAN work, 3 size fractions in the range 61-351 μ m (all from the same station) were each leached for 1 and for 3 days, measurements being made in leachants of pH 1, 6 and 10. In every case (Clark's Table 3.12 plus original (raw) data), the percent of ionization that leached increased by a factor of 3 from day 1 of leaching to day 3. This tends to support the presumed shape of the leach equilibration curve in Figure B.1, this work.

Another JOHNIE BOY experiment involved a gross sample from another station, and 3 individual particles, each of a different type, from that station. Each sample was leached for 1 hr in pH 1 solution, starting on D+2. The percent leached, on a 4 π -ionization basis, was determined immediately, and then the liquid and solid fractions were followed in the ionization chamber for 3 days. If, for a given sample, both the liquid and the solid fractions were to contain the same mixture of radionuclides, then the (apparent) percent leached would be the same at all measurement times. However, it turned out (Clark's Table 3.11) that all samples exhibited a rising percent leached during the three days of measurement, the increase being a factor of 1.5 for the gross sample, and ranging from 1.4 to 3.4 for the individual particles. Therefore, there is apparently a preferential leaching of an activity that is relatively long-lived compared to the total-fission-product mixture. This result is consistent with a preferential leaching of 8-day I-131 ($t^{-1.2}$ decay implies an apparent fission-product half life of ca. 1.6 d starting on D+2).

Finally, the extent of leaching for the gross sample just described (leached for 1 hr) was about a factor of 10 lower than those for the 1-day leach measurements on the other JOHNNIE BOY samples. This provides further support for the presumed shape of the leach equilibration curve in Figure B.1, this work (very rapid increase during the first few hours, saturation by about 3 days).

Some comments on the effect of the nature of the leaching solution are in order. In the "fallout data compilation" (Kawahara, et al., 1966) there is some pertinent information on partly siliceous fallout from another Nevada Test Site series, Teapot. Particles of size 0-5 μm from five tower shots gave the following solubilities:

Leaching Medium	Percent Solubility (Activity Basis)	
	Average	Maximum
Distilled Water	2.4 %	6.4 %
0.1N HCl	22.8	36.2
0.1N Thiosulfate	4.2	9.0
pH 7.6 Buffer	5.7	15.0

However, on the predominant-size samples (tens through hundreds of micrometers), solubilities were not entirely consistent. The order of solubility was not always the same. Nevertheless, more often than not the order was as given for the small particles above; in particular, the solubility in thiosulfate was usually higher than that in distilled water. Whether the enhanced solubility in thiosulfate was due primarily to enhanced leaching of iodide in particular or to a general effect of ionic strength (see entries for pH 7.6 buffer) is not known.

In the concurrent SEDAN experiments on exchange (of the fission-product mixture) from fallout particles to finely divided soil materials, the exchange materials were from two to seven times as effective as the straight leach environment, the extent of exchange depending on fallout-particle size and on the nature of the exchange matrix. As has been stated above, this phenomenon has implications for the inhibition of iodine volatility from fallout particles deposited on soil, particularly where there is organic material in the soil in the fallout field.

B.2.4. Summary (SEDAN)

Based on the results of the gas-sampler experiment, which appear to be unequivocal when all apparent inconsistencies are accounted for, it is concluded that the iodine volatilization rate from SEDAN fallout particles is of the order of 0.00025 percent per day, a rate that cannot produce a radioiodine-inhalation threat.

The "volatilization", or "air-exposure" experiment, which produced an apparent iodine volatilization rate of about 2.7 percent per day (for material from the same sampling station), has been shown to be a leaching experiment, in which the apparent volatilization really reflects the effect of duration of immersion on the percent of ionic species leached. That is, even with zero prior volatilization of iodine from the air-exposed particles, the changes in iodine recovery in the thiosulfate solutions used to analyze the iodine content of the particles can be accounted for entirely on the basis of variation of extent of leach with duration of immersion of such particles in the thiosulfate solutions. Lane concurs with this interpretation (Lane, December 1971).

Therefore, a volatilization rate of the order of 0.00025 percent per day is accepted as the best value for SEDAN fallout particles. It is recognized that this result is based entirely on measurements at one station in the fallout field (where the 1-hr exposure rate was about 50 R/hr). Nevertheless, since the rate is negligible, there is no reason to believe that measurements at other points in the fallout field would have compromised the conclusion that for SEDAN-type fallout, volatilization of iodine could create no radioiodine-inhalation threat in the shelter environment in the civil-defense context.

B.3. Shot SMALL BOY

B.3.1. Introduction

Since SEDAN fallout cannot be considered to represent true surface-burst fallout in all respects, the surface burst SMALL BOY could have provided definitive information on the extent of iodine volatilization from fallout particles. Unfortunately, the gas-sampler experiment was not performed on SMALL BOY; of the two SEDAN experiments, only the "volatilization", or "air-exposure" experiment, which has subsequently been shown to provide no useful information on volatilization, was done. However, some less-detailed iodine measurements were made, and total-leaching experiments provided additional pertinent information.

B.3.2. Collection of Arriving Fallout in Thiosulfate Solution

In SMALL BOY Project 2.10, concerned with physico-

chemical and radiochemical behavior of fallout particles, another kind of iodine investigation was attempted by Kawahara (Freiling, et al., 1964):

"Concern over the vaporization of fission product iodine (I-131) from fallout samples led to the collection of some fallout by trays containing fresh 0.1N thiosulfate solution..... At NRDL these samples were separated into solid and liquid fractions, and analyzed spectrometrically."

The only statement about this experiment in the RESULTS section of the Project 2.10 report was:

"Visual comparison of the gamma-ray pulse-height distributions for samples of fallout collected in 0.1N thiosulfate with those of gross fallout samples did not indicate a greater abundance of I-131 in the former."

It appears from the latter statement (and from the absence of a reported comparison of the liquid with the solid fraction) that: (1) the slurry of particles and thiosulfate was kept intact until the moment of counting; (2) the only thiosulfate-collection measurements made were on unseparated slurry mixtures; and (3) the conventionally collected "gross fallout samples" referred to were exposed to air at least while in the field before recovery on D+1, and perhaps thereafter also. With the advantage of hindsight, one can say that this would have been a crude experiment at best. If the only air exposure of the comparison samples was that prior to recovery, say for one day or less, then even with claimed iodine release rates of about 2.5% per day* (shown here to be much too high) the experiment as performed could not have verified depletion of iodine in the conventionally collected particles, since at

* From the SEDAN "volatilization" experiment, already discussed (2.7% per day claimed), and the SMALL BOY "volatilization" experiment, to be considered shortly (2.4% per day claimed).

best the iodine contents would have differed by only 2.5%; such a difference could not have been seen by the comparison spectrometry used.*

B.3.3. "Volatilization" ("Air Exposure") Experiment

Lane modified the SEDAN air-exposure experiment slightly at SMALL BOY (Project 2.9) (La Riviere, et al., 1963). 3 samples (3 air-exposure times) were taken from each of two stations (H+1 hr exposure rates about 20 and 75 R/hr). The first sample from each station was immersed in thiosulfate solution at the start of the experiment on D+2. The remaining two samples from each station were held in petri dishes in sunlight and normal air currents. Sample 2 from each station was immersed in thiosulfate on D+6, sample 3 on D+12. Counting, filtration, chemistry and final counting were done on D+13. Thus, the immersion durations, in inverse order, were 1, 7 and 11 days. On a ^{131}I -ionization basis, the observed ratios of recovered iodine to total activity, in the same inverse order, were 0.025-0.03, 0.05-0.055, and 0.07-0.08. Thus the maximum (equilibrium) fraction of iodine recovered was about the same as that for SEDAN (0.08).

* There is another reason why the experimental procedure actually used is not known with certainty. The thiosulfate trays had been set out and recovered by Project 2.9 (La Riviere, et al., 1963) whose prime responsibility was fallout collection and gross-sample analysis. Their report states, with respect to collections in distilled water, 0.1N HCl and thiosulfate: "The liquids were simultaneously filtered and transferred to polyethylene bottles at recovery time. The solid fractions on the filter were also sent to Project 2.10." This separation in the field was confirmed by Lane, at least for the thiosulfate collection (Lane, 1972). It means that the resulting separated fallout particles could have been losing iodine by volatilization (if volatilization occurred at all), thus further vitiating the experiment.

Following the same kind of calculation performed on SEDAN, Lane concluded:

"Measurements made on fallout samples exposed to air showed a continuing loss of iodine over a period of 12 days following the burst. The fraction of activity associated with the particles due to iodine was lower than expected, which may have been due to initial fractionation and inability of the analytical procedure to remove iodine trapped within insoluble particles."

The present author believes these conclusions to be unjustified, for exactly the same reasons discussed previously under SEDAN.

However, a thorough study of the implications of the (separate) SMALL BOY total- α -ionization leaching experiments for this rebuttal could not be made, because those experiments were not as complete as the corresponding ones on SEDAN. In particular, the SMALL BOY Project 2.9 leaching experiments:

(1) Covered only the larger particle sizes, $88\mu\text{m} - 1410\mu\text{m}$, in 5 groups, and were thus less representative on a specific-activity basis, and also ignored the particle sizes which should have given the highest leach percentages.

(2) Never included more than one immersion duration for a given sample (station); thus trends with immersion duration could not be examined.*

* However, SMALL BOY Project 2.10 reported some trends, presumably for samples from stations other than those used in Lane's "volatilization" experiment. Freiling indicated that, in a total-fission-product leach experiment, water leachates took 2 to 5 days to reach equilibrium, while acid leachates took 2 to 8 days.

Nevertheless, the SMALL BOY Project 2.9 leaching results indicated that for sizes less than about $175\mu\text{m}$, leaching extent rose as particle size was reduced, so that the percent leached (by water) in the SMALL BOY particle sizes corresponding to those examined in SEDAN could have been 5-10%, as in the latter fallout.*

Finally, in the SMALL BOY Project 2.9 report there is confirming gamma-spectral evidence for the preferential leaching of iodine, similar to that discussed earlier under SEDAN. Again the filtrates from the total-mixture leach experiments (and either the residues, or the gross samples before separation) were examined on the spectrometer.

Figures 3.12 and 3.13** are spectra, taken at about H+12 hr, of the gross sample from one station (203) and the water filtrate thereof. The filtrate spectra appear to be very much enriched in I-133; a peak at about 0.53 Mev is the predominant one in the spectra. The same peak is much less important in the gross-sample spectra.*** There is no

* One difference between SMALL BOY fallout (Project 2.9) and JOHNNIE BOY/SEDAN (cratering event) fallout was that in the latter, leach extent was independent of pH, over the range 1-10, while in the former, leaching percentage in pH 1 solutions was more than a factor of 10 higher than that in solutions of pH 6-10, being as high as 60% for some samples. The reasons for the difference are not clear, but the effect is not relevant to the interpretation of the "volatilization" experiment, in which the results for pH 6 and 10 are the important ones.

** None of LaRiviere's spectra are reproduced here.

*** Conversely, peaks at about 0.65 Mev and 0.75 Mev, associated with Nb-97 and Nb-97m, respectively (both short-lived daughters of 17-hr Zr-97), are prominent (predominant) in the gross-sample spectra, but missing in the water-leachate spectra, a result that is consistent with the known insolubility of Zr/Nb in water.

evidence of the 0.364 Mev peak of I-131 in any of the spectra; that peak should be absent (hidden) at times as early as H+12 hr.

Figures 3.15 and 3.16 are spectra, taken at about D+1.2, of pH 1.0 filtrates from seven size fractions from the same station as above (203). All seven spectra at each spectrometer gain setting are strikingly similar, in terms of peak locations and relative intensities. In particular, all the spectra appear again to be enriched in I-133; the predominant peak (fairly sharp) is again at about 0.53 Mev.* As in the earlier spectra, there is no evidence of the 0.364 Mev I-131 photon. Gross-sample or residue spectra were not measured.

The remaining spectra (Figures 3.17-3.22) were all taken at about D+9, by which time I-133 would have decayed away and I-131 would be a major contributor to the total activity. The discussion following will therefore be keyed to the 0.364 Mev peak of I-131. Spectra were run for filtrates of pH 1.0, 6.0 and 10.0 and for the corresponding solid residues. Most of the samples examined (from a different station (403) than that of the earlier spectra) were from a sieve fraction in the 350-710 μ m range; a few were in the 710-1410 μ m range.

With the 350-710 μ m particles, the pH 6.0 and 10.0 filtrates were highly enriched in I-131. The peak at

* In this acid filtrate, a contribution from Nb-97 and Nb-97m can be clearly seen; this is an indication that the acid tends to dissolve or leach a representative fraction of the total activity, whereas water alone is much more selective.

b-29

about 0.36 Mev was intense and sharp; in fact, it was clearly the predominant peak in the spectra. In those neutral and alkaline solutions, the same peak from the larger particles was much less intense, but still clearly present. In the spectra of the solid residues from all these samples, the I-131 peak could not be distinguished.

When the 350-710 μ m particles and also the larger particles were leached in pH 1.0 solutions, the 0.36 Mev peak was greatly reduced in intensity; in fact somewhat displaced, so that perhaps some other photon or mixture of photons was being seen. Again, the spectra of the corresponding solid residues appeared ambiguous in the region of 0.36 Mev.

B.3.4. Summary (SMALL BOY)

No direct measurement of the volatilization rate of iodine from fallout particles was made for the SMALL BOY event. The indirect measurements (volatilization or air-exposure experiment) were subject to the same kinds of error discussed in detail under SEDAN; for exactly the same reasons, the claimed volatilization rates of about 2.4% per day are unsupportable. Total-mixture leach experiments in SMALL BOY support the conclusion that the claimed extent of volatilization during air exposure was really a differential extent of leaching, resulting from variation in thiosulfate-immersion duration during the iodine-recovery process.

It is not possible to state what the extent of volatilization from SMALL BOY particles actually was. Certainly, the upper limit is 2.4% per day, but this number (air-exposure experiment) could easily be as much as a factor

of 10^4 lower, as it actually was in SEDAN. Since the extent of release of the total-fission-product mixture, and the nature of release of individual radionuclides, were the same for SMALL BOY, JOHNIE BOY, and SEDAN particles in water-leach experiments, there is no reason to believe that the true volatilization rate of SMALL BOY particles was any higher than the 0.00025% per day determined for SEDAN particles.

B.4. Overall Summary

Based on the limited direct-measurement information available from the SEDAN event, and on supporting data from that event and from JOHNIE BOY and SMALL BOY, it is concluded that the iodine volatilization rate from (dry) siliceous fallout particles is of the order of 0.00025% per day. Even if this estimate is low by a factor of 1000, the rate would still be negligible (see discussion in text) in the context of a radioiodine inhalation threat in the general civil-defense shelter environment.

C-1

APPENDIX C

FUNDAMENTAL PROPERTIES OF IODINE ISOTOPES

For the five iodine radioisotopes of interest in this evaluation, all the important properties are shown in Figures C.1 through C.5 (I-131 through I-135, respectively).

The chain parameters are taken from Kochendorfer (1969). Chain yield, a function of the type of fission, measures the total number of atoms of the particular radioiodine ultimately produced (instantaneously and by decay of precursors), and is thus a first-order estimator of time-integrated activity in the thyroid. Note from the recorded chain yields that the second peak in the bi-modal mass-distribution curve for "thermonuclear"-neutron fission of U-238 occurs at mass 134, and is fairly flat. Thus all five of the radioiodines considered here are produced in significant yield.* Note also that for each mass chain, the chain yield (through iodine) is the sum of the indicated independent yields.

The half-life of the iodine isotope itself is a measure of the period during which that isotope is a threat, particularly when all its precursors have much shorter half-lives.

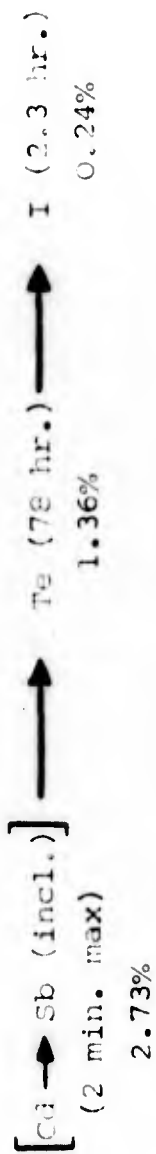
* No other radioiodines need be considered. I-128 and I-130 are shielded nuclides (that is, their tellurium "precursors" are stable) and they themselves are produced in insignificant independent yield. All the radioiodines of mass 126 and below are in the same category; that is, they are all shielded either by (1) stable tellurium "precursors"; or (2) earlier "precursors", with intervening nuclides produced in insignificant independent yield. I-127 is, of course, stable. I-129 has a half-life of 1.6×10^7 years, so is no threat. All the radioiodines of mass 136 and above have half-lives measured in seconds and all their precursors are extremely short-lived also, so none of these can survive long enough to be fallout threats.



<u>Iodine-131</u> Betas			
<u>E_{max}, Mev</u>	<u>Relative Abundance, %</u>	<u>Range in Tissue, μm</u>	<u>50%-Dose Radius, μm</u>
0.246	2.2		
0.332	10.6	1000.	150.
0.606	86.4	2100.	310.
0.807	0.8		

* Independent yields and chain yield are for U-238 fission by "thermonuclear"-neutron spectrum. Same for other isotopes following.

FIGURE C.1
IODINE-131 PARAMETERS



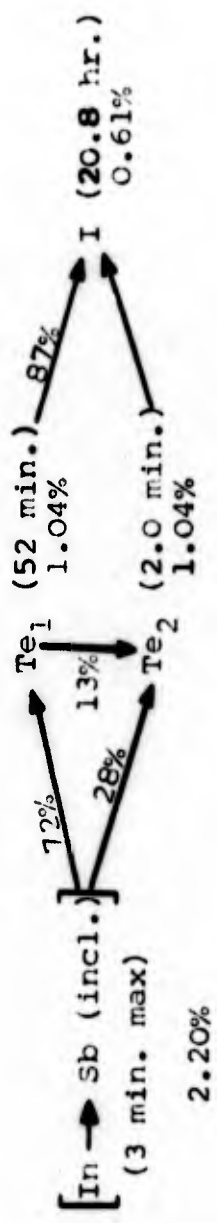
132-Chain Yield = 4.33%

Iodine-132 Betas

<u>E_{max}, Mev</u>	<u>Relative Abundance, %</u>	<u>Range in Tissue, μm</u>	<u>50%-Dose Radius, μr</u>
0.681	15.	2,500.	375.
0.934	20.	3,900.	625.
1.150	22.	5,400.	920.
1.583	22.	8,400.	1,500.
2.111	21.	11,900.	2,400.

FIGURE C.2

IODINE-132 PARAMETERS

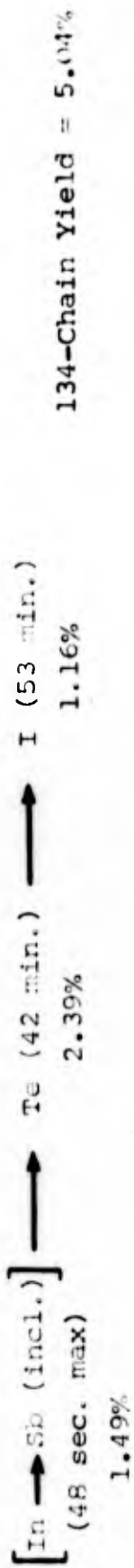


133-Chain Yield = 4.89%

<u>Iodine-133 Betas</u>			
<u>E_{max}, Mev</u>	<u>Relative Abundance, %</u>	<u>Range in Tissue, μm</u>	<u>50%-Dose Radius, μm</u>
0.49	7.	6800.	1200.
1.34	93.		

FIGURE C.3

IODINE-133 PARAMETERS



<u>Iodine-134 Betas</u>			
<u>E_{max}, Mev</u>	<u>Relative Abundance, %</u>	<u>Range in Tissue, μm</u>	<u>50%-Dose Radius, μm</u>
0.87	5.		
1.55	65.	8,100.	1,460.
2.65	25.	15,800.	3,300.
3.51	5.		

FIGURE C.4

IODINE-134 PARAMETERS



<u>Iodine-135 Betas</u>			
<u>E_{max}, Mev</u>	<u>Relative Abundance, %</u>	<u>Range in Tissue, μm</u>	<u>50%-Dose Radius, μm</u>
0.40	35.	1300.	195.
1.00	40.	4300.	690.
1.43	25.	7300.	1300.

FIGURE C.5

IODINE-135 PARAMETERS

Thus I-131 is important for about the first 40 days, I-133 for about 5 days, and I-135 for about 36 hours. I-134 with its 53 min half-life, preceded by 42 min Te-134, is important for perhaps 6 hours. The 132-chain presents a more complicated picture, in that the decay in this transient equilibrium is controlled by the 78-hr Te-132 parent, so that at any given time (say where inhalation of iodine vapor is being evaluated) only a small fraction of the ultimately available iodine exists as iodine*. Thus, for I-132 to produce a significant internal threat, compared to the other radiiodines, it must be taken in the form of the tellurium parent, and that tellurium must remain in the system for a time of the order of its own half life, at least. This problem is considered in more detail in Stewart and Simpson (1965) and in Mechali, et al. (1965 and 1966).

As indicated in the above paragraph, the independent yields of the iodine isotopes measure the extent to which decay of precursors is important in defining the ultimate iodine threat. In particular, as will be seen later in this Appendix, they have a profound impact on the phenomenon of fractionation.

The beta energies and relative abundances are taken from Miller (1957). In the context of radiiodine uptake, beta-ray absorption is the predominant contributor to exposure dose in the thyroid. Within the limits of accuracy required in the present study, it is generally safe to ignore the much smaller (additional) gamma exposure dose, particularly since it compensates to some extent for the loss of beta dose arising

* The independent yield of the 2.3 hr I-132 is very small; this, coupled with the short half-life, reinforces the importance of the transient equilibrium.

from the escape of beta rays originating near the surface of the thyroid.* Again because of the limited accuracy required (in the face of the great uncertainties with respect to volatilization and vapor diffusion, discussed in Section IV/Appendix B and in Appendix D, respectively), no attempt was made to obtain more recent decay-scheme data than that of Miller.

The 50%-dose radius is defined, for a point source in tissue, as the distance within which 50% of the dose, for that beta energy, is distributed. The data are from Loevinger, et al., in Hine and Brownell (1956). The significance of this parameter lies in the claim of Klassovskii, et al. (discussed in Appendix F) that the relatively weak betas of I-131 are not absorbed uniformly in the (vulnerable) parenchymous tissue of the thyroid, and therefore that I-131 is much less effective than the other radioiodines, on an Mev-for-Mev basis, in inducing pathological states in the thyroid.

It has been emphasized in this Appendix and elsewhere in the report that the tremendous uncertainty as to the extent and rate of volatilization and the wide variability in dispersion of iodine vapor, once formed from ground-deposited fallout particles, justify an eclectic approach to the assembling of other input data. Thus, in Section IV of the main report, the calculations of Mechali, et al. (1966) for thyroid doses resulting from inhalation of the gross-fission-product mixture have been applied to the present evaluation. Those calculations had been based on fission of plutonium-239 by fast neutrons. Table C.1, retyped from Table I of Kochendorfer (1969), shows the variability in the various radioiodine activities (dis/sec basis) for a range of fission types, the basis of comparison being fission of U-235 by thermal neutrons. Note

* The simplest calculation of beta dose assumes that all the emitted beta energy is absorbed in the thyroid.

TABLE C.1

MULTIPLIERS (RATIOS) FOR ADJUSTING IODINE ACTIVITIES FROM U-235TH FISSION TO FIT OTHER FISSION CASES

Isotope	Post-Burst Time Range	Activity Ratios for the Fission Cases:		For Comparison Pu-239TH U-235TH					
		Calculated from Computed Activities	Partly Computed from Activities, Partly Estimated *						
		U-235FI U-235HE U-238TN Pu-239FI U-235TH U-235TH U-235TH	U-238FI U-238HE Pu-239HE U-235TH U-235TH U-235TH						
I-131	1h-60d	1.28	1.42	1.35	1.58	1.08	1.67	1.24	1.29
I-132	1h	1.15	2.21	1.33	1.66	0.10*	0.20*	2.50*	1.68
"	6h	1.15	1.36	1.07	1.29	0.80*	0.90*	1.32	1.30
"	12h	1.15	1.11	0.99	1.19	1.09	1.12	0.99	1.25
"	1d-30d	1.15	1.05	0.98	1.16	1.08	1.10	0.86	1.16
I-133	1h-14d	0.86	0.80	0.76	0.89	0.82	1.01	0.73	1.05
I-134	1h	0.76	0.71	0.67	0.79	0.83	0.76	0.66	0.94
"	18h	0.76	0.61	0.71	0.73	0.99*	0.86*	0.52*	0.88
I-135	1h-3d	1.02	0.74	0.90	0.96	1.04	0.91	0.69	1.06

U-235TH = from fission of U-235 by thermal neutrons.
 U-235FI = from fission of U-235 by fission-spectrum neutrons.
 U-235HE = from fission of U-235 by 14 Mev (high-energy) neutrons.
 U-238TN = from fission of U-238 by thermonuclear neutrons.
 U-238FI = from fission of U-238 by fission-spectrum neutrons.
 U-238HE = from fission of U-238 by 14 Mev (high-energy) neutrons.
 Pu-239FI = from fission of Pu-239 by fission-spectrum neutrons.
 Pu-239HE = from fission of Pu-239 by 14 Mev (high-energy) neutrons.
 Pu-239TH = from fission of Pu-239 by thermal neutrons.

* Estimated from Independent Yields

that for the four radioiodines of major interest (I-131, 132, 133 and 135), and for all times after about 1 hr after fission, any kind of fission can be substituted for U-235TH with an error never worse than 67%, and usually no worse than 20%. Of particular interest with respect to the use of Mechali's results, the (derived) ratio Pu-239FI/U-238TN ranges from 1.03 to 1.25, over all times.

The chain parameters shown in Figures C.1 through C.5 have a profound effect on the radioiodine loading of arriving fallout particles, in terms of total activity, composition (with respect to mass chain), and location within the particles; the last, in turn, determines the extent of volatilization, as discussed in Appendix B, Section B.2.2. Radioiodine loading is controlled primarily by fractionation during fallout formation and transport.* The physical states of the very-short-lived tin and antimony precursors of iodine during particle formation are quite sensitive to the condensation temperature of the

* There is a rich literature on fractionation. In the list of references, see entries under Adams, C.E., Crocker, Freiling, Kawahara, Korts, Miller, C.F., Norman and Pascual (as sole or senior authors). Miller developed a model based on thermodynamics in the condensing fireball, extent of condensation of chains depending on Raoult's Law constants for the pertinent chain members. For application to the prediction of exposure rates, Freiling's group depended primarily on the systematics (correlation) of field data on fractionation parameters. Their predictions were based on 3 groups of mass chains: volatile, intermediate and refractory, and their approach to prediction involved parameterization of key fractionation variables for the volatile group. Norman and his colleagues adopted a kinetic approach, the output being a computer model which specifies not only quantities of each chain condensed, but also radial penetration of key chain members into particles of specified size. While that model provides a starting point for estimation of the reverse process, namely the volatilization of iodine from such particles, there appears to be no useful theoretical method for following the outward migration processes at ambient temperatures.

fallout-particle matrix in the range 1600-2000°K (Crocker, 1968). (The earlier precursors, cadmium and indium, are considered to be refractory in the formation process). Thus, radioiodine loading depends strongly on independent yields of tin and antimony (and, of course, of the much-more-volatile tellurium and iodine) in each of the four mass chains (131, 132, 133 and 135) of concern.* Furthermore, to the extent that volatilization (after condensation) occurs at all, some of the radioiodine formed by decay during fallout transport may be lost to the upper atmosphere before arrival of fallout on the ground.

In its formation-fractionation properties, iodine (affected mainly by its precursors) is intermediate in its behavior between the so-called volatile chains, such as the 137-chain, culminating in Cs-137, and the so-called refractory chains, such as 95 (Zr-95/Nb-95) or 99 (Mo-99). Fractionation is measured by the R-number (defined in Appendix B, Section B.2.2). For Small Boy, a low-yield surface detonation on siliceous soil, $R_{137,95}$ for close-in fallout was about 0.075; that is, Cs-137 was found in close-in fallout particles to about 7.5% of the extent predicted if it were to follow the refractory 95-chain, taken as the reference chain. In gross samples of close-in fallout from the same event, $R_{131,95}$ ranged from 0.25 to 1.65; that is, some samples were "depleted" of I-131, others "enriched". In sieve samples from the same locations, the larger sizes were depleted of I-131, the smaller sizes (relatively) enriched. Specifically,

Larger than 44 μm :	$R_{131,95}$	=	0.15 - 0.90
Smaller than 44 μm :	$R_{131,95}$	=	0.53 - 1.50.

* Five, for early arrival from kiloton-yield bursts, where the 134-chain also may have to be taken into account.

The ¹³¹I-chain in close-in fallout from megaton-range explosions, with their much higher condensation temperatures, would be expected to be much more "volatile" (in the formation process) than that from the low-yield Small Boy event. Therefore, in the civil-defense context, calculation of particle iodine loading on the basis of the unfractionated mixture is almost certain to overestimate the threat.

More subtle implications of formation fractionation, such as its effects on the relative loadings of the radioiodine-containing chains, are examined in Appendix B, Section B.2.2.

APPENDIX D

LEVELS OF RADIOIODINE VAPOR IN SHELTER.
A METHODOLOGY AND SOME RESULTS

In Section IV, it has been emphasized that the main potential sources of gaseous radioiodine within a shelter are: (1) fallout particles trapped in the particle filters or ducts of the ventilation system (these trapped particles may then release iodine vapor into the intake air subsequently flowing over them); and (2) fallout particles deposited outside the shelter (volatilization from these particles will depend on the action of sun and wind; the gaseous radioiodine so produced will be dissipated to some extent by turbulent diffusion in the atmosphere before reaching the shelter ventilation intake).

In this Appendix, the trapped-particle source will be considered first; the threat from the outside source will then be treated as an extension of the "inside" threat.

D.1. Particle-Iodine Loading of Ventilation System

Estimation of the amount of radioiodine in particles entering a shelter ventilation system requires specification of the radioiodine volume concentration for the arriving fallout in the air at the ventilation intake (for example, in curies of total particle radioiodine per cubic foot of air), as a function of time. With a worst-case assumption that all particle sizes present are swept into the intake, one need then specify only a ventilation rate (for example in cfm, or in cfm per person) to determine total ingress of particle radioiodine (including hold-up on conventional

filters), again on either a total or a per-person basis. The radioiodine-vapor threat to shelter occupants can then be assessed by means of assumptions as to the volatilization rate of iodine in the subsequent air flow. As it turns out, the combination of limited ingress of particles and low volatilization rate once they are trapped means that the "inside" threat is almost inconsequential. However, the methodology developed to evaluate that threat provides a direct means for evaluating the "outside" threat, which under some circumstances may be considerable.

D.1.1. Methodology

The methodology developed for computation of particle-radioiodine volume concentration in arriving surface-burst fallout vs. time* was based on the unclassified report literature on fallout phenomenology (Clark and Cobbin, 1963) and on fission-product radioiodines (Kochendorfer, 1969). The former provided: (1) data on stabilized-cloud parameters and on "standard" exposure rate as a function of distance downwind along the hot-line; and (2) a basis for determining minimum and maximum particle sizes on the ground, and arrival time as a function of particle size, at the corresponding locations; all as functions of weapon yield. A 15 mph effective wind speed was the basis for the "standard" exposure rates and particle-sizes/arrival-times on the ground. The latter report provided data on the time dependence of the activity of each individual radioiodine and of the total radioiodine mixture, as related to the "standard" exposure rate. For worst-case considerations, the unfractionated radioiodine mixture was used in the calculation. Fractionation as a

* The author is indebted to Mr. Samuel C. Rainey (present address: Naval Ship Research and Development Center, Carderock, Md.) for this work (Sequences I - XVIII below).

factor in reducing the threat, if any, could then be handled semi-quantitatively, since the uncertainties in volatilization rate override all other uncertainties by far.

A schematic picture of the methodology appears in Figure D.1. The following discussion is keyed to the Roman-numeral sequence in that Figure. The equations used are presented without derivation. At appropriate points, intermediate calculational results for an example problem are presented.

I

In the example problem, the location of interest was a point, P, on the hot-line, 58.5 mi downwind from ground zero for a 10 MT detonation at the surface; the predicted maximum "standard" exposure rate of 9,060 R/hr at 1 hr occurs at this point. Per Clark and Cobbin, this is for a 100%-fission detonation; for the more-reasonable 50%-fission detonation at the megaton-range yield, the corresponding exposure rate would be 4,530 R/hr at 1 hr. Furthermore, the recorded figures are based on "...the AN/PDR-39 (T1B) portable radiac which has a geometric and photon energy response very close to 0.75 of the true air ionization rate 3 feet above a plane source of fission products uniformly distributed on the area". Therefore, the true (air-ionization) "standard" exposure rate is $4,530/0.75$, or 6,050 R/hr at 1 hr, for 50% fission.

II

The cloud geometry is shown in Figure D.2, in which:

- h_o is the height of the (stabilized-) cloud center, the idealized cloud being an ellipsoid of revolution.
- a and b are the semi-major and semi-minor axes of the cloud, respectively.
- v_w is the (effective) wind speed, always 22 ft/sec (15 mph).

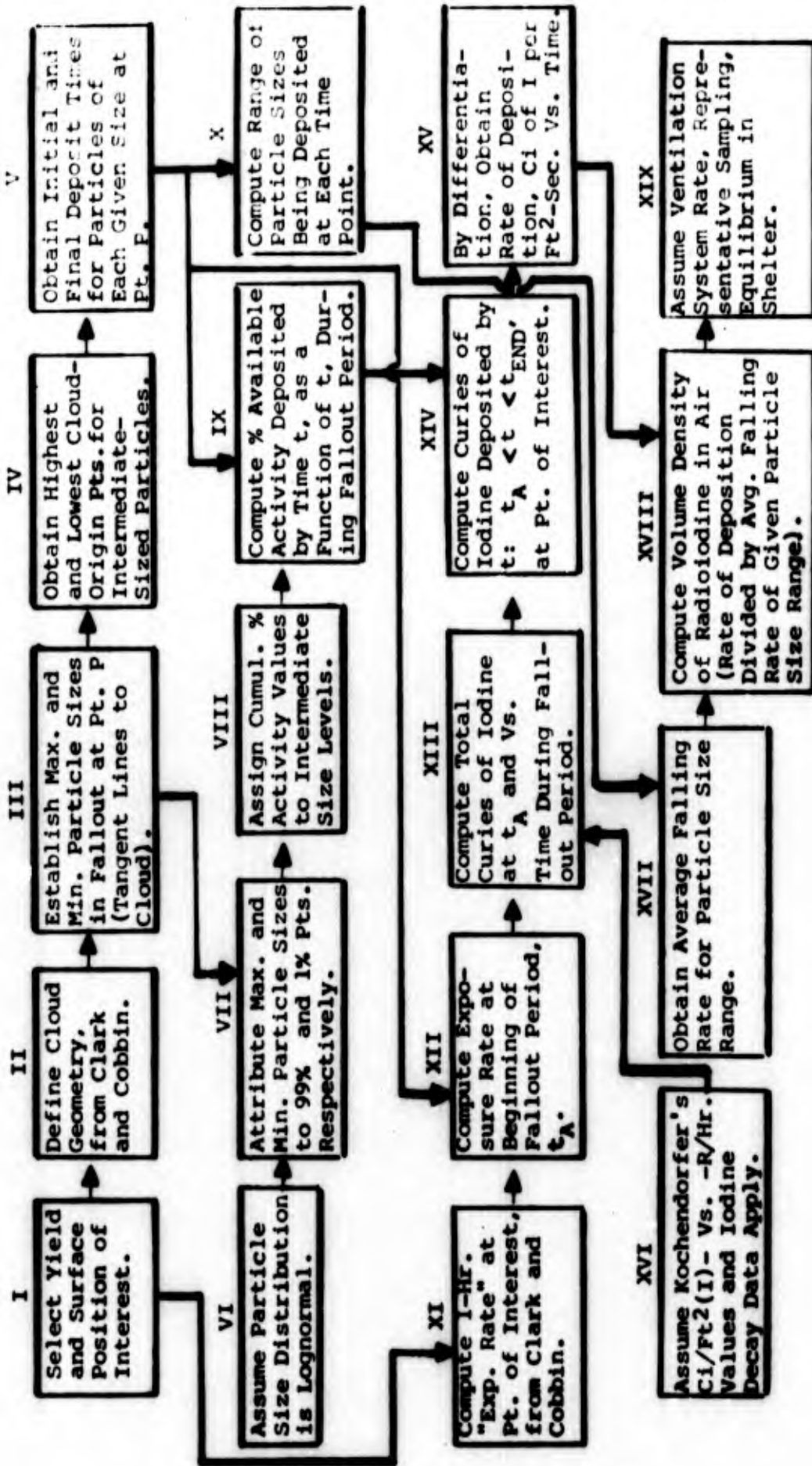


FIGURE D.1

EVALUATION OF INHALATION HAZARD FROM AIRBORNE PARTICULATE MATERIAL CONTAINING RADIOIODINE

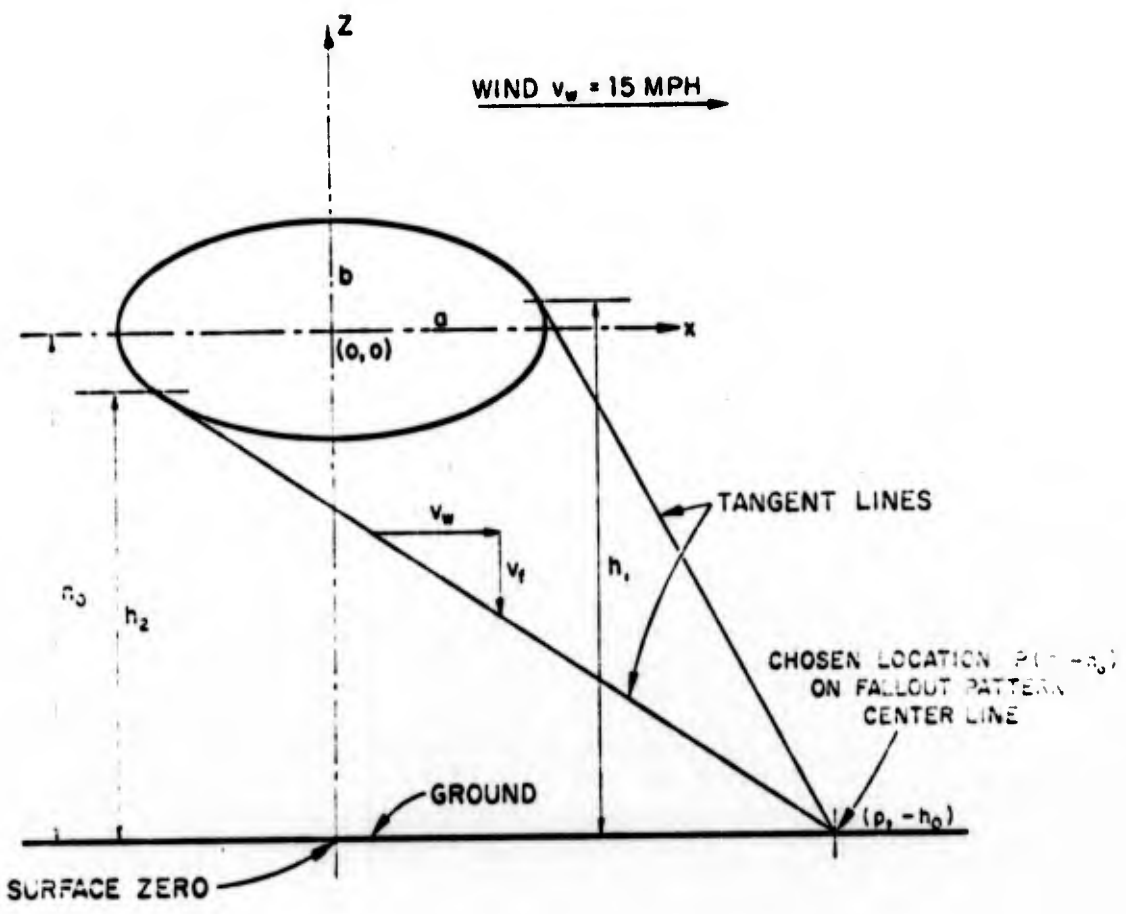


FIGURE D.2

GEOMETRY OF SIMPLIFIED MATHEMATICAL MODEL OF FALLOUT ARRIVAL FROM CLOUD

- V_f is the "effective terminal velocity" (i.e., the average falling velocity) for a particle of a given size, starting at a particular elevation (in ft/sec).
- p is the distance from ground zero to the location of interest.

For convenience, particles of a specified size, originating at a specified altitude, are treated as having a constant falling velocity, thus a straight-line trajectory. This introduces no error in arrival time at a location on the ground, since an effective (average) fall velocity for the total vertical travel is used. The two tangent trajectories shown in the figure are those for the largest particles to arrive at point P (from altitude h_1) and the smallest (from altitude h_2). Note that these are not the earliest and the latest arrivals, respectively; the earliest particles to arrive come from the leading edge of the cloud, while the last to arrive come from the trailing edge.

With the yield, W , in KT, we have:

$$\begin{aligned} a &= 2.45 \times 10^3 W^{0.431} = 129,800 \text{ ft (example problem)} \\ b &= 1.40 \times 10^3 W^{0.300} = 22,200 \text{ ft} \quad " \quad " \\ h_0 &= 1.68 \times 10^4 W^{0.164} = 76,100 \text{ ft} \quad " \quad " \end{aligned}$$

Thus, the 10 MT cloud is about 50 mi across and about 8 mi high (thick) centered at an altitude of about 15 mi.

In the example problem, p was chosen as 307,600 ft, or 58.5 mi.

A useful parameter in the characterization of a trajectory is α , defined as V_w/V_f , or the reciprocal slope of the trajectory. Each relevant combination of particle size and originating altitude has associated with it a value of α . For all situations considered here, $\alpha = 22./V_f$.

III

In particular, the maximum and minimum particle sizes encountered at point P (each coming as it does from only one altitude) are characterized by α_{\min} and α_{\max} , respectively,

where:

$$\alpha_{\max} = \frac{-h_0 p + \sqrt{b^2 p^2 - a^2 (b^2 - h_0^2)}}{b^2 - h_0^2}$$

and α_{\min} is given by the same expression, with the negative value of the radical.

The maximum and minimum particle sizes at P are then found by the following procedure:

1. Having determined a , b , h_0 and p , compute α_{\max} and α_{\min} .
2. Determine the z coordinates of the points of tangency shown in Figure D.2. For each of the two values of α ,

$$z_{\text{tang}}^* = -b^2 B/A$$

$$\text{where } A = b^2 + (a^2/\alpha^2)$$

$$\text{and } B = h_0 - (p/\alpha).$$

Thus, h_1 (Fig. D.2) is given by $(h_0 + z_{\text{tang}}(\alpha_{\min}))$, and h_2 by $(h_0 + z_{\text{tang}}(\alpha_{\max}))$.

3. Based on tabulated information in Clark and Cobbin, make a plot of $22/V_f^{**}$ (or α) vs. particle size, for heights h_1 and h_2 . By interpolation, find the particle sizes

* As calculated, z_{tang} is the vertical distance between the point of tangency and the cloud vertical center line (plane). Therefore, the actual altitude is $(h_0 + z_{\text{tang}})$. Note that $|z_{\text{tang}}| < b$. For the extremes in the altitudes of origin of intermediate-size particles, the vertical displacements from the center plane will be designated by z .

** Values of V_f are found in Clark and Cobbin's Table A.1.

conforming to α_{\min} and α_{\max} , respectively. These are then, respectively, the largest and the smallest particle sizes arriving at point P.

4. Note that, for many specific situations of interest, Clark and Cobbin provide data directly on the maximum and minimum particle sizes arriving at a surface location (and on related parameters) (their Table C.2).

In the example problem, the key values are:

<u>Largest Particles</u>	<u>Smallest Particles</u>
Size = 240.7 μm	Size = 108.6 μm
V_f = 9.93 ft/sec	V_f = 3.33 ft/sec
α_{\min} = 2.22	α_{\max} = 6.60
h_1 = 84,030 ft	h_2 = 59,490 ft
$t(\text{arr.})$ = 8,465 sec*	$t(\text{arr.})$ = 17,860 sec*

IV

The first step in determining highest and lowest cloud-origin points is to compute, for a series of values of α in the range $\alpha_{\min} \rightarrow \alpha_{\max}$:

$$z = \frac{-b^2B \mp (ab/\alpha) \sqrt{A-B^2}}{A}$$

in which all symbols have been defined. Then, plot $(h_0 + z)$ vs. α . Since z is double-valued for each value of α (except α_{\min} and α_{\max}), a plot of height of origin, $(h_0 + z)$ vs. α will be a closed (ovoid) figure, bounded by $(h_0 + b)$, $(h_0 - b)$, α_{\min} and α_{\max} . This plot, for the example problem, is shown in Figure D.3.

* Note that the time of arrival for the earliest particles to arrive (t_a) is given by $(p - a)/V_w$, and that for the last particles to arrive (t_c) is given by $(p + 2a)/V_w$. For the example problem, t_a is 8,080 sec, t_c is 19,870 sec. The corresponding sizes are 238.4 μm and 114.1 μm (both originating at altitude h_0 (vertical center plane of cloud). In Figure D.1, t_a and t_c are denoted t_A and t_{END} , respectively.

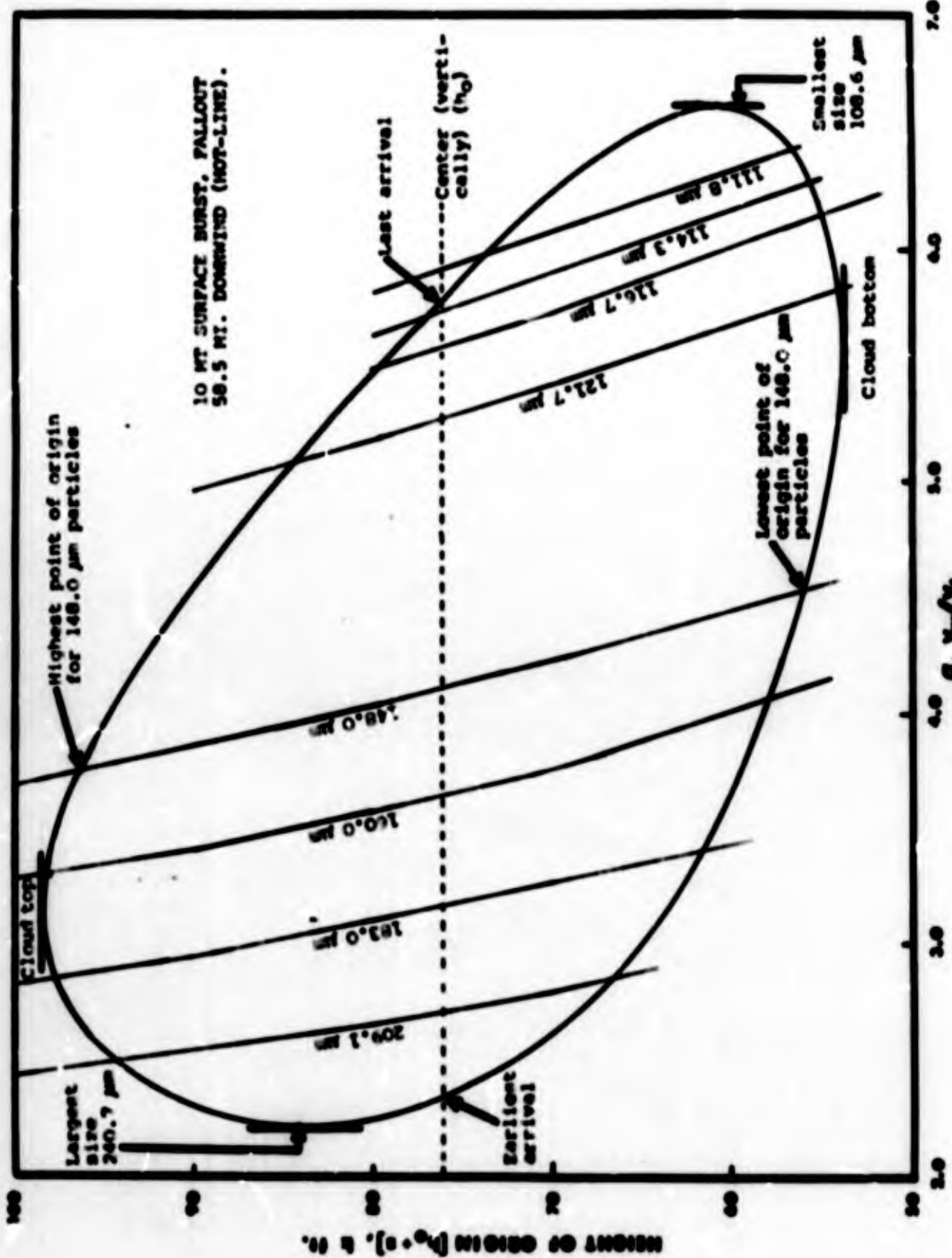


FIGURE D.3
HEIGHTS OF ORIGIN VS. σ , FOR A FAMILY OF PARTICLE SIZES

Next, for each of a series of particle sizes in the range arriving at point P, determine (from Table A.1 in Clark and Cobbin) the value of α (or $22V_f$) associated with each of a series of altitudes of origin in the range $(h_0 - b)$ to $(h_0 + b)$. For each such particle size, plot height of origin vs. α , on the plot of Figure D.3. The points of intersection of each such curve with the closed (ovoid) figure represent the lowest and highest altitudes of origin of the corresponding particle size. Again, the family of superimposed curves shown in Figure D.3 is for the example problem.

V

For each of a range of particle sizes of interest, the initial and final deposit times can be determined from the information in Figure D.3. The procedure follows:

For each particle size, minimum and maximum altitudes of origin have been determined as points of intersection, in IV above. Call these z_{\min} and z_{\max} , respectively. Since values of α for these heights may be read from Figure D.3, recompute V_f at the points of intersection (as $22/\alpha$). Then, for each particle size, start and finish are given by

$$t_s = z_{\min} \sqrt{V_f(\min)}$$

and
$$t_f = z_{\max} \sqrt{V_f(\max)}.$$

Finally, plot the pair of time extremes vs. particle size; this again produces a closed figure, this time banana-shaped. The plot, for the example problem, is shown in Figure D.4. Note that a vertical cut through the figure gives the time range over which a particular size is arriving, while a horizontal cut gives the range of particle sizes arriving at a particular time.

If, for a given time, s_a represents the smallest size arriving and s_b represents the largest size arriving, then s_b is the minimum particle size that is all deposited by that time. That is, all particles larger than s_b are down; in addition, some fraction of each size between s_a and s_b is already down also.

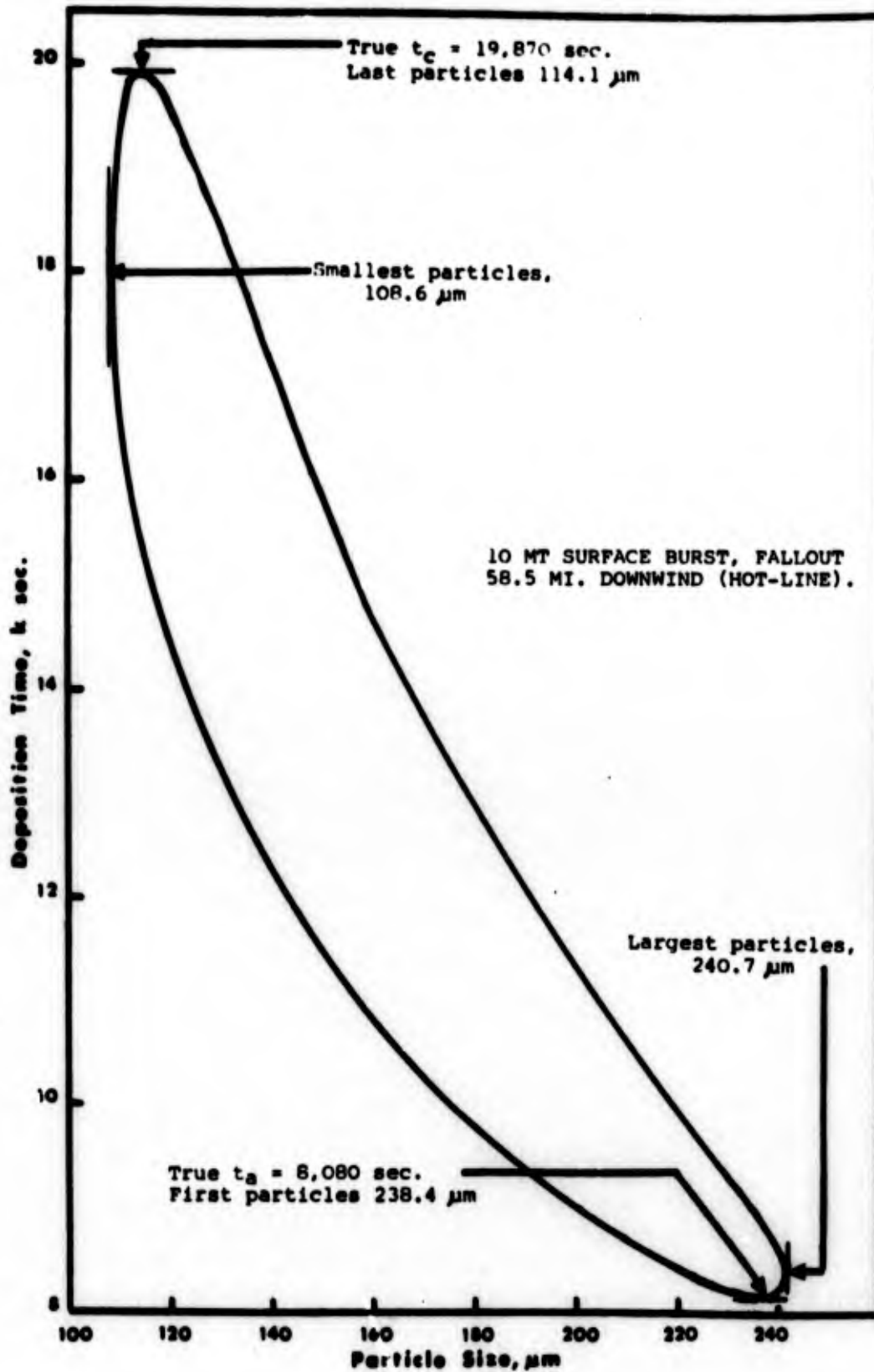


FIGURE D.4

ARRIVAL DURATION AS A FUNCTION OF PARTICLE SIZE

VI, VII AND VIII

The activity/particle-size distribution plot is generated on two-cycle log probability paper, with the largest size arriving plotted at the 99% (cumulative) point, the smallest at the 1% point, and a straight line connecting these points. The distribution for the example problem (not shown) was generated in this fashion. For that problem, the distribution used implied a "range" (1% to 99%) of 108.6 μm to 240.7 μm , with 50% of the activity contained in particles smaller than 160 μm .*

IX

The calculation of percent of activity down (on the ground) as a function of time since t_a is based on the results of V and VIII. At each time, the activity already down is made up of two components: (1) that associated with s_b , the minimum size that is all deposited; and (2) that associated with the range of sizes depositing at that time, namely s_a through s_b . The activity associated with group (1) can be read directly from the log-normal distribution curve; the activity associated with group (2) is determined by linear interpolation in Figure D.4 and in the distribution curve. For convenience, activity can be decay-corrected to the time of arrival of the earliest particles, t_a . The decay-corrected activity-deposition plot for the example problem is presented in Figure D.5.

X

As mentioned in the discussion under V, horizontal sections of Figure D.4 give the ranges of particle sizes arriving at time points of interest.

XI

As shown in the discussion under I, the 1-hr true-air-ionization ("standard") exposure rate in the example problem is 6,050 R/hr (50% fission). The value is now compatible with Kochendorfer's formulation.

* Implicit in the treatment is the assumption that there is no fractionation with particle size (at least that all sizes arriving at point P contain the same mixture of the fission-product radionuclides).

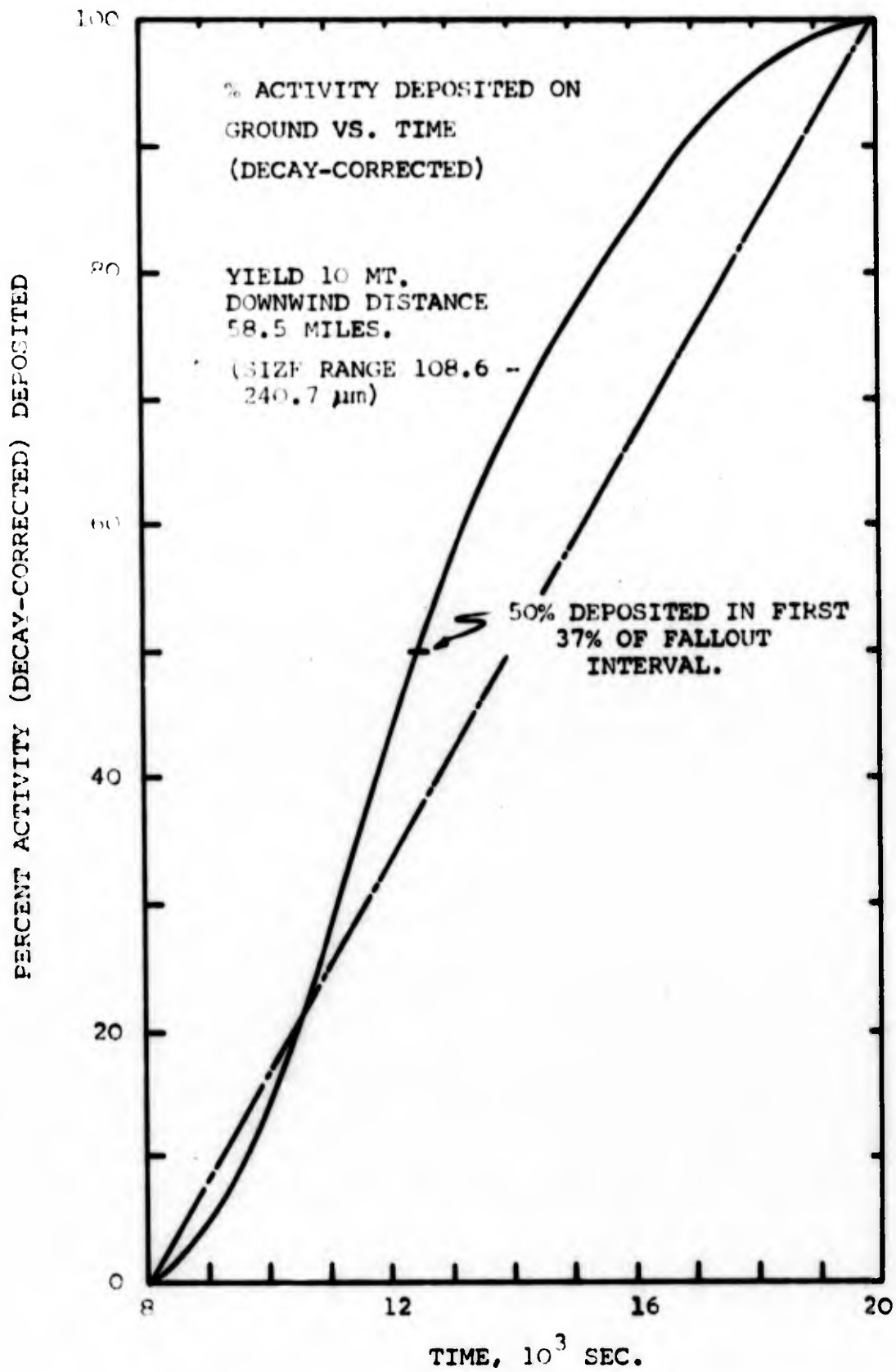


FIGURE D.5

XII

Calculation of the exposure rate at t_a is based on the $t^{-1.2}$ decay law. In the example problem, with $t_a = 8,080$ sec (2.24 hr), the (hypothetical) exposure rate at t_a is 2,300 R/hr.

XIII (XVI)

"Total curies" of iodine at t_a (and during the fallout period) is a hypothetical measure, like "standard" exposure rate. It represents the deposited iodine activity, per square foot, calculated as if all the fallout eventually deposited had actually arrived by t_a . It is determined as follows:

Kochendorfer gives factors for converting R/hr at given times after burst to curie/ft² of total radioiodine. From his Figure 24, the appropriate values for thermal-neutron fission of U-235 are obtained. At $t_a = 8,080$ sec, the factor is 7.0×10^{-4} curies/ft² per R/hr. Thus, for an exposure rate of 2,300 R/hr at t_a , the "total curies" are 1.61 Ci/ft² (at t_a).

If actual curies during fallout arrival are needed (only so if dose during fallout arrival is to be calculated), then the decay information in Kochendorfer's Figure 1 can be used to generate a correction-factor curve for total-iodine decay, normalized to 1.0 at t_a . In the example problem, the decay factor at t_c is 0.34.

XIV

Actual curies of iodine (per ft²) deposited by time t during fallout arrival are of interest only where dose during fallout arrival is to be calculated. Otherwise, the only time point of interest is t_c . For each time, t , curies deposited (per ft²) are obtained by multiplication of percent of available activity deposited (from IX) by total curies available (per ft²) (from XIII). Since arrival and decay are competing functions, the curies-deposited function peaks during the fallout-arrival period. In the example problem, the "peak" is fairly flat, covering the range 15,000 - 18,500 sec.

If the only calculation to be made is of the ultimate ventilation-system loading (i.e., dose calculations to start after fallout cessation), then it is more convenient to use a decay-corrected (normalized) value for the curies per ft². In this case, the decay function generated in XIII is not used; i.e., the results of IX (plus the one conversion at t_a) are applied directly in this and succeeding steps.

XV

The rate of deposition (in C_i/ft^2 per sec) is obtained by differentiation of the deposit curve of XIV. Where the actual C_i/ft^2 (the peaked curve) are used, the rates peak and then become negative toward the end of the arrival process. On the other hand, where the decay-corrected C_i/ft^2 (monotonic-increasing deposit curve) are used, the rates peak and then approach zero, but never go negative. In either case, piecewise differentiation is used; that is, $\Delta(\text{activity})/\Delta t$ is computed for a series of (discrete) time increments.

XVI

See discussion under XIII.

XVII

The time base for the average-falling-rate calculations is the series of intervals used in the $\Delta A/\Delta t$ calculations under XV. For the center of each such interval, t_1 , the particle size range being deposited at that time is obtained (from X, or Figure D.4, as an example). Then, Table A.1 in Clark and Cobbin is used to provide values of V_f (for the final 10,000 ft of fall, as opposed to the average V_f for the whole trajectory) for the smallest and the largest particles reaching the surface at point P at t_1 . The two fall velocities are then averaged to provide an average terminal velocity for the "parcel" of particles reaching the ground at point P during the interval centered on t_1 . If the intervals are short enough, this averaging process provides an excellent approximation to the continuous-arrival phenomenon*.

* In the example problem, ground-level fall rates range from 2.55 ft/sec for the smallest particles to 6.40 ft/sec for the largest. The average fall rate at a time half-way through the arrival period is 3.65 ft/sec. For the time at which 50% of the ground deposit is reached (Figure D.5) the average fall rate is 4.08 ft/sec. A suggested overall-average fall rate for the example problem is therefore 3.9 ft/sec, for a simplified form of the calculation.

XVIII

In an interval centered on t_i , the deposition rate $(\Delta A / \Delta t)_i$ (in Ci/ft² per sec) results from the depletion, at an average particle fall rate $(\bar{V}_f)_i$ (in ft/sec), of radioiodine-loaded particles from ground-level air whose radioiodine (volume) density is R_i (in Ci/ft³). Therefore, in each interval for which $(\Delta A / \Delta t)_i$ and $(\bar{V}_f)_i$ have been determined (in XV and XVII, respectively), R_i can be determined by:

$$R_i = (\Delta A / \Delta t)_i / (\bar{V}_f)_i$$

Depending on how $(\Delta A / \Delta t)_i$ has been computed, R_i can be expressed in terms of true activity per unit volume, or in decay-corrected activity per unit volume. In the remaining discussion, the latter basis will be used.

For the example problem, with activity decay-corrected to t_a , the radioiodine volume density ranged from 0.7×10^{-5} to 6.0×10^{-5} Ci/ft³, with the highest values occurring over a fairly wide range near the center of the arrival period.

XIX

With the particle radioiodine density in the air at the ventilation-system intake known (as a function of time), and with the worst-case assumption that all particle sizes present are swept into the intake, all that is needed is to specify a ventilation rate (most conveniently in cfm per person) and then the ingress of particle radioiodine into the ventilation system can be calculated. The extent of radioiodine inhalation by individuals then depends on the rate of volatilization of iodine from the trapped particles and on the individual breathing rate. If ventilation air is not recirculated, the calculation is straightforward, and the threat from trapped particles is minimized.

Shelter ventilation requirements and systems have been studied extensively. Ventilation rates are generally controlled

* See, for example, Allen (1970 and 1970a); Anderson, Jago and Friedman (1970); Baschiere and Lokmanhekim (1967); Kapil, Sitko and Buday (1969); Kapil and Rathmann (1971); Office of Civil Defense (1969); Rathmann (1969); Spiegel (1968); Strobe (1965); and Wright, Hill and Sawyer (1970).

by environmental considerations (cooling, dehumidification and removal of CO₂) rather than by normal breathing rates. The mean value of the breathing rate for adults, averaged over a 24-hr period, is $2.31 \times 10^{-4} \text{ m}^3/\text{sec}$ (International Commission on Radiological Protection, 1959/1960), which translates to 0.49 cfm. Spiegel (1968) has pointed out that 3 cfm per person are required to keep the CO₂ concentration below dangerous levels in a shelter situation; he recommends a ventilation rate of at least 5 cfm per person for emergency operation centers (EOC's), where alertness is required. Most codes for normal building occupancy specify ca. 10-15 cfm per person. Baschiere and Lokmanhekim (1967) have indicated that in hot and humid CONUS regions, ventilation rates as high as 40 cfm may be needed in shelters during the summer months.

In the example problem, a ventilation rate of 5 cfm has been used for calculation of the ingress, per person, of radioiodine on particles into the ventilation system. In the calculation of subsequent inhalation of volatilized radioiodine, it has been assumed that the ventilation rate remains at 5 cfm and that the adult breathing rate is 0.5 cfm. Note that as long as the ventilation rate per person remains the same after fallout cessation as it was during the arrival period, the potential inhalation of radioiodines per person is independent of that rate (except insofar as a higher flow rate may enhance percent volatilization per unit time). That is, a higher ventilation rate will introduce more particle radioiodine (per person) into the ventilation system, but the fraction of volatile radioiodine subsequently inhaled will be correspondingly reduced by dilution, at least where there is no air recirculation.

In the calculation of total ventilation-system loading of particle radioiodine, the average volume density of radioiodine (decay-corrected Ci/ft³) in each arrival increment is multiplied by the length of that interval and by the ventilation rate, to give decay-corrected curies of total radioiodine deposited in the ducts, etc. during the interval. Summation over all arrival intervals gives the total loading, in decay-corrected Ci/person. That is,

$$\begin{array}{l} \text{Incremental Particle} \\ \text{Loading} \\ \text{(Ci/person)} \end{array} = \begin{array}{l} R_1 \\ \text{(Ci/ft}^3\text{)} \end{array} \times \begin{array}{l} \Delta t_1 \\ \text{(min)} \end{array} \times \begin{array}{l} \text{Ventilation} \\ \text{Rate} \\ \text{(cfm/person)} \end{array}$$

and

$$\begin{array}{l} \text{Total Per-person} \\ \text{Loading} \end{array} = \begin{array}{l} \text{cessation} \\ \sum_{i=1} \end{array} \text{(Incremental per-person loading)}_i$$

For the example problem, the total per-person I loading so calculated was 33.0 mCi (decay-corrected to t_a)*. The shape of the trapping history (decay-corrected) for that situation is shown in Figure D.6. The non-decay-corrected trapping history can be determined by point-by-point multiplication by the decay function generated under XIII.

The total per-person loading calculated for the example problem represents a worst-case situation for the trapped-particle threat, at least for fallout from a single detonation. The inhalation implications of that and related situations are considered in Section IV of the main report.

* For the example situation, at least, a simpler approximation method gives a total loading very close to that calculated above. From XIII, we have a total ground deposit of 1.61 Ci(I)/ft² (decay-corrected to t_a). The arrival interval is 19,870 - 8,080, or 11,790 sec (from III). The overall-average fall velocity is 3.9 ft/sec (from XVII). Therefore, the time-averaged volume concentration of particle radioiodine in ground-level air is:

$$\frac{1.6}{11,800 \times 3.9} = 3.5 \times 10^{-5} \text{ Ci/ft}^3 \text{ (decay-corrected to } t_a \text{)}$$

$$= 35 \mu\text{Ci/ft}^3 \text{ (decay-corrected)}$$

With a ventilation rate of 5 cfm/person, the total per-person loading of particle radioiodine in the ventilation system is:

$$35 \times (5/60) \times 11,800 = 34 \times 10^3 \mu\text{Ci, or } 34 \text{ mCi}$$

(again decay-corrected to t_a)

This value is close enough to the more-exact value to warrant simplification of the procedure, especially in view of the major uncertainties discussed in Section IV in the text.

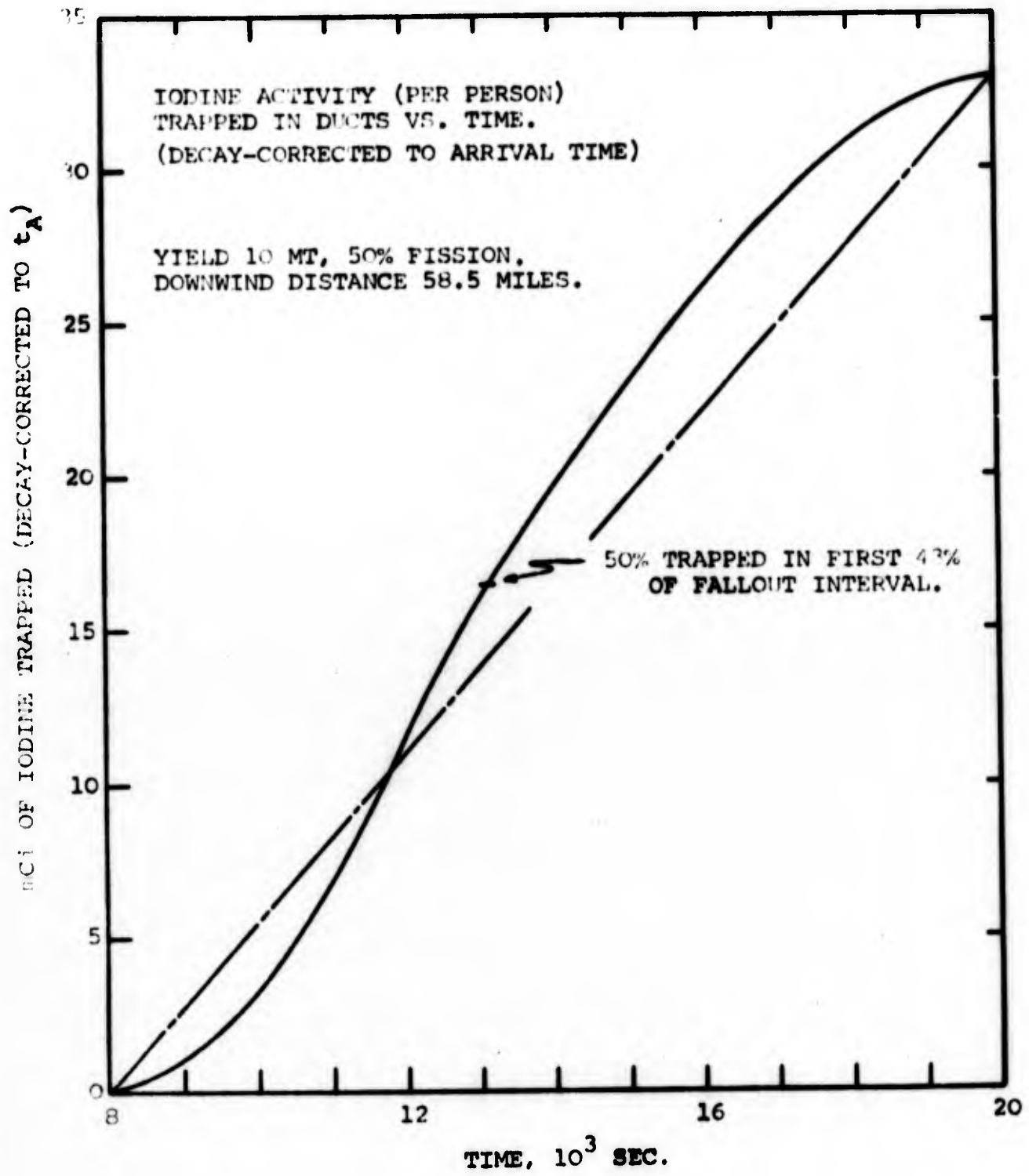


FIGURE D.6

D.2. Equivalent Ground-deposition Area "Sampled" by Ventilation System

The relative importance of particle trapping can be visualized in another way; namely, by calculation of the equivalent ground-deposit area for the total radioiodine trapped in the ventilation system, per person. In the example-problem situation, the question is: In a fallout field with a "standard" exposure rate of 6,050 R/hr at 1 hr, how much ground area contains the same amount of particle radioiodine as the ventilation system traps per person?

With all activity measurements decay-corrected to t_a , we have:

- a. On the ground by t_c : 1.61 Ci/ft² (from XIII, Sec. D.1)
- b. In ventilation system by t_c : 33 mCi/person (from XIX)

Therefore, the per-person trapped radioiodine is equivalent to that deposited on 33/1,610 or 0.02 ft² of ground surface (or, for a 100-man shelter ventilated at 5 cfm per person, the total trapped-particle radioiodine is equivalent to that deposited on 2 ft² of ground surface). The reason for this inefficient scavenging of particles (at best; that is, even under the assumption of total acceptance of all available particle sizes) is the relatively rapid average terminal fall rate of 3.9 ft/sec (range of 2.55 - 6.40 ft/sec) (from XVII, Section D.1).*

* The implications of the terminal fall velocity on trapping can be seen by reference to a second example problem, in which the shelter is at a point on the hot line 106 mi downwind from the same 10 MT (50% fission) detonation considered earlier. For the new situation, the "standard" exposure rate is 2,110 R/hr at 1 hr, a factor of almost 3 below the earlier value. Key parameters for the new situation are:

(Footnote continued)

(Footnote continued)

	<u>Largest Particles</u>	<u>Smallest Particles</u>
Size, μm	112.8	73.4
Effective V_f , ft/sec	4.03	1.90
Terminal V_f , ft/sec	2.71	1.52
Actual t_a	= 21,100 sec = 5.85 hr	
Actual t_c	= 32,900 sec	
Arrival period	= 11,800 sec as before (no dispersion in model)	
Exposure rate (hypothetical) at t_a	= 254 R/hr	
Total ground-deposit radioiodine Ci/ft ² (corrected to t_a)	= 0.220	
Overall average terminal V_f	= 2.1 ft/sec	

Using the simplified calculation, we have:

$$\begin{aligned} \text{Average volume concentration} &= \frac{0.22}{11,800 \times 2.1} \\ \text{(during arrival period)} & \\ &= 8.9 \times 10^{-6} \text{ Ci/ft}^3, \text{ or} \\ &8.9 \mu\text{Ci/ft}^3 \end{aligned}$$

With a ventilation rate of 5 cfm/person, as before, the total per-person loading of particle radioiodine in the ventilation system is then:

$$8.9 \times (5/60) \times 11,800 = 8.8 \times 10^3 \mu\text{Ci}, \text{ or } 8.8 \text{ mCi (again corrected to } t_a)$$

If this last figure were decay-corrected (by means of the iodine-decay curve) to the t_a of the first example problem, for direct comparison, it would be 27 mCi, compared to the 34 mCi for that situation. Thus the halving of the terminal fall rate in the second problem, which has had a doubling effect on the trapping, comes close to compensating for the reduction of a factor of three in the "standard" exposure rate.

(Footnote continued)

(Footnote continued)

To see the effect in another way, we calculate the equivalent ground-deposit area per person. With all activity measurements decay-corrected to actual t_a , we have, from above:

- a. On the ground by t_c : 0.22 Ci/ft², or 220 mCi/ft²
- b. In ventilation system
by t_c : 8.8 mCi/person

Therefore, the per-person trapped radioiodine is equivalent to that deposited on 8.8/220 or 0.04 ft² of ground surface, just double the 0.02 ft² of the first example problem.

(Footnote concluded)

The result implies that the ventilation-system trapped-particle radioiodine may not represent an upper limit of the inhalation threat. Suppose, for example, that volatilization is quite rapid; in particular, for purposes of comparison, suppose that volatilization is essentially instantaneous at t_c . Then, in a relatively stagnant air atmosphere outside the shelter, the vapor produced from ground-deposited fallout will be much more efficiently sampled by the ventilation system than were the particles during fallout. That is, outside iodine vapor, if any, will not be moving upward past the intake at a net average "velocity" as high as 3.9 ft/sec (2.7 mph), except under extremely gusty conditions. If it were conceptually possible to estimate an average net upward "velocity" for ground-deposit-emitted vapor under atmospheric turbulent-diffusion conditions, then the potential ingress of outside

* Admittedly, this is very unlikely, especially for dry fallout, as discussed in Section IV and Appendix B. However, as long as volatilization rates, if appreciable, are the same for trapped and ground-deposited particle radioiodine, the following argument should hold.

vapor (on an equivalent-deposit-area basis) could be measured by that "velocity" (at least, where fallout-contour gradients are not steep and/or where surface winds after deposition are negligible). In particular, under such circumstances the relative importance of the outside vapor threat to that posed by trapped particles would simply be the ratio of the average terminal velocity during fallout to that (smaller) upward "velocity". A more realistic way to measure the outside-vapor threat is presented in the next section of this Appendix.

D.3. Effect of Turbulent Diffusion at Ground Surface on Increase of Outside Vapor

In this analysis, a highly idealized situation is hypothesized. The ground deposit of particle radiiodine is assumed to be an infinite uniform field, so that (1) the effect of surface winds after deposition and volatilization (except for the creation of upward turbulent diffusion of iodine vapor) is merely to replace that vapor which moves downwind with an equivalent quantity from upwind sources; and (2) the effect of lateral turbulent diffusion is similarly nullified. Thus the problem of the history of the vertical distribution of vapor can be treated as a one-dimensional diffusion situation. Furthermore, as already indicated, the radiiodine is assumed to vaporize instantaneously at the moment of fallout cessation (and to be distributed within a limited vertical region at $t = 0$), which permits comparison with the consequences of the same assumption for the trapped particles, as discussed in Section IV of the main report. Any other volatilization history can then be treated as an extension of this simple one, for both the trapped- and the outside-particle sources, considered in relation to one another.

* It is clear from the result for the second example problem that the threat from trapped particles becomes more and more important relative to that from outside vapor the farther downwind the shelter is located.

The basic one-dimensional diffusion equation for this situation is

$$\frac{\partial R}{\partial t} = K_z \frac{\partial^2 R}{\partial z^2} \quad (D.1)$$

in which

- R is the volume concentration, decay-corrected radioiodine Ci/cm³
 K_z is the vertical turbulent-diffusion coefficient, assumed here to be invariant (Fickian), cm²/sec
 z is distance above the ground surface, cm
 and t is time, sec.

The basic equation is solved in the traditional way, on the assumption of separability of variables:

$$R(t,z) = T(t) \cdot Z(z) \quad (D.2)$$

which leads to the general (one-term) solution:

$$R = e^{-K_z \lambda^2 t} (C \cos \lambda z + D \sin \lambda z) \quad (D.3)$$

where λ , C and D are constants of integration, to be determined on the basis of the boundary conditions*, and λ is taken as a positive number.

* Normally, the solution is an infinite series of terms of the same form, each with its own set of values of λ , C and D , thus permitting imposition of a variety of boundary conditions: for example, a uniform initial distribution over a specified range of z . Another limitation of the single-term, positive- λ restriction is that the history can be followed only for z smaller than (or equal to) the upper boundary of the original distribution, and that the history at every elevation in this region is an exponential with the same "decay" constant. Finally, the treatment assumes an infinite sink (infinite height) for ultimate dispersion. Actual maximum mixing depths are limited. In the U.S., the January average over the country is 400-500 m; the July average is ca 1500 m (METEOROLOGY AND ATOMIC ENERGY (1966), Fig. 2.23).

In a vertically symmetrical situation, in which upward and downward diffusion from a source plane or slab are possible, D becomes zero. In our situation, it can still be taken as zero if the ground surface is considered as a mirror (with a doubled source strength, which is taken into account when the boundary conditions are introduced, in any event).

Thus, the simplest form of the solution for our purposes is

$$R = e^{-Kz} \lambda^2 t \quad (C \cos \lambda z) \quad (D.4)$$

which, however, forces a cosine function as the vertical distribution at each time*, and in particular at $t = 0$.

The constants of integration are obtained directly. If at $t = 0$ we define

$$R_p = \text{the peak initial volume concentration, at } z = 0$$

then $C = R_p$

and $R(0, z) = R_p \cos \lambda z$

or $R(0, 0) = R_p \cos 0^\circ = R_p$.

If we now let $z = a$ be the upper limit of the initial distribution, requiring that

$$R(0, a) = 0 \quad (\text{i.e., } \cos a \lambda = 0)$$

* A closer approach to a uniform initial distribution is to use Equation D.3 as is, with $C = D$. This is done in Case V below.

then $\lambda a = \pi/2$

so $\lambda = \pi/2a$

and Equation D.4 becomes

$$R = R_p e^{-K_z \pi^2 t/4a^2} \cos(\pi z/2a) \quad (D.5)$$

We can then determine R_p on the basis of our known value of the deposited radioiodine activity, A , in Ci/cm². That is, if at $t = 0$ A is cosine-distributed between $z = 0$ and $z = a$, then

$$\int_0^a R_p \cos(\pi z/2a) dz = A$$

from which

$$R_p = \pi A/2a$$

so the final form of the solution is

$$R = (\pi A/2a) e^{-\pi^2 K_z t/4a^2} \cos(\pi z/2a) \quad (D.6)$$

Given a ventilation rate, G , in cm³/sec per person, we can compute Q , the total (decay-corrected) radioiodine sampled to infinite time (or to 5 concentration half-lives, in practical terms), as

$$Q = G \int_0^{\infty} R dt$$

$$Q = (2aGA/\pi K_z) \cos(\pi z/2a) \quad (D.7)$$

* Earlier in this Appendix, we used units of Ci/ft² for A . It is convenient here to convert to metric units, to conform to the literature values of K_z .

Representative values of the vertical turbulent-diffusion coefficient, K_z , can be obtained from Meteorology and Atomic Energy (Slade) (1968). Table 3.2 (based on Richardson's earlier definitions) of that reference gives values of the horizontal eddy diffusivity, K_y , for various scales of turbulence. For example, under low-level wind-shear conditions, for a turbulence scale of 1.5×10^3 cm (ca. 50 ft), the smallest-scale and therefore the least-dispersing situation considered by Richardson,

$$K_y = 3.2 \times 10^3 \text{ cm}^2/\text{sec} .$$

On the basis of related information from Slade*, we calculate

$$K_z = 0.8 \times 10^3 \text{ cm}^2/\text{sec}$$

for the same conditions.

D.3.1. Upward Diffusion With No Lid (Normal Atmosphere)

We may now examine the effect of normal atmospheric turbulence, with no lid to the rise of ground-surface-originating vapor, on the ingress of radioiodine vapor:

Case I

Let $a = 100 \text{ m}$ ($\approx 10 \text{ ft}$).

That is, consider the instantaneously vaporized radioiodine to be initially cosine-distributed within the first

* If σ represents the dispersion of a point-source plume after travel for time t , then

$$\sigma = (2Kt)^{1/2}$$

or

$$K = \sigma^2/2t$$

i.e., K goes as σ^2 . For early times, and therefore limited travel of the plume, Slade's Figures 3.10 and 3.11 show, for the "slightly stable" or "moderately stable" atmosphere, that σ_y is approximately equal to $2\sigma_z$. Therefore, $K_y \approx 4K_z$. Note that the value of K_y used here represents a regime somewhere between "slightly" and "moderately" stable (Pasquill's conditions E and F, respectively); i.e., a "normal" lapse rate.

10 ft above the ground surface. Then, for $K_z = 0.8 \times 10^3 \text{ cm}^2/\text{sec}$ ("normal" lapse rate), Equation D.6 leads to

$$R = (A/200) e^{-t/50} \cos(z/200)$$

which implies a concentration half life, at each value of z , of about 35 sec.

At the ground surface,

$$R(t,0) = (A/200) e^{-t/50}$$

For a ventilation rate (intake at the surface) of 5 cfm/person ($\approx 2,200 \text{ cm}^3/\text{sec}$ per person), the total (decay-corrected) radioiodine sampled there to infinite time, Q , is then, from Equation D.7*

$$Q = (2) (100 \Pi) (2200) A / 800 \Pi$$

or

$$Q = 550A$$

That is, under all the assumptions of the derivation, the shelter ventilation system takes in (per person) outside radioiodine vapor equivalent to the total (decay-corrected) radioiodine in 550 cm^2 of ground deposit; i.e., in ca 0.6 ft^2 of ground deposit. This is 30 times the (per-person) trapped-particle radioiodine calculated earlier (0.02 ft^2 equivalent) in the first example problem.

Case II

With a ≈ 10 ft, as before, sample at the 5 ft height rather than at the ground surface. Then

$$Q = 550A \cos \Pi/4$$

$$\approx 400A$$

or the per-person shelter vapor intake is equivalent to a ground deposit of about 0.4 ft^2 , in our terms not significantly different from that of Case I. Clearly, the ground-surface

* The units of Q are (cm) (cm^3/sec per person) (Ci/cm^2) (sec/cm^2), or Ci/person . For the Case I situation, "infinity" (or about 5 concentration half lives) is about 3 min.

intake is the highest possible intake for any situation following the cosine law*.

Case III

Return to ground-level sampling, but let $a = 300\pi$ cm (ca 30 ft).

Then

$$Q = 1650A$$

or the per-person shelter vapor intake is equivalent to a ground deposit of about 1.8 ft^2 . In general, then (see Equation D.7), with everything else equal Q is proportional to a ; that is, for the same total deposit, the infinity intake is greater the flatter the original distribution**. Thus, by assuming a fairly extensive initial mixing (even to 10 ft (Case I)), we may be overestimating the threat from intake of outside vapor.

Case IV

Another series of atmospheric situations (based on work of Pasquill) is presented in Slade's*** Figure 3.11 and Table 3.3. For the "moderately stable" situation (night-time condition; less than 3/8 cloudiness; 2 m/sec wind) the value of σ_z for 100 m downwind is given as 2.3 m.

Thus, $t = 100 \text{ m} / (2 \text{ m/sec}) = 50 \text{ sec}$

and
$$K_z = \sigma_z^2 / 2t = (2.3 \times 10^2)^2 / (2)(50)$$
$$= 530 \text{ cm}^2/\text{sec}$$

which is not far from the value of $800 \text{ cm}^2/\text{sec}$ used in the first

* In general, no matter how many terms are used in the general solution of the diffusion equation, the maximum intake will occur at the point of highest initial concentration, for reasonable initial conditions.

** The concentration half-life goes as a^2 , per Equation D.6. Thus, for the Case III situation it is about 315 sec, or "infinity" is about 30 min.

*** Meteorology and Atomic Energy (1968), op. cit. The most-table situation considered there is the so-called "moderately stable" regime. This is still considerably more turbulent than a true (stagnant-air) inversion.

three cases. Converting the result of Case I by the ratio of the V_z 's, for ground-level sampling with $a = 100\pi$ (ca 10 ft) we have

$$Q = (550A) (530/800) \approx 350A$$

or about 0.4 ft^2 equivalent.

Case V

Finally, we examine a more-uniform initial distribution, obtained by addition of a $(D \sin \lambda z)$ term; that is, we return to Equation D.3 and specify that

$$R(0,0) = R(0,a) = R_{\text{ext}}$$

so that $R(0, a/2)$ is higher than the concentrations at the extremes of the range (in fact, is $\sqrt{2} \cdot R_{\text{ext}}$).

The resulting equation for R is

$$R = (\pi A/4a) e^{-\pi^2 K_z t/4a^2} [\cos(\pi z/2a) + \sin(\pi z/2a)]. \quad (\text{D.8})$$

and the corresponding equation for Q is

$$Q = (aGA/\pi K_z) [\cos(\pi z/2a) + \sin(\pi z/2a)]. \quad (\text{D.9})$$

If, as in Case I, we let $a = 100\pi \text{ cm}$,
 $K_z = 800 \text{ cm}^2/\text{sec}$
 and $G = 2200 \text{ cm}^3/\text{sec}$ per person

and sample at $z = a/2$, where the peak concentration occurs, then the peak value of Q is $(275A) (\sqrt{2}) \approx 400A$. Note that this is the same Q we had at the same $z (= a/2)$ in Case II, everything else being the same except the shape of the initial distribution.

For the turbulent-diffusion situations considered, which generally represent the ordinarily most dangerous situations* from the point of view of shelter intake of radiiodine vapor in infinite fields, the threat appears to be a factor of perhaps 10-30 times that from trapped particles (in the fallout situation of the example problem). However, as pointed out in

* Outside of temperature inversions.

Slade* (his Section 4-12), "The vertical spreading of material released from a continuous source has received comparatively little attention in diffusion experiments owing primarily to the difficulty and expense of adequately documenting this feature. Common practice has been to estimate the vertical diffusion from measurements of the ground-level crosswind spread and concentration distribution, a technique prone to some degree of error because of the effects of deposition. For release within a typical radiational inversion, it can be stated with considerable assurance that the vertical spreading will cease after some short travel from the source and the diffusion process will be dominated by the lateral spreading and the as yet not fully accounted for effects of vertical gradients of the horizontal wind. Under unstable conditions the plume will spread rapidly until some vertical lid to mixing is reached. The existence of such a lid is common.....".

Also, as pointed out in Slade (his section 5-5), "The use of conventional diffusion formulas for the calculation of concentration fields produced by sources on or near buildings often gives misleading answers..... It should be understood that the study of diffusion near buildings is in its infancy and much work is needed to validate and refine the material"...
... available.

For very-low-lying inversions, it is possible that the very rapid concentration decay exhibited in the calculations above will slow down eventually, or perhaps not occur at all, particularly under stagnant conditions near the ground. This can have a profound effect on Q , even if the peak values of R are not changed very much. The inversion situation is treated in the next Subsection.

* Meteorology and Atomic Energy (1968), op. cit.

D.3.2. Upward Diffusion With Lid (Temperature Inversion)

In the preceding Subsection, the intake of iodine-vapor-loaded outside air in a stable atmosphere was assumed to occur in one time regime. That is, after an instantaneous volatilization of iodine from fallout particles on the ground-surface, assumed to fill a narrow surface layer (say about 10 ft high) with iodine vapor at time zero,* the iodine in that layer was then assumed to diffuse (turbulently) upward without restriction; i.e., towards infinite height at infinite time. With the shelter air-intake vent taken to lie within the initial layer, usually at the ground surface, the iodine concentration in the sampled air was thus dropping exponentially to zero at infinite time; this exponential dropoff was a feature of all the calculations throughout Subsection D.3.1. Since the source was assumed to be an infinite-plane field, there was no horizontal dispersion in the time frame of interest.

The simplest possible model for an inversion situation, in which there is a ceiling for upward turbulent diffusion, requires three time regimes after the initial instantaneous volatilization to a cosine distribution. The first, as in the simple situation, is the exponential dropoff within the narrow surface layer, essentially complete after about 5 diffusion half-lives. However, at the end of this regime, the iodine concentration in air at the shelter intake vent is not zero, but rather the average concentration with the total iodine (persquare foot of deposit) distributed uniformly throughout

* The initial vertical concentration distribution usually being a cosine function, with its maximum at the ground surface, and zero concentration at the top of the layer.

the inversion layer (say about 500 ft high)*. The second regime, lasting for the life of the inversion, is one in which the iodine concentration in the sampled air is constant, at the aforementioned average figure. The third regime starts with the dissipation of the inversion, and is treated as a straight-forward upward turbulent diffusion like that of the first regime, except that the starting average concentration is that of the second regime,** and the starting layer thickness is that of the inversion itself.

For a sample calculation of iodine intake during and after a temperature inversion, we make the following assumptions:

Inversion thickness = 5,000 π cm (~500 ft)

Inversion duration = 24 hr

Other conditions as in Case I, Subsection D.3.1:

Initial-layer thickness, \underline{a} ,
(volatilized iodine) = 100 π cm (~10 ft)

Ventilation rate, G , $\approx 2,200$ cm³/sec per
person (5 cfm/person)

Deposited radioiodine activity, A , in Ci/cm²
is left in symbolic form.

Also, we let the initial K_2 (Regime I) be a factor-of-five lower than that for the "normal" (low-level wind-shear) situation considered in Subsection D.3.1, to reflect the fact that turbulence at ground level will be somewhat less in the inversion

* For continuity (matching concentrations at the interface between Regimes I and II), one would have to make Regime I last longer than 5 half-lives if the inversion-layer thickness were more than about 32 times the initial-layer thickness (i.e., half-lives for a thickness ratio of about 65, 7 for 130, etc.).

** The uniform concentration distribution at the start of the third regime can be approximated by a cosine-plus-sine function, as in Case V of Subsection D.3.1.

situation than it is in a "normal" atmosphere. In calculating the upward diffusion in Regime III, we revert to the original value of K_z .

Regime I:

Equation D.6 shows that, for equal values of a , the "thickness" of the original dispersion layer, the diffusion half-life goes inversely as K_z . Therefore, where in Case I of Subsection D.3.1 we had "infinity" (say 5 half-lives) as about 3 min, the "infinity" for Regime I here is about 15 min (K_z here is 0.16×10^3 cm²/sec, compared to 0.8×10^3 for Case I)*.

The "infinity" intake, Q_I (in decay-corrected Ci)** is given by Equation D.7; with every other parameter fixed (in particular, a), Q goes inversely as K_z . Therefore Q for Regime I is 5 times that of Case I, or $Q_I = 2750A$; that is, during Regime I under the assumed conditions, the shelter ventilation system takes in (per person) outside radioiodine vapor equivalent to the total (decay-corrected) radioiodine in 2750 cm² of ground deposit; i.e., in ca. 3.0 ft² of ground deposit. This alone is 150 times the (per-person) trapped-particle radioiodine calculated in the first example problem of Section D.1.

Regime II:

The second regime lasts for the remaining 23.75* hours of the inversion duration. For this whole period, we have a uniform, constant iodine-vapor concentration, resulting from the dispersion (during Regime I) of the total deposited iodine throughout the inversion layer. The intake is therefore the product of the ventilation rate and the (constant) concentration. During Regime II, the intake at ground level would, of course, be the same as that anywhere else in the inversion.

As in Subsection D.3.1, we define R as the iodine vapor concentration (decay-corrected curies per cm³). Thus, R_{II} is given by A/a_{II} , where a_{II} is the thickness of the inversion layer, so that

$$Q_{II} = R_{II} \times G \times T_{\text{inversion}} = AGT_{\text{inv}}/a_{II}$$

* Actually it would take about 5.5 half-lives during Regime I for the ground-level concentration to drop to the expected starting level for Regime II; the small error is ignored in the calculation of the duration and the intake of Regime II.

**Intakes and other key variables are given subscripts denoting the Regime.

Under the assumed conditions for Regime II, Q_{II} turns out to be 12,500A; that is, during the duration of the inversion, the shelter ventilators take in (per person) outside radioiodine vapor equivalent to the total (decay-corrected) radioiodine in 12,500 cm² of ground deposit; i.e., in ca. 14 ft² of ground deposit. Thus, Regime II intake is almost 5 times that of Regime I, if the inversion lasts as long as 24 hr; intake in Regime II is, of course, proportional to the length (duration) of the inversion (and inversely proportional to the inversion height), so that for every 5.3 hrs that the inversion persists, there is an intake equivalent to that of Regime I.

Regime III

The third and final regime is that of the ultimate dispersion (to infinite height at infinite time) of the uniformly dispersed mixture of Regime II, under the influence of the normal atmosphere that is assumed to follow the break-up of the inversion.* As indicated earlier, we return to the K_2 of Case I of Subsection D.3.1, but now a is about 500 ft ($a_{III} = a_{II}$), and we invoke the initial vertical distribution of Case V of Subsection D.3.1, the cosine-plus-sine function, to more closely approximate the uniform distribution than a simple cosine function would.

The intake, Q_{III} , is given by Equation D.9. The only difference from the calculation for Case V (Subsection D.3.1) is that the value of a (that is, a_{III}) is higher by a factor of 50, and we calculate here for sampling at ground level, rather than at the midpoint of the layer. Equation D.9 shows that, everything else being equal, Q is proportional to a . Therefore, Q_{III} is 275A x 50, or 13,750A. Note that Q_{III} is about the same as Q_{II} ; this result implies that Regime III can be the most important of the three, particularly where the inversion is of duration shorter than 24 hr.

In a way, the apparent importance of Regime III is an artifact, resulting from an impossibly long dispersion time, as can be seen from the following argument. First, we note from comparison of Equations D.6 and D.8 that the exponents in both have the same form, so that for a given set of K_2 and a values, the dispersion half-lives are the same in both situations. We may therefore compute the Regime III half-life by suitable modification of the 35 sec half-life of Case I (Subsection D.3.1). Equation D.8 (or Equation D.6) shows that the dispersion half-life, everything else being equal, is proportional to a^2 . Therefore, the Regime III half-life is:

* This is a worst-case assumption for intake. A more-unstable situation (stronger winds) after the breakup of the inversion would imply a higher value of K_2 , hence a lower intake during Regime III.

$$35 \text{ sec} \times (500/10)^2 = 88,000 \text{ sec} = 24 \text{ hr}$$

so that the "infinite" dispersion time is about 5 days. Even if the atmosphere causing the inversion to break up were not very turbulent, this calculated dispersion time is so long that it is unreasonable to continue to invoke the assumption, basic to all these calculations, that horizontal dispersion is not operative (because of the semi-infinite deposit field). Experience indicates that, once conditions leading to an inversion disappear, the breakup requires hours, rather than days.

In summary, the presence of a temperature inversion during the volatilization of radioiodine(s) from deposited fallout particles can markedly increase the shelter intake of radioiodine vapor. That intake is most sensitive (directly) to the length (duration) of the inversion and (inversely) to the thickness of the inversion layer; however, because of the reduced turbulence there is a significant enhancement of intake even during the first stage of vertical dispersion, independent of inversion duration and thickness. There are limits to the effect of an extended inversion period, in that the finite limits of the fallout field lead to an enhancement of the effect of horizontal dispersion, thus dissipating the iodine vapor more rapidly, as time goes on. Also, it must be remembered that all the calculated intakes above are very much upper-limit (worst-case) estimates, since they assume instantaneous volatilization of all the radioiodine burden of the fallout particles at the moment of cessation of fallout. Finally, it should be noted that the commonly occurring nighttime inversion, which dissipates as the ground warms in the morning sun, represents a threat situation intermediate between the "normal" atmosphere (Subsection D.3.1) and the long-term (> 24 hr) temperature inversion created here.

* That is, even if one makes the worst-case assumption (as done here) that the temperature inversion is replaced by a moderately or slightly stable atmosphere, rather than the more likely wind/gust situation.

D.3.3. Summary

The coupling of (1) the wide possible variation in the overall effects of turbulent diffusion on ingress of radioiodine vapor, once formed, with (2) the even greater uncertainty as to the rate and extent of volatilization of iodine from fallout particles; creates a situation in which accurate prediction of the inhalation threat is impossible.* Further discussion of this problem, and recommendations for action, appear in Section IV of the main report.

* It must be emphasized **again** that if volatile radioiodine is a threat at all, the outside-particle source is more important by orders of magnitude than the (shelter-) trapped-particle source.

APPENDIX E

THYROID FUNCTION: IODINE UPTAKE AND RETENTION

E.1. Introduction and General Description

It is well known that the thyroid gland plays a major role in metabolism and growth, and that iodine is the principal ingredient in the thyroid-produced hormones that regulate or otherwise participate in the control and feedback (homeostatic) mechanisms involved. Some understanding of this functioning is needed in the present evaluation, because:

1. The normally occurring processes determine the rate and extent of uptake of introduced radioiodines and the extent of their retention by the thyroid; and therefore the ultimate ionizing-radiation dose delivered by them to the thyroid.
2. The effects of ionizing radiation (particularly where there is a long latent period for appearance of the final pathology) depend presumably on subtle disruptions of the normal control mechanisms.
3. The application of countermeasures, either those that prevent thyroid uptake of newly introduced radioiodine or those that force release of already stored radioiodine, depends upon "tinkering" with those processes and mechanisms.

Again, as in Appendix C and in the main text, it must be noted that an eclectic approach to the gathering of the pertinent input data is acceptable in view of the tremendous uncertainties in other phenomenological areas, chiefly in the extent of iodine volatilization from fallout particles.

Useful descriptions of the human thyroid and its function can be found in textbooks such as Carlson, Johnson and Cavert (1961) and Langley and Cheraskin (1965); a brief summary of the iodine chemistry involved appears in Comprehensive Biochemistry (1971).

The fundamental functional units of the thyroid are the numerous tiny hollow sacks, called follicles, which are essentially round, with diameters varying from about 0.05 to about 0.5 mm (or about 1/500 to 1/50 in.). The shell of each follicle is made up of cells closely packed together; it is wrapped in a thin membrane, which in turn is covered with a dense mesh of blood capillaries, plus some lymph vessels and nerves. The capillaries ensure a steady and abundant supply of fresh plasma to the cells, the thyroid being in fact one of the most highly vascularized organs. The space inside the follicle is filled with a viscous fluid called colloid.

The follicles produce thyroid hormone, store it, and then release it as needed to the blood stream. The hormone is made up of two related compounds: L-thyroxine (chemically L-tetraiodothyronine), designated T₄; and triiodothyronine, designated T₃. Normally, thyroxine is the major constituent of the hormone. T₄ is 65% iodine by weight, T₃ 58%.

Iodide diffusing from the blood capillaries into the cells is concentrated in them, and then, at the edge of the cells next to the colloid, is taken up into organic compounds by a sequence of reactions to form thyroxyl and triiodothyronyl radicals, these consisting of T₄ and T₃, respectively, attached to a large protein called thyroglobulin. The hormones thus stored in the colloid have a fairly low turnover rate. Their release into the blood stream is controlled by the pituitary gland.

When the level of T4 in the circulating blood is reduced below the control level by utilization in the tissues or by degradation, the pituitary senses the change and releases the so-called thyroid-stimulating hormone (TSH), known also as thyrotrophic hormone or thyrotropin. TSH, on reaching the follicles, first induces the release of the stored thyroid hormone; the mechanism involves stimulation of the release of a thyroid-produced enzyme into the colloid. This enzyme then accelerates the breakdown of thyroglobulin into its amino-acid components. The T4 and T3 molecules freed by this process are now small enough to enter the blood stream to restore the balance.

Under normal conditions, the control mechanism is finely balanced; glands and blood are operating close to the equilibrium (steady-state) conditions. However, where overstimulation by the pituitary occurs (for any of several reasons, including thyroid, pituitary or metabolic abnormalities), the breakdown of thyroglobulin in the follicles may be extensive. Under prolonged stimulation, the amount of colloid in the follicles may diminish to the point of near disappearance. The thyroid cells increase in size by protruding inside the follicle, and also divide,* in order to increase their capacity to produce the hormone in an attempt to restore the balance. An extreme result of this kind of imbalance is the formation of neoplasms, both benign (such as goiters) and malignant.

* In a normal adult, mitosis is a rare occurrence (Conard, 1970). In children, normal growth of the thyroid is rapid, from ca. 2 gm at age 1 to ca. 17 g at adulthood. Thus, in children mitosis of the thyroid cells is a natural phenomenon.

The adenomatous (benign) lesions of the thyroid in the Marshallese children were presumably the result of this kind of process, starting with the killing or the incapacitation of some of the thyroid cells by ionizing radiation at the time of uptake and retention of the radioiodines in fallout. Total destruction (atrophy) of the thyroid gland by, say, a massive dose of ionizing radiation leads to a safer result, in some respects, for while regular administration of thyroid hormone (desiccated thyroid extract or synthetic hormone) is required for the rest of the victim's life, there is no danger of thyroid neoplasms, in particular the malignant ones.

In the blood, T4 and T3 bind loosely to plasma proteins, particularly to alpha-globulin. The level of protein-bound iodine (PBI) is a measure of the concentration of the thyroid hormone in the blood. Inorganic iodine (iodide) is also circulating continuously. It is added to the blood by digestion of iodine-containing foods and by degradation of T4 and T3, and is depleted from the blood by excretion (mostly in urine) and by uptake by the thyroid. The disposition of a given intake of iodine (whether radioactive or stable) depends, therefore, on the size of the intake (mg of iodine), the current blood levels of iodide, and the over-all state (normal or pathological) of the thyroid/pituitary/metabolic system.

In the normal state, the individual is said to be euthyroid. Total iodine in the blood is of the order of 6 $\mu\text{g}/100\text{ ml}$ (range 4-7.5 $\mu\text{g}/100\text{ ml}$)*, of which only 0.6 $\mu\text{g}/100\text{ ml}$ is inorganic; the remaining 5.4 $\mu\text{g}/100\text{ ml}$ is about 96% protein-bound, only 0.2 $\mu\text{g}/100\text{ ml}$ consisting of free thyroxin. Only about 1 mg of intake iodide (iodide is the digested form, regardless of the chemical state in food) is required per

* The figures given here are from Langley and Cheraskin (1965). $\mu\text{g}/100\text{ ml}$ are also known as μg percent.

week to maintain the balance (that is, about 1/8 mg per day). For a given individual, the basal metabolic rate (BMR) varies by no more than 1 or 2% from day to day.

When the PBI drops well below say 3 µg/100 ml, often close to zero, the individual is said to be hypothyroid. His thyroid gland is underfunctioning, or hardly functioning at all. His BMR is about 30% lower than normal, and a range of symptoms follow:

In young children: Stunted growth; mental backwardness; dry, wrinkled skin. The combination is known as cretinism; advanced stages are almost extinct because of removal of the primary causes (iodine deficiency, by addition of iodide to drinking water or salt; other causes, by thyroid-hormone therapy).

In adults: Dry skin; falling hair, obesity; lassitude and failure to function intelligently despite retention of basic intelligence; and myxedema, which is a puffiness, especially around the eyes, that results from the deposition of a semifluid albuminous material in the soft tissue beneath the skin. The name myxedema is applied to the whole complex of symptoms in the adult. It too can be effectively treated by thyroid medication, usually in the form of oral administration of desiccated thyroid extract.

Conversely, in hyperthyroidism, in which the thyroid is overactive, PBI can range up to 15 or even 20 µg/100 ml. The BMR can go up to 30% above normal. Hyperthyroid individuals have an accelerated heart rate and moist skin; seem to require very little sleep; are hyperirritable; and are "drivers". The condition is often accompanied by exophthalmos, a bulging of the eyes due to edema, lipid deposition and cellular infiltration in the eye muscles. Advanced cases may go blind from damage to the optic nerve, or at least experience drying and irreversible changes in the cornea, because of inability to close the upper eyelid over the bulging eyeball.

The treatment of choice in hyperthyroidism is partial extirpation of the thyroid, either by surgical removal of part of the gland (partial thyroidectomy) or by treatment with massive (therapeutic) doses of I-131, which concentrates in the thyroid, delivering a large dose of beta radiation locally and thus killing part of the gland. In either case, reduction of functional thyroid tissue decreases the output of T4 from then on, thereby restoring PBI and BMR to the normal range. Sometimes antithyroid drugs such as thiourea and thiouracil and their derivatives (which inhibit the oxidation step in the organification of iodide in the follicles) are used, in concert with thyroxine.

One test of thyroid function is measurement of the uptake by the thyroid, in a 24 hr period, of an orally administered small (diagnostic) dose of I-131.* Langley and Cheraskin give 15-45% uptake as the range for the euthyroid adult.** In older children, the normal range has sometimes been taken as 25-50% (Tompkins, 1970). Uptakes higher than 50% in this age group are associated with hyperthyroidism, uptakes lower than 25% with hypothyroidism. The subject of uptake extent will be developed further later in this Appendix.

Pathology has been discussed thus far mainly in terms of function. It can also be considered in terms of gland

* What is measured is the percent of the administered activity (decay-corrected) that is found in the thyroid 24 hr after administration. Most of the remainder has been excreted by this time; some is still circulating.

** Strictly speaking, the numbers in the normal range apply only if the total body store of iodine is in the normal range also (a euthyroid individual who for some time has been on a reduced iodine intake may exhibit abnormally high uptake in the test.)

structure, (size and deformity) (or in general, in terms of morphology). Such consideration will be deferred until Appendix F following, where the sequence: (1) unseen radiation damage to the thyroid; (2) apparent functional impairment; (3) irreversible morphological changes in the thyroid; will be discussed. It should be pointed out in passing that while most of the visible (palpable) morphological damage seen in the thyroid follows, rather than precedes, hormonal imbalance (for instance in endemic (iodide-deficiency) goiter), thyroid damage can occur also from causes unrelated to thyroid-hormone levels, for example from viral inflammation of the gland; generally, however, the behavior of the thyroid becomes impaired at some point in the sequence because of the homeostatic nature of the normal functioning.

E.2. Uptake in Adults

In a recent study, (Nelson, Renschler and Dowswell, 1970) the authors point out that "The thyroidal uptake of radioactive iodine in persons without thyroid disease varies with geographic location, and may change in the same location from one decade to the next....." and find that ".....in the San Bernardino Valley region of California uptake in our patients without thyroid disease did not correspond with the usual "normal ranges" of 20 to 50 percent or 15 to 45 percent.....". Their study showed that in that region of Southern California the "normal range" is 6 percent to 33 percent in euthyroid subjects, which is lower than in other areas of the United States, while urinary iodide excretion and the absolute iodine uptake (stable iodine intake, on a weight basis) are higher than those in other U. S. areas. Geographic variation in iodine abundance in food is considered to be the reason for the differences.

structure, (size and deformity) (or in general, in terms of morphology). Such consideration will be deferred until Appendix F following, where the sequence: (1) unseen radiation damage to the thyroid; (2) apparent functional impairment; (3) irreversible morphological changes in the thyroid; will be discussed. It should be pointed out in passing that while most of the visible (palpable) morphological damage seen in the thyroid follows, rather than precedes, hormonal imbalance (for instance in endemic (iodide-deficiency) goiter), thyroid damage can occur also from causes unrelated to thyroid-hormone levels, for example from viral inflammation of the gland; generally, however, the behavior of the thyroid becomes impaired at some point in the sequence because of the homeostatic nature of the normal functioning.

E.2. Uptake in Adults

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Vought and London (1967) conducted a comprehensive long-term measurement program on 3 normal adult subjects, in which uptakes and clearances on an absolute basis were determined. They reported that there appeared to be an obligatory excretion of 57 μg of iodine per day, which was independent of the iodine intake, and that with intakes higher than 57 $\mu\text{g}/\text{day}$ the fraction of intake excreted was approximately unity; i.e., that the iodide "compartment" appeared to be in a steady state.

Schneider (1964) and Rosenberg, LaRoche and Ehlert (1966) were able to show, by double- or triple-tracer experiments on rats, that thyroidal iodine exhibited a "heterogeneous" turnover. That is, the most recent intake of iodine did not mix uniformly with the previously deposited iodine in the thyroid; in fact, it was disposed of first by the thyroid (after having been present in it for some time). Apparently, the thyroid degrades or secretes recently formed organic iodine before it releases older stored iodine, and thus essentially handles iodine in a "last come, first served" mode. This result supported earlier observations that the thyroid was structurally (and functionally) heterogeneous, manifested by (1) a more rapid I-131 turnover by the smaller follicles and (2) the fact that the specific activity of radioiodine leaving the gland was higher shortly after I-131 administration than it was after a longer time.

In a metabolic-behavior study based on fallout I-131 in milk consumed on a regular long-term basis by a euthyroid adult, Boni (1965) found that for each day's intake, a constant 25% of the I-131 remained in the thyroid, 68% being excreted in the urine and the remaining 7% presumed to be distributed elsewhere in the body or excreted in the feces.

E.3. Uptake in Children, Particularly Infants

Uptake in infants is an important consideration in the present evaluation. Early in the history of uptake measurements it had been considered that infant uptake was much higher than that of adults. Ogborn, Waggener and Van Hove (1960) summarize earlier work in this area and report new results of their own. For example, they include data of Van Middlesworth (A.M.A. J. Dis. Child. 88, 439 (1954)) on 7 normal newborn boys, 2-3 days old. The uptake range was 46-97%, with a mean of 69.7%; this had been taken to be "within the range of values which would be found in hyperthyroid adults". They continue with data of Martmer et al. (1956) on 65 premature infants and 5 full-term Caesarean deliveries, whose age at time of testing had ranged from 1 to 63 days. The uptake range was 7 to 61%, with a mean of 32.4%. This range had been taken to be within the limits of normal as recorded in studies of adults. Finally, the work of Ogborn, et al. themselves was on 26 newborn infants, age range 72-180 hr (mean 90 hr), of whom 15 were male, 11 female. The uptake range was 6.3 to 36.4%, with a mean of $20.3\% \pm 8.5\%$ (std. dev.). 54% of the cases were within 1 S.D. of the mean, and all were within 2 S.D. of the mean.

Wellman, et al. (1967) used fallout I-131 in milk as their tracer in a long-term balance study on a group of 10 normal older children; their measured thyroid burdens (whole-body counting) were compatible with an uptake of 15-20% and the generally accepted effective retention half-life (I-131) of 7.6 days. They noted that, if the actual retention half-life for children were lower (and there is some basis for this possibility), the results might then have been compatible with the generally accepted uptake of 30%, but pointed out that there was literature support for an uptake of 15-20% also.

In view of the variations and the uncertainty in the literature, and because of the limited accuracy required for inputs in the present evaluation, it appears that infants and children can be assumed to have the same percent uptake as adults. This was the working assumption of Ng and James (James, 1964) in their calculation of the uptake and the thyroid dose of the Marshallese children.*

E.4. Fetal Uptake

Useful input information appears in two studies, both made possible when pregnancies had to be terminated for medical reasons. In the first, Czerniak, Soferman and Chajchik (1969) made extensive measurements on a 4.5-month and a 6-month aborted fetus, after administration (1-3 days pre-abortion) of tracer doses of I-131 to the mothers. Both mothers had uptakes in the range 42-44% in 20-24 hours. It is known that the placental barrier permits iodide and free T3 and T4 to pass easily, but stops most of the globulin complexes of the thyroid hormones. The free maternal T3-T4 seems sufficient for the fetus during the first 3 months, but is insufficient thereafter. Therefore, at about 3 months into the gestation period the fetal thyroid starts to synthesize PBI compounds. Of the dose of I-131 given to the mother,

* Karhausen, Pages and Ermans (1970) in a more elaborate study of the fate of radioiodine in infants and adolescents used the following scheme for their models:

Adults:	40% uptake at 24 hr
Adolescents (10-15 yr):	ca 50% at ca 22 hr
1-2 yr olds:	" 50% " " 12 hr
6-12 mo old:	" 45% " " 18 hr
0-6 mo old:	" 55% " " 18 hr

0.03% is taken up by the 4.5-month fetus (this low figure implies hypofunction), but 1.1% is taken up by the 6-month fetus. The latter figure is one data point indicating that the 6-month fetus is thyrotoxic (hyperfunctioning), since, according to the authors, it implies that iodine uptake in the fetal thyroid at 6 months is about 2.6 times* that of the mother (i.e., ca 100%). Further support comes from the measured results, among others, that (1) the conversion rate of I-131 to PBI is 3 times higher in the 6-month fetus than in the mother and (2) the I-131 concentration in (6-month) fetal blood is 1.5 times higher than that in the mother. The authors conclude that the 6-month old normal fetus can be expected to be thyrotoxic in the sense described, and that there is an increasing level of thyroid activity to that point in all fetuses, at least up to 6 months post-conception.

Dyer and Brill (1969) were able to make careful measurements on 9 aborted fetuses, again after pre-abortion administration of I-131 to the mothers. Fetal age at abortion ranged from 9 to 22 weeks. The authors were able to show that during the first 13 weeks of gestation there is no accumulation of I-131 in the fetal neck region; 13 weeks is the first point at which one can identify a fetal thyroid gland. There appears to be a steady rise in fetal thyroid uptake of iodine between 13 and 22 weeks. The percentage of the mother's intake of I-131 that was taken up by the fetus was about the same, for corresponding fetal age, as that reported by Czerniak, et al., op. cit., and the apparent fetal uptake percent was in the range 55-75% at gestation age 14-22 weeks, as compared to Czerniak's figure of about 100% at gestation age 6 months.

* Presumably not all of the mother's circulating iodide is available to the fetus in a given 24-hour period.

In view of the high uptake and the extremely small size of the fetal thyroid, it appears that fetal thyroid dose may be extremely high where the pregnant mother has had a substantial intake of radioiodine; combined with the fact that the fetal thyroid cells are dividing rapidly, this creates a special problem where pregnant women are exposed to high intakes of the radioiodines in the civil-defense context.

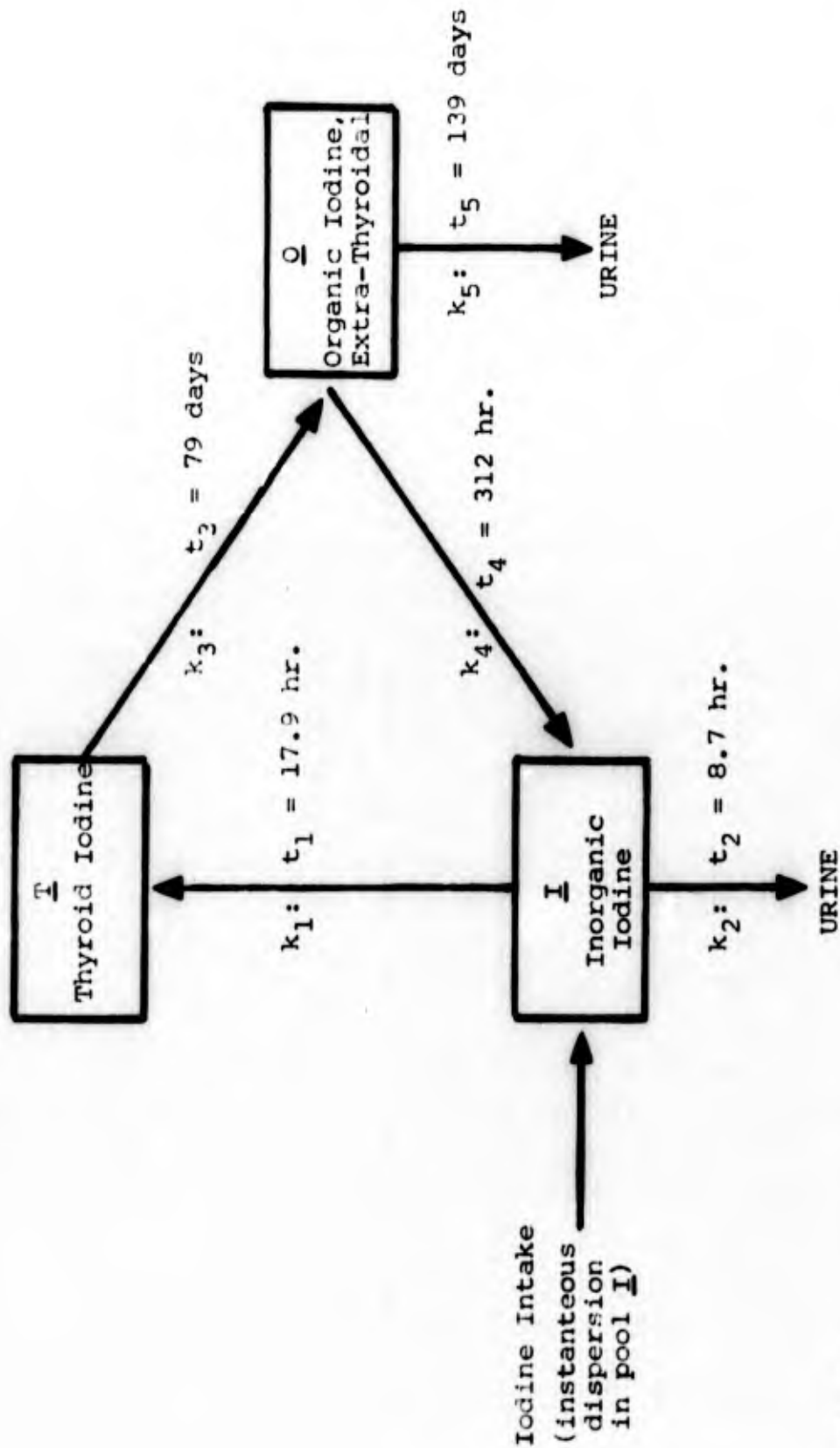
E.5. Models for Uptake and Retention

Computation of ionizing-radiation dose to the thyroid from deposited radioiodines depends on a knowledge of iodine uptake rate and extent and of iodine retention in the thyroid. Both depend in turn on the complicated homeostatic system described briefly in Section E.1. There is an extensive literature on models for iodine transfer through that system. Basic data used in model development appear in works (op. cit.) like Vought and London (1967), Schneider (1964) and Rosenberg, LaRoche and Ehlert (1966). Generally, rate processes for uptake, degradation, release, etc. are taken to be first-order. For the adult, at least, rate constants are based on the assumption of equilibrium (with respect to stable iodine) in a steady-state situation: that is, daily intake is equal to daily excretion; the quantity fixed by the thyroid per unit time is equal to the quantity released by the thyroid in the form of thyroid hormone per unit time, etc.

The so-called three-compartment model used for the dose calculations reported in Section IV is that of Mechali, et al. (1966), which was a modification of an earlier model^{*}. Mechali's model is shown in Figure E.1. The rate constants from which

* Riggs, D.S., Pharmacol. Rev. 4, 284 (1952).

Figure E.1
3-COMPARTMENT IODINE MODEL



NOTES:

1. For most applications, k_4 and k_5 are ignored. That is, a composite k_3 controls release.
2. Values of k_1 and k_2 used in this model imply ultimate thyroid uptake of 30% of intake, with effective uptake half-life $[t_1 t_2 / (t_1 + t_2)]$ of 5.8 hr.

the half-lives shown were calculated (as $0.693/k$) are:

$$\begin{aligned} k_1 &= 0.0389 \text{ hr}^{-1} \\ k_2 &= 0.0800 \text{ hr}^{-1} \\ k_3 &= 0.000364 \text{ hr}^{-1} \\ k_4 &= 0.00222 \text{ hr}^{-1} \\ \text{and } k_5 &= 0.000208 \text{ hr}^{-1} . \end{aligned}$$

Where the sole interest is in the uptake by the thyroid and the extent of retention therein (for computation of thyroid dose during the first few radiological half-lives), the model can be simplified by removal of the Q compartment; that is, k_4 and k_5 can be taken as zero, and the k_3 path can be assumed to go directly to excretion. This simplification to a 2-compartment model was done by Mechali for his calculations of thyroid dose, as used in Section IV of the present report. By straightforward calculation, it can be shown that the values of k_1 and k_2 shown in Figure E.1 imply (with no other assumptions) a peak thyroid uptake of 32.9% for stable or decay-corrected iodine, associated with an effective uptake half-life of 5.8 hr. (The low value of k_3 means that that route plays no part in the uptake calculation. At the same time, it means that the effective half-life of I-131 in the thyroid, once deposited, is only slightly less than the radiological half-life, 8.05 day.)

A slightly different set of rate constants was used by Ng (James, 1964) for the estimation of the Marshallese uptake (see discussion in Appendix A, Section A.3 of this report). For example, Ng's effective uptake half-life was 4.5 hr, compared to Mechali's 5.8 hr. As was shown in Appendix A, Ng's estimates of percent of radioiodine intake released in urine about 2 weeks after intake were consistent with Harris'

(1954) earlier estimates for the Marshallese. Mechali's model leads to similar results for release.

It is possible, of course, to produce more elaborate models, in which intermediate steps, such as conversion of iodide to organic iodine in the thyroid, are added, but these have no application in the present evaluation. In passing, it may be mentioned that Adams and Bonnell (1962) used a so-called 3-compartment model in which 9 paths were characterized, including intermediate steps and other excretion paths, and that Ramsden, et al. (1967) used a so-called 4-compartment model, in which the "iodide space" consisted of two compartments, #1 for inorganic iodine in plasma, and #2 for an inorganic diffusion volume.

As already mentioned in Section E.3, Wellman, et al. (1967) have discussed the possibility that iodine-retention times in children's thyroids may be lower than those in the thyroids of adults. Karhausen, et al. (1970), whose elaborate scheme for variation of uptake with age has also been presented in that Section, assumed that the effective thyroid retention half-life for I-131 was about 8 days for adults and adolescents, but only 4 to 5 days for infants of age 6 months to 2 years.

E.6. Dosimetry

The basic equation for beta-radiation-energy absorption is given in International Commission on Radiological Protection (1959/1960). More recent information covering special situations can be found in M. I. R. D. (1968, 1969); the most complete discussion of the background information on dosimetry

is that of Hine and Brownell (1956). The I.C.R.P. equation gives the beta energy absorbed in an organ when the energy source is uniformly distributed throughout that organ, and absent everywhere else. Its claimed accuracy is 5% for most cases of interest. It assumes implicitly that energy losses from emitters close to the surface are negligible compared to the total energy absorbed from interior emitters. For the adult thyroid, this is an excellent approximation (conservative in the sense of safety), especially if one has ignored the gamma-energy absorption in the calculation. The assumption is still acceptable for the child's thyroid,* considering the limited accuracy required in the present evaluation. It undoubtedly leads to a poor approximation in calculations involving the fetal thyroid;* however, the calculated fetal doses discussed in Section IV are based on more exact dosimetry.

For a particular beta radiation whose maximum energy (conventionally E_{\max}) is E_m , the I.C.R.P. equation is:

$$E = 0.33 f E_m \left\{ 1 - \frac{Z^{1/2}}{50} \right\} \left\{ 1 + \frac{E_m^{1/2}}{4} \right\}$$

* Klassovskii, Vasilenko and Terekhov (1971) have calculated fractional absorption of the beta energy for the four most important radioiodines, in thyroids of different sizes. In a 1.2-g dog thyroid the fractions absorbed are:

I-131	0.93
-132	.87
-133	.88
-135	.91

Thus, a 2-g child's thyroid would presumably absorb over 90% of the beta energy for each of the 4 principal radioiodines, and addition of the iodine gamma energy would bring the figures closer to 100%. Even in the 0.02g rat thyroid, the lowest beta absorption fraction (I-133) is 0.62, with an absorption fraction of 0.84 for I-131 as the highest.

where

- E = energy absorbed (Mev) per disintegration
 Z = atomic number of emitting nucleus (for iodine, $Z = 53$)
 E_m = maximum energy for the particular beta ray (Mev)
 f = fraction of disintegrations producing betas with maximum energy E_m

so for iodine,

$$E = 0.28 f E_m \left\{ 1 + \frac{E_m^{1/2}}{4} \right\}.$$

The 1959 I.C.R.P. document ascribed a relative biological effectiveness (RBE) of 1.0 to beta radiation of energy greater than 0.03 Mev. The 1971 N.C.R.P. version of this concept is the quality factor (QF), but the implication for dose effectiveness is unchanged.

For conversion of absorbed energy to dose in an infinite homogeneous medium (the assumed total absorption in an organ is equivalent to this situation), M.I.R.D. (1968) gives, for the dose in rad from a particular beta radiation:

$$D_{eq} = \tilde{C} \Delta_i$$

where

\tilde{C} is the cumulated (time-integrated) concentration of the emitter, in $\mu\text{Ci-h/g}$

and Δ_i is the energy* emitted (absorbed) per disintegration, in the form of i-type beta radiation. It has the units g-rad/ $\mu\text{Ci-h}$, and is given by

* Aside from the units, which are associated with the constant 2.13 in the defining equation for Δ_i , which follows.

$$\Delta_i = 2.13 n_i \bar{E}_i$$

in which n_i is the f of the I.C.R.P. equation (i.e., number of i-type beta particles per disintegration)

and \bar{E}_i is the mean energy per i-type beta particle

so that $n_i \bar{E}_i$ is identical to the E in the I.C.R.P. equation.

The constant 2.13 is equal to the product of

$$1.602 \times 10^{-6} \left(\frac{\text{erg}}{\text{Mev}} \right) \times 10^{-2} \left(\frac{\text{rad}}{\text{erg/g}} \right) \times 3.70 \times 10^4 \left(\frac{\text{dis/sec}}{\mu\text{Ci}} \right) \times 3.60 \times 10^3 \left(\frac{\text{sec}}{\text{h}} \right)$$

and has the units of g-rad/($\mu\text{Ci-h}$) (Mev/dis).

Application of the dose equation in the thyroid-uptake situation (for calculation of the dose accrued after peak uptake has been attained) requires specification of each isotope involved; summation over all its beta rays; and specification of thyroid mass, uptake percent, and effective retention half-life. Loevinger (in Hine and Brownell, 1956) presents the appropriate equation for I-131 uptake:

$$\text{Thyroid dose (rad)} = 15. \frac{\text{AUT}}{\text{M}}$$

where

- A = (instantaneous) intake of I-131 (μCi)
- U = fractional uptake
- T = effective retention half-life (days)
- M = mass of thyroid (g)

Pendleton, Lloyd and Mays (1963) used this simplified equation for I-131 intake by the infant or young child, with $U = 0.3$ (30% uptake), $T = 7.6$ days, and $M = 2$ g. In this situation, the thyroid dose per μCi of I-131 intake is 17 rad. Basically the same approach was used by James in computing the Marshallese children's thyroid dose from I-131 plus the other radioiodines.

If the individual is exposed to whole-body penetration from external sources, such as deposited fallout, the appropriate "external" dose must be added to the "internal" thyroid dose to give a total dose to the thyroid. The concentration factor for available radioiodine in the child and the adult is so large that thyroid-dose contributions from other internal sources, in particular from circulating radioiodine, are usually negligible. Even for the early fetus (13-22 weeks), less than 0.1% of the I-131 thyroid dose comes from outside the thyroid (Dyer and Brill, 1969).

APPENDIX F

IONIZING-RADIATION INDUCTION OF THYROID PATHOLOGY

F.1. Nature and Mechanism of Pathology

In many respects, the mechanism for the induction of pathological states in the thyroid gland by ionizing radiation (together with the associated end points) is similar to that resulting from a low-iodine diet or from administration of anti-thyroid drugs such as methyl thiouracil (MTU)*. Iodine is normally received by the thyroid in the form of iodide ion; it is there organified to T4 by a sequence of chemical reactions. Iodide intake, transport, uptake and organification must all be normal if the thyroid is to function. The first effect of a low-iodide diet is to reduce the thyroid output of T4, because of insufficient raw material. The same effect is produced by anti-thyroid drugs, which interfere with the iodide-oxidation or subsequent organification step in the production of T4 by the thyroid. The first effect of ionizing radiation, on the other hand, is to kill or at least inhibit the function of some thyroid cells. The initial result of this is the same, however; namely, the release of sub-normal quantities of T4 to the blood stream.

In order for long-term effects to appear as a result of the low-iodide diet or the administration of anti-thyroid drugs, the stimulus must continue. On the other hand, once the ionizing radiation has done its damage to the thyroid cells,

* The basis for this discussion is chiefly the work of Conard and coworkers, particularly Robbins, Rall and Conard (1967) and Conard (1970), and the discussion of thyroid function in Appendix E preceding.

the stage has been set for the long-term effects. For all three initial stimuli, however, the course following serious reduction in output T4 is about the same.

Lowered T4 levels in the blood lead to pituitary production of TSH, resulting next in (over-) stimulation of the thyroid. If thyroid stimulation is not severe or long-lasting (i.e., long-term overstimulation), this may solve the problem.* In a "borderline" gland, however, a cycling effect may ensue. Some cells or areas of the thyroid may atrophy, while those cells that respond to the continuous TSH stimulation grow selectively and form separate nodules or a diffuse growth of the gland (generalized hyperplasia, or goiter). The continuing overstimulation may also lead to a mutant form (clones of cells) that is malignant.

Ionizing-radiation-induced functional pathology of the thyroid can be in: (1) iodide (intake) transport within the gland; or (2) organification to hormones; or (3) both 1 and 2. If the failure is in organification alone, then the gland may still take up iodide, but not convert it. Standard uptake tests on humans may therefore still indicate normal functioning, even though there is trouble. Even if both functions are apparently unaffected (no symptoms and no clinical-test abnormalities), late onset of hypothyroidism may ensue, due to grave but undiscovered injury to some thyroid cells. One possible mechanism for this is lethal mutations in chromosomes of thyroid epithelial cells, unaccompanied by damage to the limited number of genes regulating thyroid-cell function.

* Where thyroid function has been reduced or lost completely, continuing administration of the appropriate quantity of T4 (lifelong, if necessary) provides a sufficient replacement for the lost function, and thus turns off the overproduction of TSH by the pituitary. In this manner, the pathology mechanism is inactivated, and no further damage ensues.

A body of information on the long-term effects of ionizing radiation has been built up as a result of medical use of radioiodine in diagnostics and therapy (Robbins, et al., 1967). In therapeutic applications to be described, massive dosages of I-131 have been used (since the first availability of I-131 in large quantities in the mid 1940's) for destruction of part or all of the thyroid. This is made possible by the selective uptake of iodine in the gland; for example, the circulating I-131 may deliver only 10 rad to the blood*, while the deposited radionuclide delivers more than 50,000 rad to the thyroid. For the cure of intractable angina pectoris, or for the destruction of a potentially metastasizing thyroid carcinoma, thyroid doses of 50,000 to 75,000 rad have been used to destroy the thyroid completely. The sequence of events here is: (1) acute radiation injury; (2) inflammation of the gland; (3) tissue destruction; and (4) fibrotic healing, with no retention of function. Lesser dosages of I-131 have been used in the treatment of hyperplastic thyroids in hyperthyroidism; for example, thyroid doses of the order of 10,000 rad from deposited I-131 have been used to destroy only a fraction of the thyroid cells, leaving sufficient function to achieve euthyroidism. Patients given such treatment have developed an observed accrual of hypothyroidism at a rate of ca. 2% per year. This could be the result of cumulative cell death resulting from the lethal mutations mentioned above, in concert with the normal cell-division rate in growing children or the very slow cell-division rate in adults.

According to Robbins, et al., "In the spectrum of radiation dosage to thyroid cells, one might expect to find the situation in which the cell's function is partially impaired but its growth potential is not. Alternatively, unequal damage to cells in the same gland may result in some with impaired function and growth potential and others with less severe injury. The net result could be the development of mild hypothyroidism, or

* In a study involving a very large population, Saenger, et al. (1968, 1971) found no excess incidence of leukemia in hyperthyroid patients treated with I-131 (over those treated surgically).

the maintenance of euthyroidism only as a result of continued overstimulation by TSH. Under such circumstances, those cells capable of responding may grow and multiply. Furthermore, if the radiation has produced a non-lethal mutation, the progeny of the cell may be abnormal....Radiation injury in general may be expressed as the product of the degree of cell damage times the mitosis rate of the cells."

This suggested mode of development is compatible with known results of intentional or accidental uptake of radioiodines or of delivery of penetrating ionizing radiation to the thyroid, namely:

- (1) Long latency period for appearance of neoplasms.*
- (2) Enhanced effects in children, with their high mitosis rate.

The importance of the homeostatic mechanism in determining the time of appearance of the final damage is evidenced by the relatively high rate of appearance of damage during puberty or pregnancy, when metabolic demands are high. (The importance of the homeostatic mechanism in general is indicated also by the observations of Vasilenko and Klassovskii (1971), who found in studies on rats that damage to the thyroid from uptake of radioiodines was accompanied by benign lesions (adenomas) of the pituitary gland. This result, seen over a wide range of thyroid doses, cannot be due to direct irradiation of the pituitary, which is insignificant, but can result only from functional

* However, Kalinin, Odinokova and Talantov (1971) have shown, by means of observations on autopsied small experimental animals (mice, rats, rabbits) that biochemical changes can be seen in the thyroid within seconds after uptake of I-131 in the therapeutic range (but well below dosages for total destruction), and that morphologic changes in the thyroid can be noted within hours.

overstimulation of that gland following unbalancing of the homeostatic mechanism.) Again, continuing administration of suppressive doses of thyroid hormone in humans or experimental animals prevents both goiter (adenoma) and carcinoma in a hypothyroid situation.

As has been indicated, both benign and malignant thyroid neoplasms can be produced by ionizing radiation. In both humans and experimental animals, the rate of incidence of malignant tumors, per rad to the thyroid, is lower than that for the benign tumors. This will be examined quantitatively in the next section. The benign tumors in humans are usually generalized hyperplasia (diffuse adenomatous goiter) or localized adenomatous nodules, single or multiple. The malignant tumors are almost always carcinomas.*

The medical history of the Marshall Island children and adults who developed benign thyroid neoplasms indicates that the treatment of choice in children with even benign neoplasms is surgery (partial or total thyroidectomy). The success rate following surgery for benign nodules is very close to 100%. In a recent review of thyroid carcinoma mortality risks (Otway and Erdmann, 1970), time of incidence and mortality risks for different types of thyroid cancer are presented. Papillary carcinoma, which peaks in the third and fourth decades of life, accounts

* Vasilenko and Klassovskii (1971a) have investigated the pathology induced by uptake of the radioiodines in rats and dogs (see also Vasilenko and Klassovskii (1971) and Klassovskii (1971)). They found that some of the benign tumors (adenomas) were hormonally active, others hormonally inactive. Most adenomas of the follicular and papillar structure of the thyroid were active, while all adenomas of the parenchymatous structure were inactive. The malignant tumors, consisting of adenocarcinomas and adenopapillar and undifferentiated cancer, were all invasive, metastasizing to the larynx and to the muscles and veins of the neck; 30% of them metastasized to the lung also.

for half of the adult thyroid cancers and 70% of those found in children. It may exist for several decades without apparent harm to the victim. About 10% of the papillary cancers are fatal. Follicular carcinoma, peaking in the fifth decade and representing about 25% of the thyroid cancers, has a mortality rate of about 33%. The undifferentiated (anaplastic) tumors, which are found mostly in individuals past 60 years, are of the rapidly growing, invasive variety, and are fatal in about 76% of the cases. The high fatality rates for thyroid cancer, coupled with the possibility that benign nodules will transform into malignant varieties, argue for surgical treatment of benign nodules, especially because of the high success rate for such surgery.

F.2. Risk of Neoplasm Formation

Accurate prediction of late thyroid casualties in the civil-defense context requires a reasonably accurate method of predicting both: (1) dose to the thyroid; and (2) dose-vs.-effect relationships, the latter for late development (10-15 years later) of both benign and malignant nodules. These dose-vs.-effect relationships are the subject of this section. The recent medical history of the Marshallese themselves, particularly the children, provides some information on risk (dose/effects); however, as the discussion in Section III of the main report shows, the thyroid-dose data are not very reliable.

The most useful literature data, as summarized by Conard, et al. (BNL-50220) (1970), is on the incidence of thyroid nodules and cancer following therapeutic x-irradiation of the neck region; this development, particularly when the irradiation occurs in infancy and childhood, is well documented.* In the

* Pincus, R.A., Reichlin, S. and Hempelmann, L.H., Ann. Intern. Med. 66, 1154 (1967); Toyooka, E.T., Pifer, J.W. and Hempelmann, L.H., J. Nat. Cancer Inst. 31, 1379 (1963); and Hempelmann, L.H., Science 160, 159 (1968); see also Pifer and Hempelmann (1964).

days before the delayed consequences of thyroid irradiation were known, very young children were treated by x-irradiation of the cervical region, for disorders like thymic hypertrophy and even tonsillitis (Robbins, et al., 1967).

Development of thyroid lesions from radioiodine uptake has been noted in animals, but much less frequently in humans. Conard, et al. summarize the results of Sheline, et al. (Sheline, G. E., Lindsay, S., McCormack, K. R. and Galante, M., J. Clin. Endocrinol. Metab. 22, 8 (1962)), who reported 8 cases of nodular goiter in a follow-up study of 250 individuals thus treated for hyperthyroidism. Of the eight, 6 had been given the radioiodine (I-131) therapy before 20 years of age, and of these 4 had had the treatment prior to age 10. If one considers that therapeutic doses to the thyroid from radioiodine administration are in the order of thousands of rads, sometimes tens of thousands, Sheline's reported incidence rate appears low (compared, for example, to the extremely high incidence in the Marshallese children who received of the order of 1000 rad, mostly from radioiodine uptake)*. Possible explanations for the apparent discrepancy will be developed later in this Appendix.

Risk per rad is usually expressed on the basis of measured or expected incidence of neoplasms per 10^6 persons per rad per year** a basis that is reasonable in the medical or public-health

* Conard, et al. note that the medical profession has generally considered radioiodine exposure to be less effective than x-irradiation in producing thyroid neoplasms, primarily because few thyroid tumors have been seen following radioiodine therapy (they quote L. W. Sloan and V. K. Frantz, "Thyroid cancer: clinical aspects," in "The Thyroid," 2nd Ed., S. C. Werner, Editor, Harper and Row, New York, 1962).

** Use of the concept means implicit acceptance of a linear dose/effect response, with no threshold. Such an approach is generally accepted as a conservative basis for assessment of the consequences of exposure to even low levels of ionizing radiation.

context, where a large population is receiving doses that are spread over time, with onset of pathology also spreading over time. This risk-per-rad-per year (per 10^6 persons) statistic may be less useful in the nuclear-explosion context, where (1) the dose is received by the entire affected population at about the same time; and (2) the onset of pathology may be bunched in time after a long delay, as it was for the Marshallese children. In such a situation, it may be more realistic simply to talk about (ultimate) risk per 10^6 persons per rad. Either basis, however, can be calculated easily from a given set of dose/effects data (if the period of observation embraces the appropriate latency period).

We consider first the risk for benign tumors. Pincus, et al., and Hempelmann (op. cit., as quoted by Conard, et al.) determined risk per rad per year for two groups of patients who had been exposed when young to x-irradiation of the neck region. For one group they found an incidence of thyroid nodularity (benign lesions) of 24 cases per 10^6 persons per rad per year; for the other the figure was 64. If the average value of 44 is applied to an average thyroid dose of 1000 rad (that experienced by the Marshallese children under 10 years of age at the time of exposure^{*}) and multiplied by the 15-year period during which nodularity in the Marshallese children was observed,^{**} the predicted total incidence is 660,000 cases per 10^6 persons so irradiated, or 66%. This compares to an

* Conard, et al. and other authors interpret James' (1964) dose calculation to be 1000 rad (close to the geometric or the arithmetic mean of the extremes of the 700-1400 rad range estimated by James) from radioiodines plus 175 rad whole-body; the present author, however, interpret's James' average of 1000 rad to include the 175 rad whole-body (see Section 3.2). The difference is not significant in the evaluation; however, it is pointed out here to prevent confusion between the two resulting sets of predictions.

** Even though development of palpable nodules was bunched toward the end of the period.

observed incidence of 17 out of 19, or 90%. (The upper risk-per-rad-per year figure of 64 leads to prediction of an incidence of 96%).

The risk of thyroid cancer is much lower than that of benign nodules. For example, Robbins, et al. (1967) (op. cit.) present information (three earlier reports) on thyroid carcinoma in children, resulting from x-irradiation of the cervical region for thymic hypertrophy or tonsillitis. Thyroid doses in the range 90-1300 rad were involved. 0.5% of the subjects developed malignant tumors; the average latent period for tumor development was 11 years. If the average dose is taken to be 700 rad, an approximate value for the risk is 0.65 cases per 10^6 persons per rad per year. Otway and Erdmann (1970) (op. cit.) present several estimates of thyroid-carcinoma risk. Specific quoted estimates for x-irradiation of children, in (ultimate) carcinomas per 10^6 persons per rad, are:

1. 35
2. 10 - 30 (20 years at risk)
3. 10 - 20 (same basic data as in No. 2)
4. 60 (25 years at risk). Here, the authors cited by Otway and Erdmann had estimated tissue dose rather than air dose. The 2,878 children in this prospective study had all been irradiated before 6 months of age, when the thyroid may be at its most sensitive.
5. 100 (same data as in 4, interpreted by new authors).

Thus, thyroid-carcinoma risks for children were in the range 0.5 - 4. cases per 10^6 persons per rad per year, or factors of 10 to 100 below the risks for benign lesions of the thyroid. The risks for many kinds of cancer, including leukemia, resulting from ionizing-radiation doses to the appropriate organs are in the 1-case-per- 10^6 -persons-per-rad-per-year area.

Otway and Erdmann interpret some limited additional data to mean that thyroid-carcinoma risks for x-irradiated adults are lower than those for children; the cited data imply a ratio of about 1/8. They state, "It appears that the child's thyroid is considerably more radiosensitive than the adult's - perhaps by a factor of 5 for equal radiation exposures."*

F.3. Apparent Discrepancies Associated with Radiation Type

It has been noted in the previous section that radioiodine (I-131) exposure has generally been considered by the medical profession to be less effective than x-irradiation in producing neoplasms.** Conard, et al. (BNL-50220 (1970)), noting that thyroid tumors had not been seen in large numbers following administration of therapeutic dosages of I-131 (5,000 - 10,000 rad in treatment of hyperthyroidism) in the past, concluded that such doses are "probably so destructive that they preclude proliferative activity and malignant transformation." Such an argument is consistent with known dose effects in skin-cancer and breast-cancer production in rats, sheep and other experimental animals, in which there appears to be an optimal dose for cancer; i.e., an incidence-probability vs. dose function

* On the other hand, Sampson, Key, Buncher and Iijima (1969), in an examination of Hiroshima/Nagasaki data, could find "no indication that dose-prevalence relations differed between the youngest and oldest age groups"; however, their dose-effect difference statistics for the younger-than-20 age group did not reach significance ($p < 0.14$), because of the small number of cases.

** Some authors (for example, Hempelmann (1968)) have accepted the concept that beta radiation in general is only 1/15 to 1/10 as efficient as x-irradiation in producing thyroid neoplasms, and therefore they (and others, including Conard (1970a) and LeRoy (1970)) found the extent of the pathology in the Marshall-ese children to be inconsistent with the (relatively low) reported dose from radioiodines.

that rises, perhaps linearly, to a maximum at some fairly high dose, but then drops steeply with further increase in dose, because cell destruction becomes so severe that there is no focus for mutation. Further support is provided by the work of Vasilenko and Klassovskii (1971 and 1971a) who found, in experiments on rats, that a thyroid dose of 5000 rad from I-131 intake led to the peak tumor-production rate of 94%, 50% of these tumors being malignant (doses of 150,000 rad from the same source producing massive atrophic and dystrophic changes in the thyroid, but no tumors).

Another possible explanation is that therapeutic dosages are designed to reduce hyperthyroid function to the euthyroid state; if successful, they have restored homeostatic balance, and therefore further overstimulation by TSH, presumably the trigger for neoplasm induction, does not occur. Continued administration of thyroid hormone provides further protection. E. A. Tompkins (1970) has unpublished data in support of this idea.

However, neither of these explanations accounts for the fact that there is no recorded association of neoplasm development with earlier administration of diagnostic dosages of I-131 (to patients who were obviously not all hyperthyroid). With modern techniques, dosages of the order of 1 μ Ci (leading to doses of the order of 17 rad to the child thyroid) are sufficient for uptake tests. However, in the earliest days of radioiodine diagnostics, dosages of tens of μ Ci or more (leading to thyroid doses of hundreds of rad, or more) were common.* Furthermore, as Bizzell pointed out recently (Mann and Garfinkel, 1970), activity calibrations in hospitals up to about 1950 were quite inaccurate; in 1949, Manov found variations by factors of 3 to 4 in activity measurements. It was only following these intercomparisons that administered I-131 activities were brought

* LeRoy (1970), for example, has estimated that tens of thousands of children received diagnostic doses in the 20-50 μ Ci range. E. A. Tompkins (1970) quotes the early-day dose range as 4-2500 rad.

into better agreement with NBS calibrations. Thus, it is not inconceivable that some early patients received close to or more than 1000 rad from diagnostic dosages of I-131.

A closer approach to resolution of the discrepancy is provided by Otway and Erdmann (1970): "The ^{131}I is deposited in the thyroid and therefore delivers an unevenly distributed dose at a low rate. An externally delivered X-ray dose is spatially well distributed and is delivered at a higher dose rate. Because of these differences X rays are more effective per unit dose in producing thyroid malignancy."^{*}

The final answer appears to have been given by Klassovskii (1971).^{**} Klassovskii starts with a statement to the effect that it is established that the "RBE" for x- and gamma-rays in the thyroid is 10-20 times higher than that for incorporated I-131, and that up to now the reason for this has been obscure. He then claims that his experiments on rats show that the discrepancy is due to the uneven dose distribution from the (relatively soft) beta rays of I-131, and that no such discrepancy exists for the other radioiodines, with their harder beta emissions. The experiments were done with I-131 alone and with a radioiodine mixture in which 80% (basis unspecified, but presumably activity, in Ci) was I-132 plus I-133. The beta energies of the latter two nuclides are said to be about 2.5 times that of I-131, the ranges to be about 3-4 times greater than that of I-131. (See Appendix C for details of energies and ranges of the radioiodines).

* While it is well known that dose rates are important in determining the early effects of a given total dose, it is not clear that such an importance has been established for late effects, such as neoplasms.

** This reference, like the other 1971 references to the work of that author and his colleagues (Vasilenko and Kalinin as senior authors), is a recent translation of a Soviet literature reference from an earlier period, in this case 1967.

Klassovskii used historadiography and quantitative autoradiography to determine the spatial distribution of the radioiodines in the thyroid. He found that iodine concentrates almost entirely in the colloid of the follicles and that, because the distances between the follicles are from tens to hundreds of micrometers, the dose from the weaker betas of the I-131 is concentrated almost entirely in the follicles, whereas that from the stronger betas of the other radioiodines is more uniformly distributed throughout the thyroid.* He then measured (or calculated) "coefficients of inequality of dosage distribution" (undefined, at least in the translation), and stated that those for I-131 were 3-5 times higher than those for the 80% I-132/I-133 mixture. (In another report (Klassovskii, Vasilenko and Terekhov, 1971) he points out that I-131 has an average beta energy, \bar{E} , of about 0.2 Mev, and proposes a "coefficient of distribution of dose" (something like an "RBE") that is 1.0 for $\bar{E} > 0.2$ Mev, and 0.1 for $E \leq 0.2$ Mev).

Pathological effects on the thyroid were characterized by histological changes, uptake-capacity measurements over time, degree of atrophy, and tumor effects, the latter in the pituitary gland as well as the thyroid. The over-all results showed the 80% mixture to be 10-25 times more effective than I-131 for the same thyroid-averaged dose.

The effect of non-uniform dose distribution in limiting the effectiveness of a given dose from I-131 was explained, finally, on the basis that the follicular colloid, where that dose is primarily absorbed, is much less radiosensitive than the parenchymatous formations of the gland and also, in contrast to those formations, is not significant in the regeneration of the gland.

* Anspaugh (1965/1966), on the other hand, has calculated that the I-131 dose, even in a 1.7 g child's thyroid, is uniformly distributed. While the basis for the disagreement has not been investigated here, the experimental results of Klassovskii appear to support the argument for inhomogeneity.

Supporting information for this claim appears in Vasilenko and Klassovskii (1971), reporting experiments on dogs, with a radioiodine mixture that was 12% I-131, 38% I-132 and 50% I-133, and on rats, with this mixture and with I-131 alone.*

Klassovskii (Klassovskii, Vasilenko and Terekhov, 1971) cautions the reader against unwarranted extrapolations from animal experiments to man, but points out that if allowance is made for the known differences in (1) organ size and level of differentiation; (2) metabolic rate; (3) life span; and (4) latency period for neoplasm induction; then reasonable extrapolations can be made. For example, a 1-yr latency period in the rat implies a 25-yr latency period in the adult human.

Examination of Appendix C of this report, which presents the characteristics of the radioiodine mixture likely to be found in fallout (more specifically, in inhaled iodine in a fallout field), will show that Klassovskii's experimental mixture has radiological properties very close to those of the fallout threat, particularly at the very early times post-fallout, when I-131 has not yet reached its peak relative contribution. Thus, the pathology in the Marshallese children is now explicable; with a thyroid dose of about 1000 rad from the radioiodine mixture (as calculated by James), they were actually exposed to a thyroid dose equivalent to perhaps 10,000 rad from I-131 alone. (Or, to put it another way, a 1000-rad dose from mixed radioiodines is equivalent to a 1000-rad dose from x-irradiation, so that the Marshallese tumor-incidence rates should be expected to fall on the straight-line curve of incidence vs. x-irradiation dose, as presented by Hempelmann (1968) (as they indeed do)).

* NOTE ADDED IN PROOF: Somewhat earlier, Saenger and his colleagues had reported the possibility that the apparently reduced effectiveness of ingested I-131 in producing thyroid carcinoma was due to uneven distribution of I-131 beta dose in thyroid tissue (SAENGER, E.L., SELTZER, R.A., STERLING, T.D. and KEREIAKES, J.G. (1963), "Carcinogenic Effects of I¹³¹ Compared with X-Irradiation - a Review," Health Physics 9, 1371).