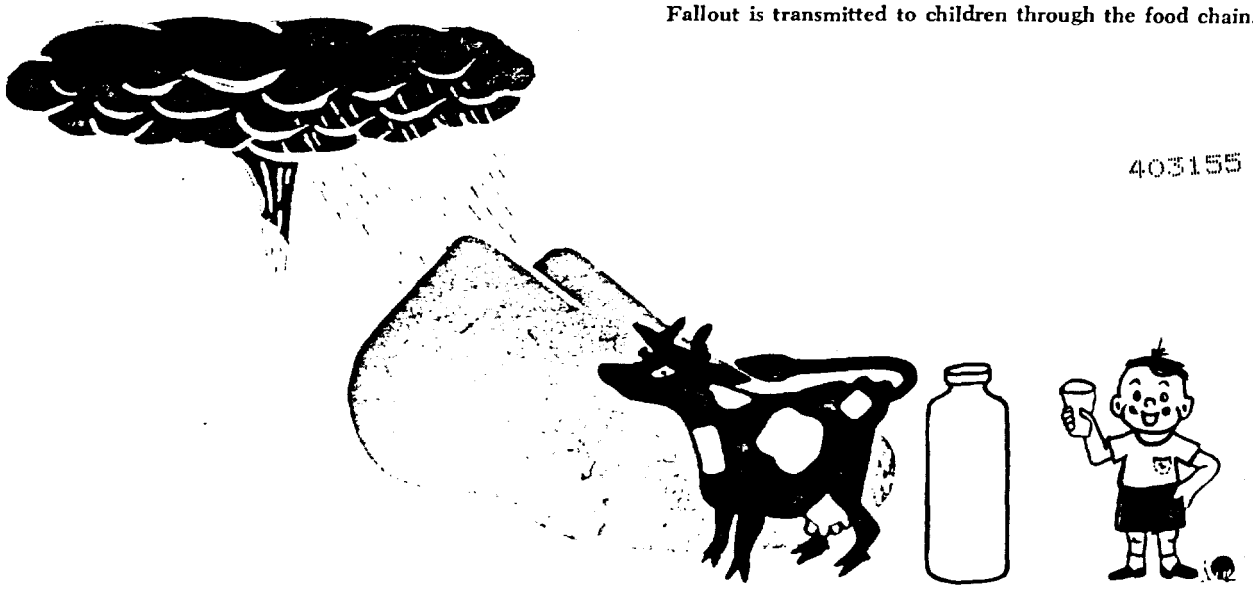

Fallout is transmitted to children through the food chain.

THYROID IRRADIATION IN UTAH INFANTS EXPOSED TO IODINE 131

By CHARLES W. MAYS

THE INADVERTENT EXPOSURE of about 250 thousand Utah infants to average thyroid doses estimated at 1.3-10 rads may provide a unique opportunity to establish the effects of low doses of iodine 131 irradiation in children.

Iodine 131 is produced in nuclear explosions. If cows eat forage which is contaminated with fresh fallout, iodine 131 appears in their milk. If a child drinks this radioactive milk the iodine 131 concentrates in his thyroid gland and irradiates it. Infants are considered to be the critical members of the population. The transmission of fallout to children is illustrated in the figure.

Dr. Robert C. Pendleton (Associate Professor of Molecular and Genetic Biology, University of Utah) was the first to discover that fallout from the Nevada nuclear tests of 1962 would cause significant contamination from radioactive iodine 131 in Utah.[1] Both our measurements,[1] and those of the U.S. Public Health Service,[2] showed that iodine 131 levels in Utah milk exceeded the radiation protection guide of the Federal Radiation Council.[3] Following Pendleton's vigorous urging that steps be taken to reduce the exposure, the Utah State Department of Health recommended: transfer of cows from highly contaminated pasture to stored feed; and diversion of highly contaminated milk from the fresh market.[4] This protective action set a precedent: it marked the first official attempt in this country to prevent the intake of fallout-contaminated food.

In analyzing the 1962 incident I became deeply concerned about the exposures from the previous years of testing. Unknown to me at the time, this problem was also being investigated through different approaches by Dr. Harold A. Knapp, then with the Atomic Energy Commission (AEC), and by members of the St. Louis Citizens' Committee for Nuclear Information (C.N.I.). Working independently, we all came to the same conclusion: significant exposures were indicated. For example, thyroid doses to infants in St. George, Utah, from the "Harry" shot of 19 May 1953, were estimated at 120-440 rads* by Knapp; 100-700 rads by C.N.I., and using a less reliable method, 68 rads by myself.[5]

The problem was complicated enormously by the fact that iodine 131 had not been properly recognized as a fallout hazard during the early years of testing (1951, 1952, 1953 and 1955) and, unfortunately, iodine 131 had not been measured in milk during that time. Therefore, attempts to reconstruct the probable exposures during this period must be indirect because the eight-day iodine 131 has long since decayed (but see Appendix). Knapp used the gamma-ray intensity above a contaminated field as an index of its iodine 131 content,[5] while C.N.I. used this and the beta-particle disintegration rate from fallout collection trays.[5] I used the observed beta activity in the air and the fission yield.[5] Knapp and C.N.I. were concerned chiefly with exposures in Southern Utah near the Nevada Test Site, because that was where the highest individual doses were indicated. My major concern was the North-Central portion of Utah because it contained so many more people.

During the 1963 Congressional Fallout Hearings, Dr. Eric Reiss of C.N.I. and I both urged that a study be made of the irradiated Utah children. This study, supported by the U.S. Public Health Service, is now in progress. Originally, the study had two objectives—to search for medical effects, and to estimate the radiation dosage. The medical phase is progressing well, but the dose program requires much more work. If the dosimetry is not completed soon, the best opportunity to reconstruct the exposures may be gone forever. Memories fade, records are destroyed, and these children will soon reach adulthood and scatter across the nation. Even if no effects appear, the exposures should be evaluated as accurately as possible. If a safe dose exists for iodine 131 in children, proper study of the 250 thousand irradiated Utah infants may provide a unique chance to establish it.

In this paper I present my estimates of lower and higher limits for the average doses received by Utah infants. I do this for two reasons. First, I hope that the uncertainty of these limits will stimulate interest in improving the dosimetry. Secondly, despite dose uncertainties, the indicated exposures are sufficient to justify continued study of the Utah children.

DOSIMETRY

Prior to the 1963 Fallout Hearings, I calculated the number of radiation-induced cancers that might be anticipated in the 250 thousand Utah children exposed to fallout iodine 131 as infants. I withheld publication or public discussion of these predictions primarily because I felt that most of my estimates of dose might be somewhat unreliable. Working in isolation at the time, I was aware of milk measurements of iodine 131 for only one of the seven years of Nevada testing; doses for the other six years I estimated from measurements of beta-activity in the air or from fission yield during the growing season.[5] New information now permits improved estimates for five of the seven test years, and for the remaining two I have set lower and higher limits.

For 1962, our analysis of milk from Pendleton's 39 stations scattered throughout Utah indicated that for the daily consumption of one liter (1.06 quarts) of milk, the average yearly iodine 131 intake was 45 thousand picocuries* assuming a three day delay from milking to consumption.[5] This agreed closely with the 37 thousand picocuries yearly intake reported by the U.S. Public Health Service for the Salt Lake milk pool.[2] The corresponding infant thyroid doses** were 0.77 or 0.63 rads respectively.

TABLE 1
ESTIMATED AVERAGE THYROID DOSES TO ALL UTAH INFANTS

YEAR OF TESTING	DOSE IN RADS LOWER LIMIT		HIGHER LIMIT	METHOD OF DOSE ESTIMATION
1962	0.63	–	0.77	S.L. Milk Pool & Pendleton's Milk Sta.
1958		0.2		USPHS Salt Lake Milk Pool
1957		1.3		USPHS Salt Lake Milk Pool
1955	0.2	–	20	Fission Yield
1953	2	–	12	Field Beta Activity After Shot "Nancy"
1952	3	–	18	Field Beta Activity After Shot "Easy"
1951	0.04	–	4	Fission Yield

For 1958 and 1957, USPHS analysis of the Salt Lake milk pool indicated average yearly intakes of 11 thousand and 74 thousand picocuries of iodine 131 with associated infant thyroid doses of 0.2 and 1.3 rads respectively.[6]

For 1953 and 1952, the beta activity in fallout collection trays at Salt Lake City was 15 million disintegrations per minute per square foot at twelve hours after the detonation of shot "Nancy" on 24 March 1953 and 23 million disintegrations per minute per square foot at twelve hours after the detonation of shot "Easy" on 7 May 1952. Infant thyroid doses have been calculated by C.N.I. as two to twelve rads for this 1953 shot and three to thirteen rads for this 1952 shot (see pages 529-530, ref.5). A number of limitations exist in using these values. First, they are for only one shot during

*A rad is the absorption of 100 ergs of energy per gram of tissue.

*A picocurie is 2.22 disintegrations per minute.

**Assuming 30% uptake in a 2 gram thyroid with a subsequent effective retention half-time of 7.6 days, and the absorption of 0.2 million electron volts of energy per disintegration of iodine 131.

each year; additional shots may have caused additional contamination. Hence the true "upper limit" may exceed my so-called "higher limit." Second, the measurements were made in Salt Lake City, not in the pasture lands; pasture contamination could have been higher or lower. My presumably less reliable estimates (see page 561, ref. 5) for these average yearly exposures were 6.3 rads for 1953 (from the fission yield) and 1.6 or 5.9 rads for 1952 (from the fission yield or air beta activity, respectively).

For 1955 and 1951, I only have my estimates of 2.0 and 0.4 rads from the fission yields of 84 and 18 kilotons during the growing season (see page 561, ref.5). Comparing my approximate fission yield estimates of dose with more reliable methods suggests that the fission estimates averaged over a year of testing might be accurate within a factor of ten. Therefore, I have assigned dose ranges of 0.2-20 rads for 1955 and 0.04-4 rads for 1951. It is not my intent to deceive the reader into believing that the true doses are well established for the years 1955, 1953, 1952 and 1951. More work is needed.

My best estimates of the *average* yearly thyroid doses for Utah infants are summarized in Table 1 for each year of Nevada testing. *Individual* doses were, of course, much higher; Knapp estimated doses of 120-440 rads for infants in St. George, Utah, following the "Harry" shot of 19 May 1953.[5] The dose for our highest station in 1962 was 9-26 times greater than our average.[5]

TABLE 2
INFANT EXPOSURES BY YEAR OF BIRTH

YEAR OF BIRTH	BIRTHS DURING YEAR	AV. THYROID DOSE (RADS) Age 0.5 to 1.5	Age 1.5 to 2.5	TOTAL DOSE
1962				
1961	26,000	0.6 – 0.8		0.6 – 0.8
1960	25,000		0.6 – 0.8	0.6 – 0.8
1959				
1958				
1957	24,000	0.2		0.2
1956	23,000	1.3	0.2	1.5
1955	22,000		1.3	1.3
1954	22,000	0.2 – 20		0.2 – 20
1953	21,000		0.2 – 20	0.2 – 20
1952	21,000	2 – 12		2 – 12
1951	20,000	3 – 18	2 – 12	5 – 30
1950	20,000	0.04 – 4	3 – 18	3 – 22
1949	19,000		0.04 – 4	0.04 – 4
TOTAL	243,000		AVERAGE DOSE	1.3 - 10

Next I tabulate the total number of exposed Utah infants, and compute the average dose for all of them (Table 2). The yearly births were derived from the U.S. Census (1960 and 1950). The average birth time was taken as mid-year, followed by an average delay of about six months before appreciable consumption of fresh cow's milk. Thus, exposures were regarded as beginning at the start of the calendar year immediately following the year of birth. Only the dose during infancy has been computed. However, dose calculations can easily be extended throughout childhood using the method shown in Table 2 and correcting for the increasing mass of the thyroid with age.*

Table 2 indicates that about a quarter of a million Utah infants were exposed to fallout iodine 131, with an indicated average thyroid dose of 1.3-10 rads.** The significance of these exposures is not the size of the average dose (which is small) but in the enormous number of irradiated children.

THYROID CANCERS

The natural occurrence of childhood thyroid cancer is extremely low. Values from Mustacchi and Cutler indicate that by age fifteen years only 25 thyroid cancers are expected to appear per million children.[8] Thus only about six "natural" childhood thyroid cancers are anticipated in the 250 thousand exposed Utah children by age fifteen. The fraction of these so called "natural" cancers which were in fact induced by medical X-rays may be appreciable. In a series of childhood thyroid cancers collected by Winship and Rosvoll, about 80 per cent showed a history of prior irradiation.[9] In the United Kingdom only about three children per million develop thyroid cancer by age fifteen.[10]

X-rays can induce thyroid cancer. About 20-30 years ago, it was common in some hospitals to X-irradiate infants in the neck region for benign conditions. Thyroid cancer has followed in an unpleasantly large number of these exposed children.[9] Beach and Dolphin[11] found reports of 132 post-irradiation thyroid malignancies in the published medical literature; the additional number of unpublished cases remains unknown. They analyzed the relation between incidence and dose in 4673 exposed children for whom the individual doses were obtainable. The incidence of thyroid cancer increased with dose to 1.7 per cent at 500 rads. Assuming incidence proportional to dose, they derived a cumulative lifetime incidence of 35 cancers per million infants each receiving

*The enlargement of the thyroid gland with age reduces its iodine 131 concentration and the resulting radiation dose from a given intake of iodine 131. For example, the intake of one microcurie (1,000,000 picocuries) of iodine 131 gives a seventeen-rad dose to the two-gram thyroid of a one-year old infant, a 6.8-rad dose to the eight-gram thyroid of an eight-year old child, and a 1.7-rad dose to the 20-gram thyroid of an adult. Furthermore, the weight of evidence indicates that the radiation resistance of the thyroid increases with age,[7] although the exact sequence of changes in sensitivity has not yet been established precisely.

**My earlier crude methods (See Ref. 5) yielded an estimated thyroid dose averaging 4.4 rads to this population. I am pleased at the agreement.

one rad. However, they were careful to point out that their analysis did not exclude the possibility of a curved dose-response relation. The data in their figure five can also be interpreted to suggest a "threshold" at perhaps 50-100 rads, below which cancers would not be induced. Perhaps roughly half of the radiation-induced thyroid cancers may appear within the first fifteen years (compare values in Ref. 11 with those in Ref. 12), but this fraction is uncertain.

There is some experimental evidence that iodine 131 is less effective than X-rays in inducing cancer. Doniach found that the iodine 131 dose had to be about ten times greater than an abrupt dose of X-rays to cause equal effects in adult rats.[13] However, adult rats are not infant humans and the relative effectiveness of iodine 131 vs. X-rays in children is yet to be established. Perhaps the iodine 131 exposures in Utah could provide information on this point.

The dose-response relation is very difficult to establish at very low doses because the incidence of effects is so very small. Some radiobiologists feel that cancer induction is not proportional to dose, but that a certain "threshold" level must be exceeded before any cancers can be induced. If such a threshold exists and if the doses fall below this level, no induced cancers will appear. It is entirely possible that no cancers will result from the Utah exposures.

TABLE 3
ESTIMATED THYROID CANCERS
FOR THE 250,000 IRRADIATED UTAH INFANTS

ASSUMPTION	NUMBER OF CANCERS
(A) Iodine 131 effect equals X-ray effect	11 – 88
(B) Effect equals 1/10 of X-ray effect	1 – 9
(C) High Threshold	0
"NATURAL" INCIDENCE BY 15 YEARS OF AGE	6

Theoretical estimates of the number of radiation-induced thyroid cancers will now be calculated for each of the following three assumptions: (A) a linear dose-response with iodine 131 irradiation equally as effective as X-irradiation, and causing a lifetime incidence of 35 thyroid cancers per million infants each receiving one rad; (B) a linear dose response, but iodine 131 irradiation only one tenth as effective as X-irradiation; and (C) a high threshold. Assumption (A) probably sets an upper limit, for it is unlikely that iodine 131 is more effective than X-rays. Assumption (C) certainly sets the lower limit—the number of induced cancers cannot be less than zero! Assumption (B), while between the upper and lower limits, is not necessarily the best estimate but it yields reasonable values for planning the experimental search for thyroid cancer. Estimates of the number of radiation-induced thyroid cancers which might appear during the lifespan of the 250 thousand Utah infants for doses of 1.3-10 rads are compared in Table 3 to their expected natural incidence by age fifteen.

Similarly, it is instructive to calculate the anticipated number of radiation-induced thyroid cancers which might appear during the lifespans of the 565 infants in Washington County, Utah, exposed to higher thyroid doses (estimated at 120-440 rads[5]) following the "Harry" shot of 19 May 1953. These estimates are shown in Table 4.

TABLE 4
ESTIMATED THYROID CANCERS
FOR THE 565 INFANTS NEAR ST. GEORGE IN 1953

ASSUMPTION	NUMBER OF CANCERS
(A) Iodine 131 effect equals X-ray effect	2 – 9
(B) Effect equals 1/10 of X-ray effect	0.2 – 0.9
(C) High Threshold	0
"NATURAL" INCIDENCE BY 15 YEARS OF AGE	0.01

If the additional doses received before birth and in later childhood had been included, the estimated number of radiation-induced cancers would have been even higher than shown in Tables 3 and 4.

DISCUSSION

1) The St. George study has the advantage that the "natural" occurrence of childhood thyroid cancers is extremely unlikely among the 565 infants exposed in 1953. The probability of one natural case is one in one hundred whereas the chance for two natural cases is only one in ten thousand. Thus the observation of any childhood thyroid cancer would seem suggestive of radiation damage, unless it could be shown that other factors render these children exceptionally susceptible.

Is it possible that genetic or environmental factors might increase the "natural" incidence in this region by a factor of one hundred to ten thousand times greater than for the rest of the USA? I do not know. But I do know how to test this hypothesis. Individual estimates should be made of the most probable dose to each St. George child based on source of milk and

thyroid size.* These children should then be classified into graded dose categories. The high-dose children will probably be those who drank local milk as infants from 19 May-19 June 1953 (and to a lesser extent, from 17 March-17 April 1953, and from 31 August-30 September 1957). Low-dose children should be those whose infant years occurred after these times (or long before), plus those who drank powdered milk or uncontaminated fresh milk during these incidents.

If non-radiation factors predispose the children of this region to a high incidence of thyroid abnormalities, these abnormalities should occur *throughout* the dose categories.

However, if the incidence is significantly greater in the higher-dose groups, radiation would be implicated as a causative factor. In a study of this nature it is extremely important to have low-dose subjects from the same region and culture to serve as controls.

In the thirteen years which have elapsed since the incident of 1953, perhaps half of the total cumulative radiation-induced cancers should have appeared among the St. George children; or one—four cases under assumption (A), 0.1-0.4 cases under assumption (B), and no cases under assumption (C). The fact that no thyroid cancers have yet been observed** in these children[14] suggests that assumption (A) overestimates the true effect and therefore constitutes an upper limit. Establishment of an upper limit is of real value, but establishment of the true dose-response relationship would be of even greater importance. To establish the true dose-response relationship, the study needs to be expanded. Note that under assumption (B), less than one radiation-induced cancer is predicted to have occurred before now (1966) among the St. George children.

2) For the entire state of Utah, the estimated number of radiation-induced cancers is five—ten times larger than for the limited St. George area, assuming a linear dose-response relationship (compare Tables 3 and 4).

How might the present study be expanded to obtain additional information? The following plan is suggested:

a) The fallout deposition should be established for each one of the Nevada test shots. The actual deposition of fallout should be evaluated by records of field gamma-ray intensities and fallout tray beta-activities, when available. When measurements are lacking, the deposition on the ground should be estimated from meteorological considerations. Ecological factors could have caused the doses to the northern part of the state to be materially higher than calculated for the early tests. Rainfall is greater in northern Utah, and rain brings down fallout from the air. Also, the studies of Pendleton have shown that plant uptake is higher from wet pastures than from dry.[1] In the 1962 incident, the highest levels of iodine 131 observed in Utah milk were not in the south-west and central farms first traversed by the fallout; the highest observed levels were in the north-east portion of the state, over 400 miles from the Nevada test site.

b) From feeding practices, the transfer of iodine 131 to milk should be estimated for each milkshed. Very little iodine 131 appears in milk when the cattle are on aged stored feed,[1] as, for example, during the winter. Special efforts should be made to estimate the iodine 131 levels in the milk of those dairies which supplied large populations.

c) The populations served by the major dairies should be identified and recorded. Dose estimates to these populations should be made, and the populations classified into graded dose-levels.

d) Procedures should be established to have all thyroid abnormalities reported to a central file where periodically the effects would be correlated with dose-level. Detailed dosimetric reconstructions should be made for those individuals developing thyroid abnormalities, with particular emphasis given to evaluation of prior X-ray therapy. In fact, a retrospective survey should be made to document the prevalence of medical irradiation of Utah infants to determine what effect this might have on the iodine 131 studies. Also, new techniques should be explored for improved estimates of iodine 131 dose (see Appendix).

e) If the Utah study is to yield maximum information, it *must* receive long-term support. Many years may elapse before the appearance of delayed effects of irradiation. If no effects appear, it will be even more imperative to document the study extremely thoroughly, because it is much more difficult to prove that a given dose is safe than to verify that a higher dose is dangerous. However, the establishment of a safe dose is of much greater importance.

3) While thyroid cancer has been of major concern in this paper, other conditions should be considered, such as the more frequent benign thyroid tumors. In a study (Starr et. al.) of 73 children and adolescents treated with iodine 131 for hyperthyroidism (an overactive thyroid) and followed for two—fourteen years, there were five thyroid tumors of which only one (a papillary adenocarcinoma) was malignant.[15] In a different study, out of eighteen children treated with iodine 131 for hyperthyroidism, six thyroid tumors were observed five—fourteen years later.[16] More cases may yet appear after longer follow-up times.

*The approximate size of a child's thyroid at iodine 131 intake can be calculated from his age at intake.

**The children who moved from this area before the recent survey have not been examined.

In the Marshall Islands, twelve children under five years of age were exposed to heavy fallout in 1954. Their total body gamma-ray dose was about 175 rads while their thyroid dose from iodine 131 is estimated at roughly one thousand rads. Of these twelve children, two have become hypothyroid with marked growth retardation, while five others have developed adenomatoid goiters. No thyroid abnormalities have been observed in the 75 unexposed comparison children from nearby islands.[17]

Concerning the St. George children, it is uncertain whether their incidences of non-malignant thyroid abnormalities, such as thyroiditis and thyroid nodules, were influenced by iodine 131 irradiation.[14] It is also unknown whether these abnormalities might later have progressed into cancers if not discovered in the course of the Utah study and treated surgically.

CONCLUSION

What are the effects of low doses of iodine 131 in children? How safe is the Federal Radiation Council's new Protective Action Guide for thyroid doses of 30 rads to individual infants and ten rads to groups of infants?[18] Proper study of the Utah children might provide at least partial answers to these questions.

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APPENDIX

OTHER POSSIBILITIES OF IODINE 131 DOSE EVALUATION

The central problem in this study is one of reconstruction in the absence of complete data. Lacking direct measurements of iodine 131 levels during the 1951-55 tests, estimates have been based on retrospective calculations of the available data—gross beta and gamma levels—and on assumptions on the relation of these levels to the iodine 131 levels.

It is fully recognized that at this point serious objections may be raised. Firstly, the yields of various radioactive isotopes have to be assumed in the absence of full information on the major nature of the fissionable material used in each explosion. Secondly, our knowledge of the fractionation process is incomplete. Nevertheless it may be useful to pursue these calculations through as many independent methods as we can find. Not all these methods may prove practicable after detailed examination, but we cannot afford to reject suggestions too quickly.

A new possibility has been pointed out to me by Merril Eisenbud (Professor of Environmental Medicine, New York University Medical Center, Tuxedo, New York). The concept is described by Edwards.[19] Both iodine 131 and iodine 129 result from fission. While the iodine 131 has decayed because of its short eight-day half-life, the iodine 129 with its 16-million year half-life has hardly decayed at all. Thus, the iodine 129 content in the thyroids of people who died soon after an early nuclear test might indicate the iodine 131 dose which was received. It may be possible to obtain sufficient pathological tissue preserved from autopsies performed at that time, and thus gauge the iodine 129 levels.

For example, one microcurie of iodine 131 consists of 3.72×10^{10} atoms. The fission yields have been estimated (Weaver, et.al.) at about 3.0 per cent for the iodine 129 chain, and 4.6 per cent for the iodine 131 chain, from plutonium 239 fission by fission-spectrum neutrons.[20] Thus, there are about 2.4×10^{10} atoms of iodine 129 associated with an *initial* iodine 131 activity of one microcurie.

To assay iodine 129 it can be activated to iodine 130 by neutron bombardment.[19] A saturation activity of 73 disintegrations per second of iodine 130 (easily counted) results from the bombardment of 2.4×10^{10} atoms of iodine 129 (cross-section 30×10^{-24} cm^2 per atom) by a flux of 10^{14} neutrons per cm^2 each second.[19]

Iodine 129 atoms are removed from the adult human thyroid with a biological half-time of about three months, although this varies from person to person.[21]

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Therefore, the appropriate thyroid samples would be from thyroid tissue obtained after a nuclear test of interest. Additional pathological samples obtained just prior to the test of interest would be needed to correct for environmental iodine 129. In this connection, note that little stratospheric fallout was present during the 1951-55 tests for which dose estimates are most urgently needed.

James Arnold (Kansas City General Hospital, Kansas City, Missouri) has pointed out that if milk samples taken from contaminated areas at the time of contamination can be located, these might be suitable for iodine 129 analysis.

A final suggestion: it might be possible to use strontium 90 or cesium 137 as radioactive indicators. All are retained on pasture vegetation with a biological half-time of about thirteen days.[22] All are absorbed by the cow, transferred to milk and assimilated by man. Strontium 90 has the advantage that it is not vaporized during sample ashing. Cesium 137 is easily detected non-destructively by gamma counting. The biological half-times of retained cesium 137 and iodine 129 in the bodies of adult humans are roughly the same, (three months). For example, cesium 137 can be evaluated by total body counting without the necessity of removing samples. The cesium 137 to potassium 40 ratio can be evaluated even in poorly preserved tissue.

Once again, it must be acknowledged that the above methods are possibilities rather than proven methods of procedure. But, if practicable, any or all of these methods could be of great value in establishing better estimates of dose.

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