Task 1 Extended Summary
Iodine-131 Releases from Radioactive Lanthanum Processing at the X-10 Site in Oak Ridge, Tennessee (1944-1956)
An Assessment of Quantities Released, Off-Site Radiation Doses, and Potential Excess Risks of Thyroid Cancer

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Introduction
Oak Ridge National Laboratory (ORNL), originally known by the code name X-10, released radioactive iodine ($^{131}$I) to the air from 1944 through 1956 as it processed spent nuclear reactor fuel. The process recovered radioactive lanthanum (RaLa) to support weapons development at Los Alamos, for atmospheric radiation tracking, and for radiation warfare experiments. Iodine concentrates in the thyroid gland. Therefore, the health concerns stemming from exposure to $^{131}$I include various diseases of the thyroid such as thyroid cancer and non-neoplastic abnormalities such as autoimmune hypothyroidism, Hashimoto's thyroiditis, and Graves disease.

This study investigated the health outcomes from the releases of $^{131}$I between 1944 and 1956 at 41 representative locations within 38 kilometers of ORNL. Communities within this region include the City of Oak Ridge, Clinton, Oliver Springs, Kingston, Harriman, Lenoir City, Sweetwater, Maryville, and Knoxville. At each of these locations, the risks of developing thyroid cancer, the relative risk with respect to an unexposed population, and the probability of causation for diagnosed cases of thyroid cancer were determined for individuals of both genders and of various age groups at the time of exposure. The numbers of cancers that could have resulted from exposure to the $^{131}$I releases within 38-km, 100-km, and 200-km radii of the X-10 facility were also estimated. The overall impact of $^{131}$I exposure from the combined contributions of X-10 releases and fallout from atmospheric testing of nuclear weapons at the Nevada Test Site was also evaluated. The results of this study were found to be comparable to results from similar studies conducted at other sites in the United States. The probability of occurrence of non-neoplastic thyroid disease in the 38-km vicinity of X-10 has been discussed on the basis of new evidence on the doses of radiation required to induce such diseases.

A number of recommendations have been made in this report that can be used in follow-up work to reduce the uncertainty in dose and risk and to identify and eliminate any remaining sources of possible bias. Important components of the study, the processes addressed within each component, and intermediate results are summarized below, followed by a discussion of the overall health impacts of the $^{131}$I releases from X-10.

Source Term
Over the 13-year period of the RaLa operations at X-10, approximately 30,000 reactor fuel slugs were dissolved in about 731 batches during the process of separating over 19,000 TBq (1TBq = $10^{12}$ Bq; approximately 500,000 Ci) of radioactive barium as a source of $^{140}$La for shipment to Los Alamos. The analysis of the source term (the amount of $^{131}$I released to the atmosphere) involved estimation of the annual quantities of radiiodine released from vents and openings in Buildings 706-C and 706-D and from the X-10 stacks 3020 and 3039, as a result of routine normal and off-normal conditions. For a large
accident that occurred on April 29, 1954, release estimates were made for each half-hour period.

The most important source of radiiodine releases from RaLa processing was exhaust ("off gas") from the slug dissolver. Volatile gases, which included radiiodine, were withdrawn from the dissolver by negative pressure, passed through a condenser and a chemical scrubber, and then routed through piping to the 200-foot tall 3020 stack. After March 1950, exhausts from the chemical scrubber were routed to a central treatment facility in which contaminated air was passed through an electrostatic precipitator and particulate filters prior to release up the 250-foot tall 3039 stack.

The iodine and barium content was estimated for irradiated fuel slugs from the X-10 Clinton Pile (Graphite Reactor) and the reactors at Hanford. For each of the 731 dissolving batches, the potential for "direct" releases of untreated exhaust to the atmosphere through building vents, windows, and other openings was evaluated. Because of the absence of monitoring data, expert opinion was extensively used to quantify the $^{131}$I collection efficiency of the condenser and caustic scrubber for routine operations. Expert opinion was also used to quantify the potential degradation of collection efficiencies during the April 1954 accident.

Approximately 8,800 to 42,000 Ci (0.3 to 1.6 PBq; 1 PBq = $10^{15}$ Bq) of $^{131}$I was released between 1944 and 1956, of which 6,300 to 36,000 Ci (0.23 to 1.3 PBq)) was in the elemental (most environmentally reactive) form of iodine; the remainder was in the nonreactive volatile organic form. The largest releases occurred between 1952 and 1956 when the freshly spent uranium fuel slugs came from Hanford reactors. The April 29, 1954, accident, which lasted a total of 2.5 hours, released 105 to 500 Ci (3.9 to 21 TBq), accounting for about 6.5% of the total releases for 1954.

**Atmospheric Dispersion**

After being released into the atmosphere, $^{131}$I was transported by the prevailing winds. A fraction of the iodine released in the elemental (reactive) form was chemically transformed during transport to particulate and organic (nonreactive) forms within a few kilometers of the RaLa processing facility. The ground-level concentration of $^{131}$I in air is affected by several factors including the distance of the location of interest from the RaLa processing facility, the dilution of the concentrations in the air during atmospheric dispersion or mixing, the depletion of iodine from air by the processes of wet and dry deposition, and the chemical form in which iodine is present.

Annual average ground-level concentrations of $^{131}$I for the routine releases and time-integrated ground-level concentrations of $^{131}$I for the 1954 accident were estimated using SORAMI, a mathematical model that accounts for all of the relevant processes that are important during the transport of $^{131}$I in the
atmosphere. The model was benchmarked using another public-domain model. The model was validated using site-specific release and monitoring data, and the validation results indicate that the model predictions were within a factor of 2 of the annual average measurements.

For the analysis of routine releases between 1944 and 1956, electronic data collected at X-10 from 1987 to 1996 were analyzed statistically to generate surrogate hourly meteorological data. For the April 29, 1954, accident, the SORAMI model was supplied with half-hourly meteorological data obtained from documented records of the specific meteorology prevailing at the time of the accident. Uncertainties in all input parameters to SORAMI were quantified before the concentrations were estimated for each of the 41 locations of interest.

**Transfer from Air to Vegetation**

Consumption of contaminated milk and meat from cattle grazing on pastureland contaminated by the deposition of $^{131}$I from air is one of the most important pathways by which $^{131}$I enters the human body. Once $^{131}$I is transported to a given location, it is transferred from the atmosphere to vegetation and the ground surface by precipitation scavenging of the plume and by dry deposition processes. The rates of transfer from the atmosphere to vegetation surfaces are dependent on the chemical form of iodine in air; therefore, the total amount of $^{131}$I transferred was estimated by accounting for all three forms of iodine (elemental, particulate, and organic).

For routine releases, annual average concentrations of $^{131}$I on vegetation were estimated using a constant rate of deposition of $^{131}$I from the air for a given year and the assumption that the annual average concentrations of $^{131}$I in vegetation and in air were in equilibrium with each other. For the 1954 accident, deposition of $^{131}$I onto vegetation lasted for a period of 2.5 hours after the accident, but it remained on the vegetation for a longer period of time until the processes of removal from vegetation and natural radioactive decay eliminated it completely. A time-integrated concentration of $^{131}$I on the vegetation was, therefore, estimated to account for the longer-term availability of contaminated feed to the cattle.

**Transfer from Pasture to Various Food Products**

Once $^{131}$I is transferred to the surfaces of vegetation, it is available for ingestion by grazing animals. Once ingested, it is further transferred into milk, meat, cheese, and eggs. Estimates of the transfer of $^{131}$I from pasture to milk and beef were based on information from available literature and on unpublished measurements of the transfer of $^{131}$I into the milk of various breeds of dairy cattle used on farms in East Tennessee during the 1950s and 60s. No significant difference was observed among the various breeds of cows; however, an inverse relationship was observed between $^{131}$I transfer to milk and milk yield for those animals producing less than 10 L/d. For this reason, a distinction was made
between the transfer of $^{131}$I into milk for low-producing "backyard" cows and for higher-producing cows belonging to commercial dairies. Estimates of $^{131}$I concentrations were also made for goat's milk, human breast milk, cottage cheese, eggs, and beef.

Using average concentrations of $^{131}$I in raw milk collected and pooled from milk sampling stations around X-10 in 1962 and 1964, the modeling approach used in this study for estimating concentrations of $^{131}$I in milk was validated. The average values of $^{131}$I measurements in milk in both 1962 and 1964 were found to lie well within the 95% subjective confidence intervals of the average value predicted by the model for milk concentrations pooled from 8 locations in the vicinity of the sampling stations.

The concentrations of $^{131}$I in animal food products were estimated with mathematical models. For routine releases, the annual average concentration on pasture was related directly to the annual average concentration in animal food products. For the 1954 accident, time-integrated concentrations of $^{131}$I in milk and meat were estimated based on the time-dependent concentration of $^{131}$I in pasture grass.

**Distribution of Contaminated Food Products**

The distribution of food products from various producers to potential consumers is a complex process that is difficult to reproduce with high accuracy. Contaminated foodstuffs produced in an affected area may be distributed to other areas not directly exposed by the radioactive plume. Conversely, individuals in the affected area may consume uncontaminated products imported from unaffected areas. In this analysis, food products produced at a given farm were generally assumed to be consumed locally or distributed to the local population for consumption. Milk was often exchanged between counties to cover consumption needs in milk-deficient areas. The effects of the distribution system for a given food type were considered by accounting for radioactive decay between the time of milking or harvest and the time of human consumption of the food product, for the fraction of the food consumed that originated from uncontaminated areas, and for the amount of radioiodine lost during food preparation. Eggs and cottage cheese were assumed to be produced and consumed locally, and only the reduction of contamination due to radioactive decay during the time between production and consumption was considered.

**Inhalation and Food Consumption**

Human exposure to $^{131}$I is dependent on the concentration of $^{131}$I in air and in food at a given location and on the rates of inhalation and food consumption. This study included a detailed investigation of inhalation and ingestion rates by gender and age. Different rates of inhalation and ingestion were considered for estimating exposures from the routine releases of $^{131}$I and from the 1954 accident.
accident. Because of the importance of human exposure via the consumption of contaminated milk, this study focused heavily on the estimation of consumption rates of fresh milk for infants and children under the age of 10. These estimates included differences in the consumption of locally produced milk and milk obtained at school. The estimated rates for inhalation of contaminated air for infants, children, teenagers, and adults accounted for the amount of time spent indoors and the differences between the concentration of $^{131}$I in air between the indoor and outdoor environments.

Consumption of meat, leafy vegetables, eggs, and cheese was also considered in addition to the ingestion of milk. In all, doses and risks were estimated for 11 individual exposure pathways: ingestion of backyard cow’s milk, commercial milk, regionally mixed commercial milk, goat’s milk, meat, leafy vegetables, eggs, or cottage cheese; inhalation of contaminated air; prenatal exposure from $^{131}$I ingested by the mother; and ingestion of contaminated mother’s milk during infancy.

Individuals living near X-10 may have been exposed via more than one exposure pathway at a time. Three special exposure scenarios are designed to match the most likely dietary habits and lifestyles in the vicinity of the Oak Ridge Reservation. The first exposure scenario (called “diet 1”) refers to individuals living in a “rural farm” setting. The intake for this exposure scenario is obtained from ingestion of backyard cow’s milk, beef, leafy vegetables, eggs, and cheese and from inhalation. The second exposure scenario (called “diet 2”) refers to individuals in a rural area who buy milk from a local dairy farm. They are also exposed to contaminated beef, leafy vegetables, eggs, cottage cheese, and air. The third scenario (called “diet 3”) refers to individuals in a more “urban” setting, who buy milk and food products from the grocery store. The intake for this exposure scenario is obtained from ingestion of regionally averaged commercial milk and from inhalation. Given that the doses and risks from ingestion of goat’s milk are substantially larger than the doses and risks from any other exposure pathway, it is addressed separately under the “diet 4” scenario.

**Internal Dosimetry**

After it is inhaled or ingested, $^{131}$I is absorbed into the bloodstream and then metabolized in a manner that is identical to the absorption and metabolism of stable iodine in the human body. The thyroid gland preferentially absorbs iodine from the extracellular fluid into the thyroid cells and follicles. Iodine is then used in the production of hormones essential for human metabolism. In this analysis, the absorbed thyroid dose per unit intake of $^{131}$I was estimated as a function of age. Since the uncertainty in the dose per unit intake is largely affected by the interindividual variability in the thyroid mass, this investigation employed the most recent information on thyroid volume as determined by ultrasonography. It was found that the mass of the thyroid was somewhat smaller than assumed in past studies. However, this finding was offset by the finding of a more rapid biological
clearance rate from the thyroid, which resulted in a central estimate of the dose per unit intake that was similar to values recommended by the International Commission on Radiological Protection for newborn children up to children 15 years of age.

**Excess Lifetime Risk per Unit Absorbed Dose**

It is well established that X- and gamma irradiation of the thyroid at doses approaching 10 cGy will result in increased incidence of thyroid carcinomas and adenomas in children exposed before the age of 15. At higher doses, radiation might also induce non-neoplastic thyroid conditions such as autoimmune hypothyroidism, Hashimoto's thyroiditis, or Graves disease. The thyroid gland in children has one of the highest radiogenic risk coefficients of any organ. Fortunately, fatal thyroid cancers are rare (the 5-year survival rate is 95%). The effectiveness of $^{131}\text{I}$ in producing thyroid cancers is a subject that is still under investigation with ongoing epidemiological studies at numerous other locations. The most convincing evidence of the link between $^{131}\text{I}$ exposure and thyroid cancer is still emerging from reports and preliminary results from the follow-up of children exposed to $^{131}\text{I}$ from the 1986 Chernobyl accident. Additional supporting evidence exists from animal studies and from epidemiological investigations of Utah school children exposed to $^{131}\text{I}$ from atmospheric weapons testing at the Nevada Test Site.

In this study, a relative risk model was used to estimate the chance of acquiring a thyroid cancer from an absorbed dose of $^{131}\text{I}$. This model calculates the excess lifetime risk of thyroid cancer per unit dose as the product of the excess relative risk per unit absorbed dose and the background lifetime risk for an unexposed individual, including a series of modifying factors to account for differences in radiosensitivity by gender and age at time of exposure and for the effectiveness of $^{131}\text{I}$ in producing thyroid cancer compared with that of X-rays and gamma rays. The background lifetime risk of thyroid cancer was obtained from incidence data for the state of Tennessee, excluding the counties of Anderson, Roane, Loudon, and Knox, which were affected by releases of $^{131}\text{I}$ from X-10.

The excess lifetime risk of thyroid cancer per cGy changes markedly depending on the gender of the individual and the age at time of exposure. For females exposed to 1 cGy before the age of 5 years, the excess lifetime risk ranged from about five chances in one hundred thousand to sixteen chances in ten thousand, with a central value of three chances in ten thousand. At the same dose, the risk to males in this age group would be about four times less than that for females. Females who were over the age of 20 at time of exposure to 1 cGy would have had a risk almost 80 times less, while males over the age of 20 would have had a risk about 300 times less than females who were under the age of 5 years.
Results
Females born in 1952 who consumed goat’s milk (diet 4) received the highest
doses and have the highest risks of contacting thyroid cancer during their
lifetime. The next highest dose results from the consumption of milk from a
backyard cow, followed by milk from a local commercial dairy and milk that was
regionally mixed. Since the concentration of $^{131}$I in regionally mixed retail milk is
about the same regardless of location within the 38-km domain, its importance
with respect to the consumption of local produce or to inhalation varies from
location to location.

Lower doses are obtained from inhalation or from the consumption of locally
produced beef, cottage cheese, mother’s milk (with the mother assumed to be on
diet 1), or leafy vegetables. The doses from inhalation or from the consumption of
one of these food types for a child under the age of 5 at the time of exposure are
several hundred to more than 1000 times less important than the dose from the
consumption of backyard cow’s milk. The thyroid dose from prenatal exposure
during the first part of 1952 (assuming the mother to be on diet 1) is about equal
to the 5-year total thyroid dose obtained from the consumption of beef or cottage
cheese. Risks were estimated specifically for the four diet categories, each of
which consists of a combination of pathways.

Among the 41 selected locations within the 38-km vicinity of the X-10 facility, the
highest doses occurred at Gallaher Bend, located a little more than 6 km to the
east of X-10, while the lowest doses occurred at Wartburg, located 27 km
northwest of X-10. For example, at Gallaher Bend, the thyroid dose ranged from
about 4 to 250 cGy for individuals of each gender born between 1940 and 1952
who consumed milk from a backyard cow and food products from a local garden
or farm. A similar group of individuals residing in Wartburg would have received
doses ranging from about 0.08 to 6 cGy. Doses from the consumption of
regionally mixed commercial retail milk ranged from about 0.3 to 10cGy for
individuals born from 1940 to 1952 and did not vary by location.

Assuming the same dietary sources for ingestion of $^{131}$I at a specific location,
differences in gender account for only minor differences in the estimation of the
thyroid doses. More significant differences are determined by the year of birth,
with the lowest doses being for individuals born in 1920, 1930, and 1956. These
doses are about one-fourth to one-fifth of the largest doses received by
individuals born between the years of 1944 and 1952. Individuals born in 1954
have about the same doses as those born in 1940, which are about 65% of the
doses for those born between 1944 and 1952.

The highest excess risks of developing thyroid cancer for a female born in 1952
on diet 1 occur at the agricultural communities of Bradbury and Gallaher Bend.
For these locations, the risk estimates are confidently above one chance in one
thousand ($1 \times 10^{-3}$) but less than 1 chance in ten ($1 \times 10^{-1}$). In addition, at these
locations, the central estimate of the probability of causation approaches or
exceeds 50% for females born in 1952 on diets 1, 2, and 4, meaning that a diagnosed thyroid cancer has more than an even chance of being due to exposure to $^{131}$I released from X-10. The central estimate of risk for a female born in 1952 on diet 1 is likely to exceed 1 chance in 1,000 (1 x $10^{-3}$) up to distances of 35 km to the southwest and more than 38 km to the northeast of X-10. A risk of more than one chance in ten thousand (1 x $10^{-4}$) is likely with a subjective confidence of over 50% at all locations in the 38-km vicinity of X-10.

Depending on the year of birth, the excess lifetime risk to females is 3 to 4 times larger than the risk to males. The lowest risk is for a male born in 1920, who has an excess lifetime risk of thyroid cancer almost 1000 times less than the highest risk for females born in 1952. A female born in 1920 has a risk about 350 times lower than that for a female born in 1952. Individuals of the same gender born in 1944 have about 50-60% of the risk that those born in 1952 have, while individuals born in 1940 or 1956 have risks about 5 times lower than those for individuals born in 1952. However, a male born in 1940 or 1956 has a risk almost 20 times less than that of a female born in 1952.

The primary locations affected by the April 29, 1954, accident are those situated to the north and northwest of X-10, such as Jonesville, Norwood, East Fork Poplar Creek in Oak Ridge, Oliver Springs, and Wartburg. The excess lifetime risk of thyroid cancer to females on diet 1, exposed in their early childhood at either Jonesville, Norwood, East Fork Poplar Creek, or Oliver Springs ranged from a few chances in 10 million (3 or 4 x $10^{-7}$) to nearly one chance in one thousand (1 x $10^{-3}$). In general, the total doses and risks from exposure to the April 29, 1954, accident are much lower than those resulting from exposure to the April 29, 1954, accident and the routine emissions combined.

Most of the uncertainty in the estimates of risk is associated with the uncertainty in the estimates of dose (55%), followed by the uncertainty in the dose response for cohorts exposed to external sources of radiation. Uncertainty in the dose estimates are dominated by the uncertainty in the estimated concentrations of $^{131}$I in milk (45%), followed by the uncertainty in the internal dose conversion factor (40%). The uncertainty in the estimates of milk concentrations is dominated by the uncertainty in the transfer of $^{131}$I from air to pasture (71%). The uncertainty in the internal dose conversion factor is dominated by the uncertainty in determining the actual mass of the thyroid for any individual of a given gender and age.

Fallout from atmospheric testing of nuclear weapons at the Nevada Test Site (NTS) during 1952, 1953, 1955, and 1957 was a significant contributor to the total $^{131}$I exposure for individuals located within 38 km of the X-10 site. Beyond this distance, $^{131}$I from NTS fallout was clearly the dominant source of exposure. For a female born in 1952 who consumed backyard cow's milk, central estimates of the thyroid dose from combined exposures from X-10 releases and NTS fallout within the 15 km vicinity of X-10 ranged from 25 to 30 cGy, with upper bounds of
the 95% subjective confidence interval exceeding 200 cGy. At all locations within 15 km of X-10, the risk to a female born in 1952 on a rural diet (diet 1) exceeds one chance in one thousand \((1 \times 10^{-3})\). The upper bound of the 95% subjective confidence interval for the excess lifetime risk of thyroid cancer exceeds several chances in one hundred \((6-9 \times 10^{-2})\) up to more than one chance in ten \((1 \times 10^{-1})\) at the communities of Bradbury and Gallaher Bend. The doses from the combined exposure to \(^{131}\text{I}\) released from X-10 and \(^{131}\text{I}\) deposited via NTS fallout are sufficiently high to have possibly manifested excess cases of non-neoplastic disease, namely autoimmune thyroiditis.

**Number of Excess Cancers Expected in the 38-, 100-, and 200-km Vicinity of X-10**

Within the 38-km vicinity of X-10, the 95% subjective confidence interval of the number of excess thyroid cancers resulting from consumption of cow's milk (commercial and backyard milk, combined), contaminated by \(^{131}\text{I}\) released from the RaLa processing facility between 1944 and 1956, ranges between 6 and 84, with a central estimate of 21. For the consumption of backyard cow's milk alone, the 95% subjective confidence interval ranged from 1 to 33 excess cases of cancer, with a central estimate of 7. Commercial cow's milk contributed more to the number of excess cancers because of the larger quantity of commercial milk produced and the larger number of individuals exposed via its consumption.

The 95% subjective confidence interval of the number of excess thyroid cancers from consumption of cow's milk (commercial and backyard milk, combined) ranges from 14 to 103 (central estimate of 35) within a 100-km vicinity of X-10, and from 25 to 149 (central estimate of 58) within a 200-km vicinity of X-10. These cancers are expected to manifest between 1950 and 2020, with the majority occurring after 1970.

These estimates were made using a baseline of thyroid cancer diagnoses within the regional population with the assumption that the individuals in this population were unexposed. Only about 28% of the total number of thyroid cancers in a population are diagnosed and reported. Therefore, it is likely that the total number of diagnosed and occult cases of thyroid cancer is about 3 to 4 times greater than the total estimate given in this study, which are based on the incidence of thyroid cancer reported only through clinical diagnosis. The excess incidence of benign nodules will be about 8 to 10 times the excess incidence of thyroid cancer if the detection of nodules is made by palpation of the neck. An additional factor of 7 increase in benign nodules can be expected if diagnosis is determined with ultrasonography. The clinical significance of an excess incidence of benign nodules is not evaluated in this study, but it is noted that about 9% of benign nodules diagnosed through palpation and about 28% of those diagnosed through ultrasound will be surgically removed.

In the 38-km vicinity of X-10, an increase in the number of cases of thyroid cancer is forecast by both this study and by the incidence rates in the disease
registry maintained by the Tennessee Department of Health. The number of excess cases of thyroid cancer forecast by this study due to $^{131}$I releases from X-10 are about 4 to 50% of the excess cases estimated using the disease registry. Because of the presence of uncontrolled confounding factors affecting the results recorded in disease registries, extreme caution must be used when interpreting the overall significance of this finding.