

Native American Exposure to Iodine-131 from Nuclear Weapons Testing in Nevada

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ABSTRACT

A great deal of work has been done reconstructing doses from Nevada Test Site fallout, yet the unique exposures of Native American communities continue to be neglected. It is possible to estimate the exposures of these communities through a process of collaborative information gathering and analysis. This paper builds on a previous exercise that demonstrated the substantial doses received through the consumption of contaminated game. The updated model includes new information on the deposition of iodine-131, an assessment of the neonatal thyroid doses received through breast milk, an exploration of the effect of population mobility on dose estimates, and estimates of thyroid cancer risk. All thyroid dose estimates from the rabbit exposure pathway are comparable in magnitude to National Cancer Institute comprehensive dose estimates that assume exposure to contaminated milk from backyard cows and goats. Dose estimates from the rabbit exposure pathway are larger than estimated doses from store-bought milk by an average factor of 6. Taking historical population mobility patterns into account may result in slightly lower estimates of dose. The quantification of this exposure pathway is considered to be an advance toward a more appropriate dose reconstruction for communities with diets high in wild game.

Key words: Native American; radiation; nuclear weapons testing; thyroid cancer; dose reconstruction

INTRODUCTION

In 2001 the Centers for Disease Control and Prevention (CDC) and the National Cancer Institute (NCI) began collecting comments on a draft report describing the health effects of nuclear testing (NCI 2001), and in 2002 NCI began a thyroid cancer awareness campaign aimed at educating the public about potential thyroid cancer risks (NCI 2002). These are major new efforts toward informing people about the legacy of nuclear testing and addressing potential consequences. In this context it is important to identify significant exposures that have not been covered in the CDC/NCI analyses, and it is especially important to identify specific populations who may have had particularly high exposures.

Frohberg *et al.* (2000) showed that the consumption of wild game is one exposure pathway experienced by Native Americans that has previously been overlooked. We feel that this reflects a continuing lack of attention to Native Americans that has characterized the weapons testing program from its beginning. The location of the Nevada Test Site (which is on Western Shoshone land) was chosen without considering Native American interests. Native communities may have experienced unique additional risk yet no specific efforts at warning or informing community members were made. The new NCI outreach campaign includes a flip chart for Native Americans that would benefit from the inclusion of important community-specific exposure pathways that are currently not mentioned (NCI 2002).

The purpose of this paper is to update the Frohberg *et al.* (2000) illustration with new information and to update our perspective on the feasibility of dose reconstructions for small populations with unique lifestyles that are believed to influence exposure. We do not provide a full dose reconstruction but rather a case study of one community, Duckwater, Nevada, and one exposure pathway, the exposure to iodine-131 (¹³¹I) through the consumption of wild rabbits. The Native Community Action Council (NCAC), a community group of Western Shoshone and Southern Paiutes, together with a group at Clark University, has accumulated lifestyle information about several Western Shoshone and Southern Paiute communities; we plan to publish more comprehensive results for these communities at a later time.

After a brief historical description we discuss methods in two sections. The first methods section describes the changes made to the Frohberg *et al.* (2000) model and outlines a new set of model parameters. The second methods section describes a GIS analysis of the effect of human mobility on the dose estimate. The results and discussion sections compare the model outputs to NCI dose estimates and consider the thyroid cancer risk experienced by Duckwater residents.

History

The Western Shoshone homelands include much of Nevada and parts of Idaho and California (Figure 1), and the 1863 Ruby Valley Treaty granted the Shoshone sovereignty over this territory. Traditionally the Shoshone lived in the valleys in the fall and winter and then moved to the mountains in the spring and summer, and although they had settled on reservations by 1950 they were still very mobile people. The reservation at Duckwater was established in 1940 with 21 families (Duckwater Shoshone Tribe 2003).

Ten years later the Nevada Test Site was established 100 miles south of Duckwater as the location for continental US nuclear weapons testing.

Over 900 nuclear tests were conducted at the Test Site between 1951 and 1992, releasing roughly 5.55 EBq of ^{131}I (NCI 1997a). This is roughly three times the amount of ^{131}I released by the Chernobyl accident (1.76 Ebq; UNSCEAR 2000), but a significantly smaller amount than the total released by the U.S. in its Pacific testing or the total released by testing in the Soviet Union. The vast majority of these releases occurred during the atmospheric testing era of the 1950s. The most exposed regions were southwestern Utah, east of the test site, and the portion of Nye County just north of the Test Site, including Duckwater (NCI 2001). Over the period of testing no health protective measures were attempted and only minimal efforts were made to inform people about the testing and the associated risks. The DOE began its dose reconstruction work in 1979 with the Off-Site Radiation Exposure Review Project (ORERP; Church *et al.* 1990). Another dose reconstruction from the NCI was published in 1997 (NCI 1997a). Neither project estimated exposures unique to Native Americans.

In 1993 the Western Shoshone National Council, representing several tribal communities in the Great Basin region of California and Nevada, requested the support of the Childhood Cancer Research Institute, a small non-profit organization working to build community capacity to deal with the health effects of radiation. This partnership became the Nuclear Risk Management for Native Communities Project and came to include a community advisory board and community staff representing several Western Shoshone and Southern Paiute communities (now incorporated as the NCAC), and a revolving team of Clark University faculty, staff, and students. The team collaboratively investigated the wild game exposure pathway and published an analysis that suggested the possibility of increased food chain-related thyroid dose and consequent risk due to consuming rabbits (Frohberg *et al.* 2000). Through analysis of community-generated lifestyle information it had become clear that wild game consumption alone might have resulted in thyroid doses that were greater than the official dose estimates covering all assumed pathways. The data also indicated that other pathways contributed substantial doses to portions of the population, especially milk obtained from backyard livestock.

METHODS

The Original Model

The original dosimetric model is depicted in Figure 2a. The model was intended to follow ^{131}I contamination in plants and rabbit thyroids through a rabbit consumption scenario and arrive at an estimate of dose to the thyroid of a human¹. The units of the model dealing with the interception of fallout by vegetation and the transfer to jackrabbits were defined by the work of F. Turner and W. Martin at four locations after the Sedan test in 1962 (AEC 1965a, AEC 1965b, Martin 1965, Turner 1963). In the model both plants and rabbits are considered to lose ^{131}I through radiological decay and through weathering (plants) or excretion (rabbits). Rabbit consumption rates were defined by the community through a series of informal interviews and focus group meetings. Direct

¹ It is important to note here that the thyroids of rabbits and other small mammals were routinely consumed; methods of cleaning the animals minimized waste by removing only intestines (Frohberg *et al.* 2000).

estimates of ^{131}I deposition for multiple test events and locations were not available and the model was used to evaluate doses based on a 1962 test event.

Modifications of the Original Model

The new model (Figure 2b) includes information on test-specific deposition of ^{131}I . At this point there are several sources of data from which one can derive estimates of deposition:

- **Town level ^{131}I deposition data.** The most direct source for estimating exposure at Duckwater is a set of annexes to the 1997 NCI report, available online (NCI 1997b) that list ^{131}I deposition for points identified by number but not by name. We observed that point 84 was located at Duckwater (see Figure 1) and used these data for our analysis; where these data were unavailable we used data for point 83 (Currant) 15 miles to the southeast.
- **County level ^{131}I deposition data.** The NCI also provides county- and sub-county level ^{131}I deposition estimates. These were used to generate thyroid dose estimates that could be compared with the NCI online dose calculator (NCI 2002), a tool that provides dose estimates at the subcounty level¹. The area that we used for our comparisons was Nye county, subcounty 2 (Nye 2), shown in Figure 1.
- **External exposure rate data.** Two other sources of information are DOE fallout contour maps and the ORERP Town Database. Both of these sources provide data in units of external exposure at 12 hours after each test event. These can be converted to iodine deposition using coefficients provided by Hicks (1981,1982)². The Town Database was used to validate the NCI annexes. The DOE contours were used in our GIS analysis, described below.

Another modification of the model dealt with the quantification of rabbit consumption. The original model assumed that the average adult ate 1.9 rabbits per week, year round. In fact, this part of the Shoshone diet varied seasonally. We used two separate community-defined rates of consumption- one for the fall, the main rabbit eating season (3.2 rabbits per week), and one for the rest of the year (1.5 rabbits per week). These estimates were based on only nine interviews that quantified this value and this parameter was therefore associated with more uncertainty than the others.

Where the original model included assumptions about the rabbit consumption rate of infants, the new model treats adults and newborns differently, substituting a breast milk pathway for newborns. Transfer to newborns was characterized using the value of Simon *et al.* (2002) to define the transfer of ^{131}I from mother to newborn and a neonatal dose

¹ Institute of Medicine recommendations (NAS 1999) against estimating doses at the county or subcounty level do not apply in our study area. Nye County is within the region of “close-in measurements of environmental radiation” where ample data from portable survey instruments are available (NCI 1997a).

² Based on measurements of individual radionuclides in fallout clouds Hicks tabulated the conversion of external gamma radiation exposure rate (mR/hr) to radionuclide ground deposition (nCi/m²) (Hicks 1981 and 1982, NCI 1997a).

coefficient (Harvey *et al.* 2003) to convert neonatal ingestion to a thyroid dose. The central estimate of the neonatal rate of milk consumption was the same as that used in the NCI dose reconstruction (Simon *et al.* 2002) but we could not locate information about the variability of this parameter. We therefore applied the variability in caloric intake of a 1-year old, the youngest available age category, as observed in the Third National Health and Nutrition Examination Survey (NHANES 3; NCHS 1997). We also estimated the thyroid dose received by a hypothetical 5-year old child. According to NHANES 3 a five year-old child consumes about 75% as many calories as an adult (NCHS 1997). We scaled the rabbit consumption rates accordingly in our model.

Coefficients relating ingestion to thyroid dose were taken from Dunning and Schwarz (1981) for adults and Harvey *et al.* (2003) for children. These estimates are similar to those reported by the International Commission on Radiological Protection¹ but are preferred because they include quantified uncertainty and variability in metabolic and physiological parameters. Original units of equivalent dose (Sv) were replaced with units of absorbed dose (Gy)².

Table 1 lists model parameters. The model was created as a Microsoft Excel spreadsheet and Monte Carlo simulations were run using Crystal Ball (Decisioneering, Inc. 1996). Each parameter was treated as having a lognormal uncertainty distribution defined by a geometric mean and a geometric standard deviation (GSD). Each model calculation was repeated 1,000 times to generate an output distribution and sensitivity analysis in terms of contribution to output variance.

Mobility Analysis

An important attribute of the new model is a GIS-based analysis of mobility patterns. This is done by running the rabbit consumption model with different iodine deposition inputs and modifying the dose estimates to reflect the fact that Duckwater residents were not necessarily in Duckwater at the time of a given test. Duckwater residents in the 1950s were very mobile, hunting and eating over a wide area, and we characterized this mobility using 32 interviews of Duckwater elders that were conducted by the community. We assumed that rabbit mobility was negligible and that rabbits ate contaminated vegetation from the same area in which they were caught. The GIS analysis was done using Idrisi software (Clark Labs 2001). Idrisi uses a raster environment, meaning that it represents space as a grid of pixels (a vector-based GIS would use points, lines and polygons). Each test event therefore had to be interpolated over a continuous surface where each pixel has a value. We used two tests to evaluate our options for interpolation. After interpolating the NCI database points, the ORERP town database points, and the DOE contours it became clear that only the DOE contours would generate meaningful interpolations because the point data were too sparse. For each test we

¹ Dunning and Schwarz (1981) provided median dose coefficient estimates of 5.3×10^{-6} Sv/Bq (newborns) and 3.0×10^{-7} Sv/Bq (adults). Harvey *et al.* (2003) give median estimates of 5.31×10^{-6} (newborns) and 2.15×10^{-6} (5-year olds). These compare with values of 3.7×10^{-6} (newborns), 2.1×10^{-6} (5-year olds) and 4.3×10^{-7} Sv/Bq (adults; ICRP 2000) and the EPA adult value of 4.8×10^{-7} Sv/Bq (USEPA 1993).

² Both sources of the thyroid dose coefficient (Dunning and Schwarz 1981, Harvey *et al.* 2003) assumed a relative biological effectiveness of ¹³¹I beta radiation of 1 and reported the dose coefficients in units of Sv; this was a change in unit but not in value. We use units of absorbed dose for compatibility with published risk estimates.

interpolated the DOE contours to create a surface of external exposure rates (an example is shown in Figure 3). A second map layer was generated to represent the mobility of Duckwater residents (Figure 3). Each pixel in this layer was coded with a probability that an individual would occupy that pixel. We assumed that probability was higher closer to Duckwater and based on the interview data we assigned zero probability to all pixels more than 50 miles from Duckwater. We also assumed higher probabilities moving from north to south, again based on interview data. The pixel probabilities were calibrated to sum to one allowing us to treat each pixel as a weight in a weighted-average function of the exposure layer.

To summarize, the weighted average thyroid dose accounting for mobility was calculated as:

$$D(\text{test}) = \sum_{\text{pixel}=1}^n [\text{EER}(\text{test}, \text{pixel}) * \text{ID}(\text{test}) * \text{T}(\text{test}) * \text{P}(\text{pixel})]$$

Where $D(\text{test})$ is the thyroid dose for a given test, $\text{EER}(\text{test}, \text{pixel})$ is the external exposure rate for a given test and pixel (mR hr^{-1} at H+12), $\text{ID}(\text{test})$ is the test-specific ^{131}I deposition coefficient from Hicks ($\text{nCi m}^{-2} \text{mR}^{-1} \text{hr}$ at H+12), $\text{T}(\text{test})$ is the test-specific thyroid dose coefficient from the rabbit pathway model ($\text{cGy nCi}^{-1} \text{m}^2$), and $\text{P}(\text{pixel})$ is the probability weight of each pixel (these probability weights will sum to 1). The resulting thyroid dose estimates are not directly comparable with the results of the first analysis because different sources of deposition data were used (interpolated contour data instead of point deposition data). Instead, a dose estimate was made for Duckwater using the DOE contours as source data and assuming that movement was restricted to the reservation (the implicit assumption of the first analysis). The ratio of these two estimates, the mobile dose and the stationary dose, is the true measure of the effect of our mobility assumption. This analysis lacks a measure of uncertainty or variability and therefore we discuss mobility-adjusted results separate from the primary model results.

RESULTS

Thyroid Doses of ^{131}I

We found 25 test events that were reported to have deposited fallout in Duckwater or within 50 miles of Duckwater. We chose to analyze only the five most significant test events, which together accounted for over half of the total adult thyroid dose. These tests are: Easy (May 7, 1952), George (June 1, 1952), Apple 2 (May 5, 1955), Shasta (August 18, 1957) and Galileo (September 2, 1957). Adult, child and newborn committed thyroid doses from each test are shown in Figure 4.

Neonatal doses were consistently greater than adult doses by a factor of 5, and this reflects age-specific thyroid mass and metabolism of iodine; neonates ingested less iodine (about 1/3 of maternal ingestion) but experienced a much greater dose per unit intake (see dose coefficients in Table 1). Thyroid dose estimates for 4-year olds are approximately equal to neonatal dose estimates; young children experience a lower dose per unit intake than a newborn but also ingest more iodine.

We also calculated the committed thyroid doses from the rabbit pathway using the estimates of deposition for the subcounty Nye 2 (which includes Duckwater). This was done for the purposes of comparison with the doses reported on the NCI website (NCI 2002). Table 2 shows a comparison of our estimates of thyroid doses from the rabbit pathway with the doses estimated by the NCI for all pathways that they considered. We report NCI estimates for the same five tests that we discuss above assuming three possible exposure scenarios. It can be seen that the dose estimates for the rabbit exposure pathway are greater than those for average commercial milk consumption (or a breast milk diet in the case of newborns) by factors of 4-9. Rabbit pathway estimates are generally larger than backyard cow milk exposure estimates and smaller than goat milk exposure estimates.

Results of the Mobility Analysis

As discussed above, our mobility analysis used the same rabbit pathway model but with a different deposition estimate derived from the Department of Energy's contours of external exposure. Therefore this set of results is not directly comparable to the results described above. We calculated a dose estimate for the entire region of mobility as a weighted average using probabilities of location as weights. Using the same deposition values we also calculated a dose estimate for Duckwater. The ratio of these values is the desired result as it describes the relative change in dose estimate that we expect when adding mobility assumptions to our model. For the Apple 2 test the interpolated contours show stationary adult Duckwater residents receiving a thyroid dose of 0.6 cGy. Duckwater adults behaving according to our mobility assumption would have received a thyroid dose of 0.4 cGy. The mobile:stationary dose estimate ratio is in this case 0.66. The cumulative exposure contours show a Duckwater dose of 2.3 cGy and our mobility assumption gives us a dose of 2.0 cGy, for a ratio of 0.87. Thus, our mobility assumptions result in a dose estimate that is lower by 33% (Apple 2) or 13% (cumulative dose).

Thyroid Cancer Risk Estimates

Lifetime thyroid cancer risks can be estimated using different published risk coefficients. Ron *et al.* (1995) pooled the data of 5 previous external radiation studies for an estimated excess relative risk per gray (ERR/Gy) of 7.7 (95% CI 2.1, 28.7) for children exposed before age 15. These authors also analyzed two sets of data for adult exposures- atomic bomb survivors and cervical cancer patients treated with x-ray therapy and/or radium implants. The cervical patients risk estimate was very high (ERR/Gy 34.9) but also very uncertain (95% CI -2.2, ∞). The atomic bomb survivors above age 15 at the time of the bombing showed an ERR/Gy of 0.4 (95% CI -0.1, 1.2). For the NCI dose calculator a model of exponential decline in risk with age was applied to the same data analyzed by Ron *et al.* (Apostoaeci *et al.* 2003, NCI 2003). We used these modeled risk estimates in our analysis (Table 3). The NCI also adjusts the risk estimate with a dose and dose rate effectiveness factor (DDREF). Linear risk models based on atomic bomb survivor data are often adjusted with a DDREF when applied to fractionated or low-dose exposures. A standard default value of 2 was proposed by the International Commission

on Radiological Protection (ICRP 1991) but there is still considerable uncertainty around this factor (UNSCEAR 2000). Based on the current range of evidence the NCI gives the DDREF a discrete probability distribution that includes values above and below one. The possible values are 0.5, 0.7, 1, 1.5, 2, 3, and 4 and occur with probabilities of 1%, 4%, 35%, 23%, 23%, 10%, and 4% (Apostoaie *et al.* 2003). We also applied a DDREF with this distribution. The dose-response is thus modeled as:

$$\text{ERR}(\text{age}) = (\beta(\text{age})/\text{DDREF}) * \text{dose}(\text{age})$$

Where $\beta(\text{age})$ is the age-specific excess relative risk per unit dose. We derived probability distributions of risk using Crystal Ball and the above distributions of ERR and DDREF.

Adult thyroid cancer risks were relatively small with median relative risks for individual tests all less than 1.1 (less than 10% increase in thyroid cancer risk). Risks for people exposed as neonates or young children are more substantial (Table 4); based on the central risk estimates we can assume that each test increased the thyroid cancer risk of an exposed child by 10-30% and we cannot rule out increases in risk of up to 8-fold.

DISCUSSION

This exercise demonstrates that doses from fallout can be estimated specifically for Native American communities. The major qualitative conclusion that we can draw from this exercise is that the NCI report, which was written with a focus on the general population, may not be appropriate for communities with unique diets. The NCI report assumes that the major exposure route was the ingestion of milk; additional exposure routes considered were inhalation of contaminated air and the ingestion of contaminated greens, cheese, and eggs. The NCI model also included the transfer of ¹³¹I from mother to child via breast milk; this exposure route reflects the assumed exposure of the mother. Our estimates indicate that for people eating a diet heavy in small wild game the major exposure route may be the wild game. Our analysis also suggests that Duckwater residents, who were exposed to contaminated milk in addition to contaminated game, experienced a greater thyroid cancer risk than people whose primary exposure pathway was cows milk.

There is substantial uncertainty around the dose estimates that we present. The central dose estimates were most prominently affected by test-specific fallout deposition around Duckwater, which ranged over an order of magnitude. Variance in model output, on the other hand, was most sensitive to the parameters describing dose coefficients and rates at which people consumed rabbits, with these two parameters contributing roughly 50% of the variance. The rabbit consumption patterns were defined using the same interview data as in the original dose model (Frohberg *et al.* 2000). More interviews have been conducted in this community since that time and although the information regarding rabbit consumption was quantified in a different way the new results are similar. This parameter estimate is potentially biased, but information was collected as part of a much larger project documenting community experience and history and subjects were not typically aware of the particular use of information represented in this

paper. Interview methods have been informal and this clearly affects the precision of the rabbit consumption estimate.

Uncertainty in vegetation density on the ground could not be quantified based on available information. There was a suggestion made by Martin (1965) that weathering loss may be slower at low levels of deposition; this possibility is not addressed in our model although this parameter makes a relatively small contribution to output variance.

It is important to note that we are describing a model of one pathway and not a comprehensive dose reconstruction. For example, rabbits were one component of a broader diet that included other small mammals, also almost entirely consumed, preferentially hunted in the spring and early summer (NCAC 2002). This would be another important exposure pathway, especially considering that three of the five most significant test events for Duckwater occurred in the spring. We also did not estimate the milk consumption patterns for Duckwater; based on anecdotal evidence we assume that Duckwater residents drank milk from backyard cows and it can be seen in Table 2 that this milk exposure would have increased the dose from each test event by up to two-fold.

Although this paper is limited to a discussion of one reservation we are in the process of developing similar dose and risk estimates for other reservations. Our initial results for the Southern Paiute reservation of Shivwits in Washington County, Utah, for example, mirror the results for Duckwater. According to community interviews Southern Paiutes also had a diet heavy in small game in the 1950s. Fewer test events affected this community, but doses from individual tests were very high. The test event Harry (May 19, 1953), for example, resulted in a neonatal thyroid dose of roughly 14 cGy through the store-bought milk pathway (NCI 2002).

The results of our mobility analysis might seem counterintuitive; Duckwater residents tended to travel more often to areas south of the reservation, which brought them closer to the tests site. However these results suggest that the areas they frequented were, on average, less contaminated. This may be explained by the more rapid fall-off in exposure in transverse directions compared to changes longitudinally. Of course it must be noted that our mobility assumption is highly uncertain and that this uncertainty is unquantified. Additional refinements to this assumption could include further mobility restrictions based on elevations and landcovers that were more or less likely to be visited. Another possibility is defined variability and uncertainty functions of the mobility assumption that could be generated through further focus group meetings with the Shoshone.

Epidemiological studies of exposure to ^{131}I in fallout are relevant to this paper but it is very difficult to draw direct comparisons. Results from children exposed to Chernobyl fallout seem to suggest that the risk from ^{131}I may be as high or higher per unit dose. Jacob *et al.* (1999) reconstructed ^{131}I thyroid doses for children in 2,729 settlements and 3 cities in Belarus and Russia and followed thyroid cancer incidence during 1991-1995. They report an ERR/Gy of 23 (95% CI 8.6, 82). There is a possibility that this cohort was deficient in natural iodine; this would have led to above-average sequestration of ^{131}I and doses may therefore have been underestimated, inflating the risk estimate. This possibility was addressed in another study of Bryansk children (ages 6-18) by Shakhtarin *et al.* (2003); they show that dietary iodine did appear to play a role in radiation-induced cancer risk so that although the overall ERR was 19.4/Gy (95% CI 16, 22.7), the ERR in areas with sufficient dietary iodine was 13/Gy (95% CI -11, 71.2). This information does

not justify an assumption that ^{131}I beta radiation is more biologically effective than external gamma radiation but does the possibility. Studies of fallout ^{131}I exposure in the US have had mixed results. Kerber *et al.* (1993) conducted a cohort study of people who had been exposed as children to NTS fallout. This study found a significant excess of thyroid neoplasms and a dose-response coefficient that was very close to that reported by Ron *et al.* (1995) for external radiation exposures. The Hanford Thyroid Disease Study (Davis *et al.* 2002), on the other hand, found no statistically significant association between ^{131}I dose and thyroid disease in people who were exposed as children to emissions from the Hanford facility in Washington. HTDS and the study of Kerber *et al.* (1993) were comparable in cohort size, in numbers of thyroid cancers, and in mean estimated dose (17 cGy in both cases). In light of the above considerations we feel that the external exposure-based risk estimates generated for the NCI dose and risk calculator (Table 3) are the best estimates currently available.

It is very important to note that the dose and risk calculations for newborns and young children in our study only address exposures that they would have received from a few major test events. These individuals would have received additional exposures over the course of their childhood that would substantially increase their doses and therefore their thyroid cancer risks. For example, according to the NCI dose calculator an individual born in subcounty Nye 2 on May 1, 1952 would have received roughly one-tenth of his or her cumulative dose from the George and Easy tests. Both the thyroid dose coefficient and the thyroid cancer risk decrease with increasing age, so we should not assume that this person would have ten times the thyroid cancer risk, but clearly the final, lifetime exposure-based risk will be greater than we calculate for exposures to fallout from individual test events.

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Table 1: Model parameters

Parameter	geometric mean	GSD	Source
Interception by vegetation (%)	18.0	1.8 ¹	Martin (1965) ²
Density of vegetation (g/m ²)	215.3		Martin (1965)
Weathering half-life (d)	14.7	2.0	Martin (1965)
Rabbit consumption of vegetation (g/d)	99.5	1.1	Frohberg (1997)
Rabbit thyroid absorption (%)	23.0	1.5	Frohberg (1997)
Half-life in rabbit (d)	3.01	1.3	Frohberg (1997)
Adult consumption of rabbits in the fall (rabbits/person/d)	0.46	2.1	Interview data
Adult consumption of rabbits in other seasons (rabbits/person/d)	0.22	2.0	Interview data
Ratio of 5-yr old consumption:adult consumption	0.748		NCHS (1997)
Adult dose coefficient (cGy/nCi)	1.12 x 10 ⁻³	1.9	Dunning and Schwarz (1981)
5-year old dose coefficient (cGy/nCi)	7.96 x 10 ⁻³	1.7	Harvey <i>et al.</i> (2003)
Neonate dose coefficient (cGy/nCi)	1.97 x 10 ⁻²	2.6	Harvey <i>et al.</i> (2003)
Neonatal consumption of maternal milk (L/d)	0.77	1.5	Simon <i>et al.</i> (2002), NCHS (1997)
Milk transfer coefficient (d/L)	0.37	1.5	Simon <i>et al.</i> (2002)

¹ Distributions of parameters in unit of percent (interception by vegetation and rabbit thyroid absorption) were truncated at 100% so that values greater than 100% were not generated.

² Martin (1965) calculated interception to be about 8%. We recalculated the deposition (nCi m⁻² mR⁻¹ h) based on Hicks (1981), and together with exposure rates (mR h⁻¹) and ¹³¹I collected from plants (both reported by Martin) we estimated the interception parameter described in this table.

Table 2: Comparison of thyroid dose estimates from the NCI and from the rabbit pathway, both for subcounty Nye2 (median dose in cGy and 95% CI)

Test	NCI dose estimates for various exposure scenarios ¹			Estimated dose from rabbit pathway
Adult	Goat milk 1-3 glasses per day	Milk from a backyard cow	Store-bought cow milk 1-3 glasses per day	
Easy	0.49	0.17	0.05	0.24 (0.02, 2.7)
George	0.39	0.12	0.04	0.17 (0.01, 1.9)
Apple 2	0.09 ²	0.25	0.06	0.45 (0.04, 4.9)
Shasta	0.76	0.22	0.07	0.49 (0.04, 5.3)
Galileo	0.26	0.09	0.03	0.26 (0.02, 3.2)
4-year old	Goat milk 1-3 glasses per day	Milk from a backyard cow	Store-bought cow milk 1-3 glasses per day	
Easy	5.0	0.99	0.32	1.27 (0.15, 11.4)
George	3.3	0.69	0.25	0.93 (0.08, 8.6)
Apple 2	0.47	1.5	0.33	2.16 (0.20, 20.8)
Shasta	5.3	0.67	0.34	2.90 (0.30, 22.20)
Galileo	2.2	0.27	0.13	1.33 (0.13, 14.2)
Newborn	Goat milk 1-3 glasses per day	Milk from a backyard cow	Breast milk ³	
Easy	15	1.7	0.30	1.31 (0.08, 18.5)
George	9.8	1.2	0.22	0.80 (0.04, 18.3)
Apple 2	1.3	2.6	0.35	1.88 (0.08, 44.8)
Shasta	19	2.2	0.44	2.25 (0.14, 38.8)
Galileo	7.8	0.9	0.18	1.58 (0.10, 28.24)

Table 3. Risk coefficients used in this analysis (from Apostoaei *et al.* 2003)

Exposure Age	Geometric Mean Excess Relative Risk Gy ⁻¹	GSD
0	9.46	2.18
5	6.26	1.92
35	0.52	2.38

Table 4. Risk estimates for exposures through the rabbit pathway in Duckwater (relative risk and 95% CI)

Test	Adult exposure	4-yr old exposure	Newborn exposure
Easy	1.0 (1.0, 1.0)	1.1 (1.0, 2.0)	1.1 (1.0, 3.2)
George	1.0 (1.0, 1.1)	1.2 (1.0, 3.5)	1.2 (1.0, 7.8)
Apple 2	1.0 (1.0, 1.1)	1.2 (1.0, 4.0)	1.3 (1.0, 10.1)
Shasta	1.0 (1.0, 1.0)	1.1 (1.0, 3.0)	1.2 (1.0, 5.3)
Galileo	1.0 (1.0, 1.0)	1.0 (1.0, 1.9)	1.1 (1.0, 3.2)

¹ Confidence intervals are provided by the online dose calculator; the NCI dose estimates have an average GSD of 5.

² NCI dose estimates for the goat milk exposure pathway were unusually low for the Apple 2 test, apparently as the result of a calculation error.

³ Neonatal dose estimates for a normal amount of cow milk (not from a backyard cow) were roughly twice as large as those for breast milk.

Figure 1: Map of Newe Sogobia, the Western Shoshone homeland, showing Nevada, Nye County subcounty 2, Duckwater, and the Nevada Test Site

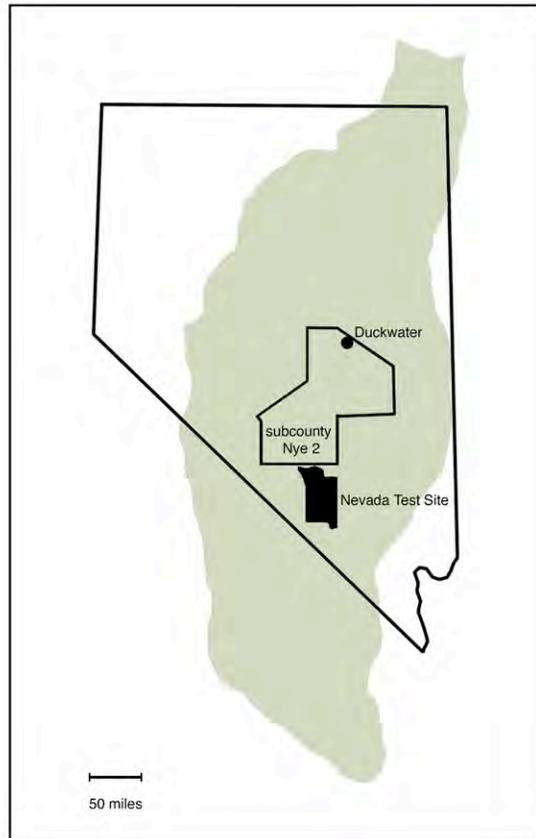


Figure 2: Original (top, Figure 2a) and revised (Figure 2b) model of the rabbit exposure route.

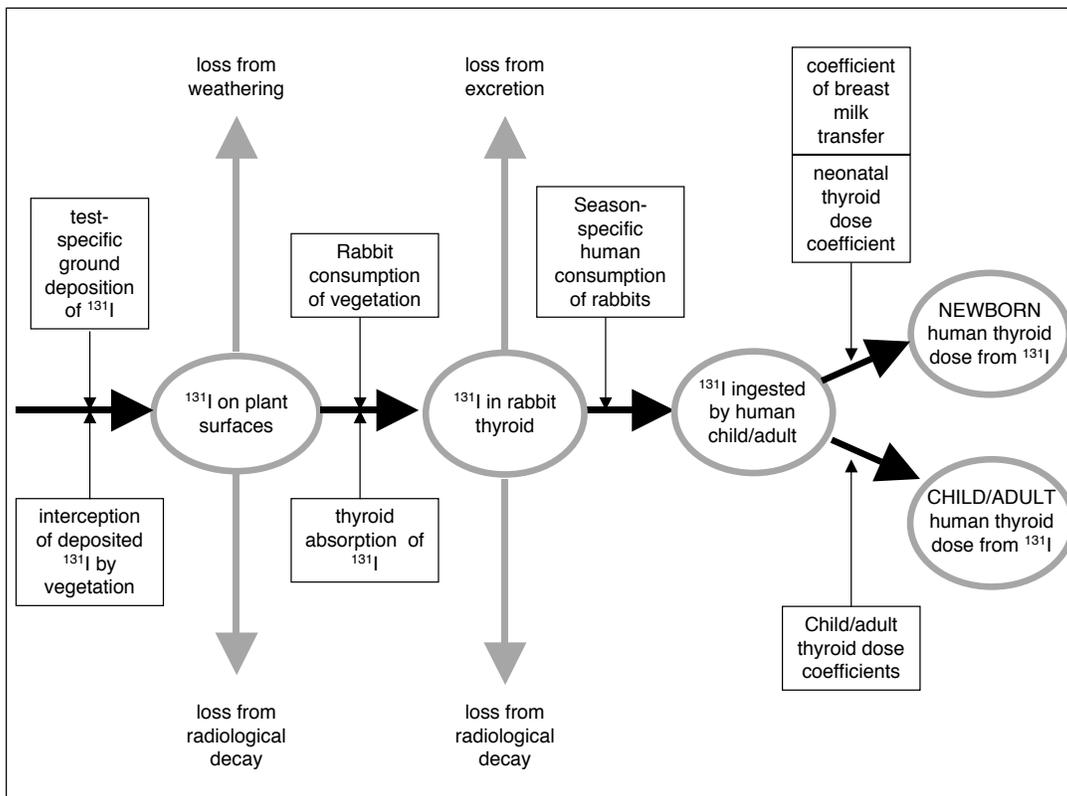
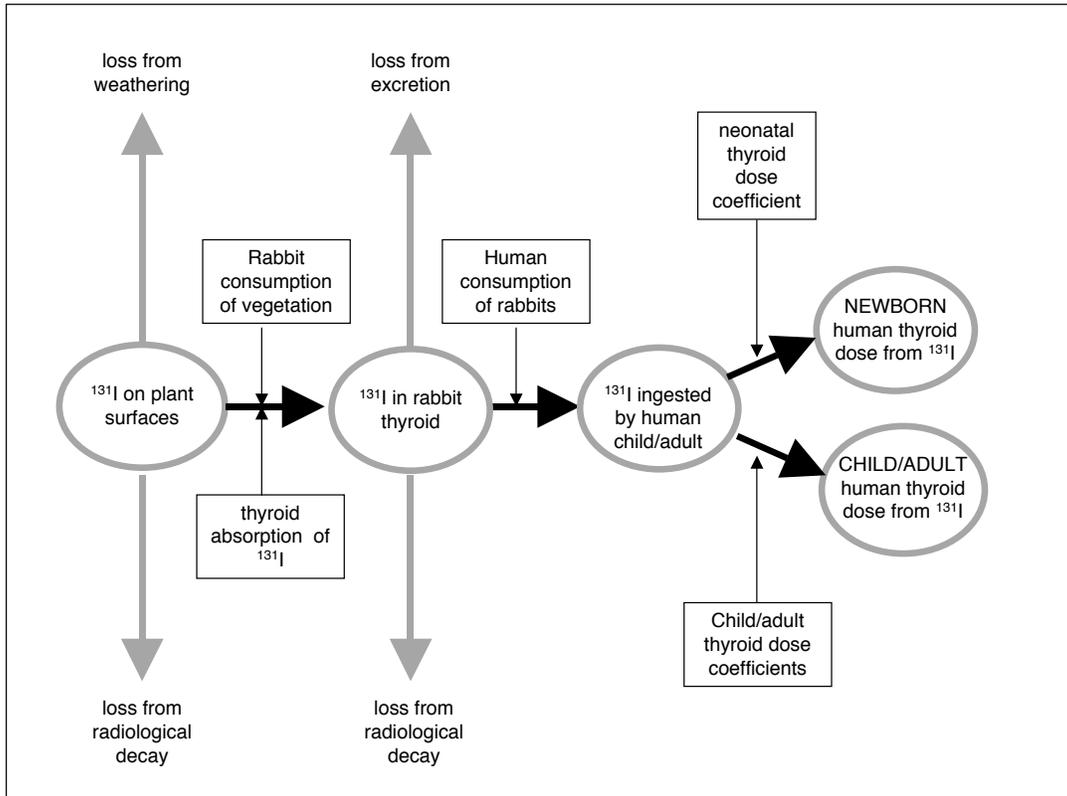


Figure 3: Fallout pattern for the test Apple II, May 5, 1955. Contour lines represent external exposure rates in $mR\ hr^{-1}$ at three feet above the ground and twelve hours after detonation. Shading within fallout contours is a product of our GIS interpolation. Shading around Duckwater represents assumptions about the mobility of Duckwater residents. Darker areas were more likely to be visited and mobility was assumed to be limited to a 50-mile radius.

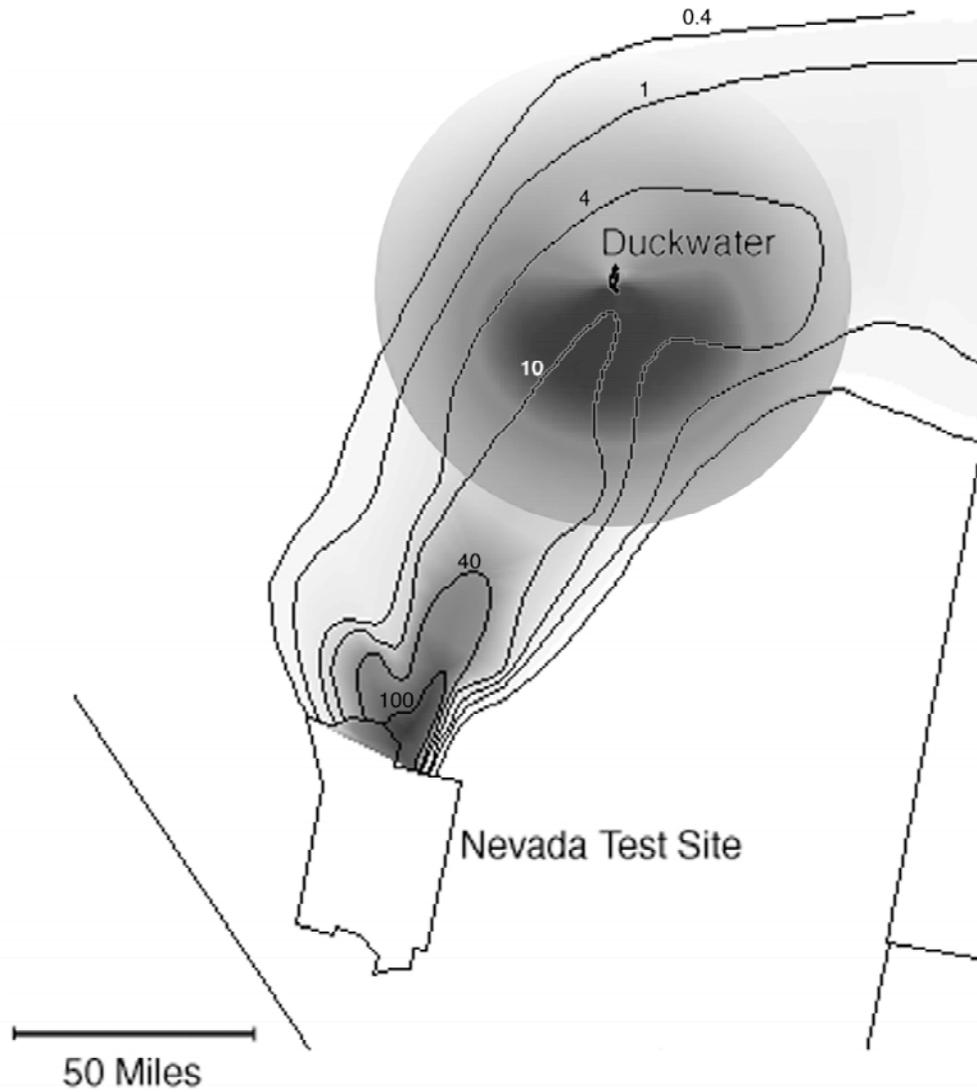


Figure 4: Thyroid doses (cGy) received by residents of Duckwater through the consumption of contaminated rabbits, and in the case of neonatal exposure via contaminated breast milk (bars represent the 95% upper confidence limit).

