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Thyroid Cancer Rates and ^{131}I Doses from Nevada Atmospheric Nuclear Bomb Tests: An Update

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Abstract

Exposure to radioactive iodine (^{131}I) from atmospheric nuclear tests conducted in Nevada in the 1950s may have increased thyroid cancer risks. To investigate the long-term effects of this exposure, we analyzed data on thyroid cancer incidence (18,545 cases) from eight Surveillance, Epidemiology, and End Results (SEER) tumor registries for the period 1973-2004. Excess relative risks (ERR) per Gray (Gy) for exposure received before age 15 were estimated by relating age-, birth year-, sex-, and county-specific thyroid cancer rates to estimates of cumulative dose to the thyroid that take age into account. The estimated ERR per Gy for dose received before one year of age was 1.8 (95% confidence interval (CI), 0.5-3.2). There was no evidence that this estimate declined with follow-up time or that risk increased with dose received at ages 1-15. These results confirm earlier findings based on less extensive data for the period 1973-1994. The lack of a dose-response for those exposed at ages 1-15 is inconsistent with studies of children exposed to external radiation or ^{131}I from the Chernobyl accident, and results need to be interpreted in light of limitations and biases inherent in ecologic studies, including the error in doses and case ascertainment resulting from migration. Nevertheless, the study adds support for an increased risk of thyroid cancer due to fallout, although the data are inadequate to quantify it.

INTRODUCTION

In 1997, a report of the National Cancer Institute (1) provided estimates of thyroid doses from exposure to radioactive iodine (^{131}I) resulting from atmospheric nuclear tests conducted in Nevada, primarily in 1952, 1953, 1955 and 1957 for representative categories of individuals residing in each county of the continental United States (US). Based on studies of persons exposed to external sources of radiation (2), and confirmed by recent studies of persons exposed to ^{131}I as a result of the Chernobyl accident (3, 4), excess thyroid cancers would be expected, especially among those who were children during the period of

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exposure. Shortly following the publication of the NCI report, we examined the relationship of US thyroid cancer mortality and incidence rates and the estimated thyroid doses from fallout (5); little indication of a dose-response relationship was found although both mortality and incidence data suggested an association for dose received under one year of age. The lack of evidence for persons exposed at ages over one year was in contrast to substantial evidence from studies of persons exposed to external radiation, and was attributed to “the limitations inherent in ecological studies, including error introduced when studying a mobile population.”

In this report, we evaluate thyroid cancer incidence data for the period 1973-2004 compared to 1973-1994 in our previous analyses, more than doubling the number of thyroid cancers. Since thyroid cancer is rarely fatal, this re-evaluation is restricted to incidence data. In the US and other parts of the developed world, thyroid cancer incidence rates have increased sharply for the past three decades (6-9). Radioactive fallout from atmospheric weapons testing (10, 11) or Chernobyl (12, 13) has been suggested as one possible reason. Concern about thyroid diseases and other health consequences from exposure from fallout continues (14). An objective of this report is to further investigate the long-term effects of exposure to atmospheric fallout on thyroid cancer incidence.

MATERIALS AND METHODS

Thyroid Cancer Incidence and Dosimetry Data

Thyroid cancer incidence rates were available by single calendar years (1973-2004) and single-year age groups for 194 counties covered by eight Surveillance, Epidemiology, and End Results (SEER) tumor registries (Atlanta, Connecticut, Detroit, Iowa, New Mexico, San Francisco, Seattle and Utah). The population estimates used in calculating these rates are provided by the US Census Bureau (15). Methods used to estimate doses from fallout are described in detail in the NCI report and summarized by Gilbert et al. (5). Mean thyroid doses received in each of several years and age categories were estimated. For the counties included in the SEER program, the mean cumulative dose was 25 mGy (milligray), which was quite similar to the mean cumulative dose of 24 mGy for all counties in the U.S. Mean doses (in mGy) for the SEER counties by calendar year were 0.2 in 1951, 9 in 1952, 6 in 1953, 2 in 1955, 7 in 1957, <0.1 in 1958 and 1 in 1961+. No dose was accumulated in 1954, 1956, 1959 and 1960 because exposure was negligible in those years. The estimated doses depend strongly on age at exposure with mean doses (in mGy) for the SEER counties of 47 for *in utero*, 136 for >0 and <1 y, 114 for 1-4 years, 77 for 5-9 years, 50 for 10-14 years, 35 for 15-19 years, 1 for 20+ years. Dose estimates reflect mean doses for groups and cannot reflect individual variation within these groups. Even the mean doses are subject to large uncertainties.

Statistical Methods

Statistical methods were similar to those used in our earlier paper (5). Each category defined by county, sex, calendar year and single years of age-at-risk was assigned a total dose, which is the sum of the county-specific doses received in the 7 calendar years (see above paragraph) taking account of age at exposure. Dose for the period 1961+ was assumed to have occurred in 1962, when most of it was received. For example, the dose assigned to attained age 35 and calendar year 1980 for a particular county would be the sum of the county-specific doses of a person age 6 in 1951, aged 7 in 1952, aged 8 in 1953, aged 10 in 1955, aged 12 in 1957, aged 13 in 1958, and aged 17 in 1962. Because adequate data for tracking migration from county to county are unavailable, this calculation was necessarily based on the assumption that persons remained in the same county from the period 1951 to 2004. Categories of attained age 65 and older and persons born before 1937 were excluded

because such persons would have been at least 13 in 1952 when the first nuclear test contributing substantially to dose was conducted. Persons born in years later than 1963 were also excluded since they had no opportunity for exposure. Data were collapsed across counties into cumulative dose categories with the following cut points (with a separate category for 0): 0, 1, 2, 5, 10, 15, 20, 30, 40, 60, 90, 120, 160, 240, 360, 480, and 640 mGy.

Dose-response analyses comparing thyroid cancer incidence rates in high-dose counties with those in low-dose counties were performed using Poisson regression methods, and were implemented using the AMFIT module of the software package EPICURE (16). In these analyses, strata including subjects who were young during the period of high exposure had both the largest doses and greatest exposure variability and therefore contributed the most to the dose-response analysis. All p-values reported are two-sided. Dose-response analyses were based on the linear relative risk model in which the cancer risk is given by $\lambda_j [1 + \beta z]$, where j indexes strata defined by age (5-year categories), birth year (single years), and sex, λ_j is the rate in unexposed individuals, β is the excess relative risk (ERR) per Gy, and z is the dose in Gy. The linear relative risk model has been used extensively in analyzing epidemiologic data on radiation risks and was used in our previous study. Confidence intervals for β and tests of the null hypothesis that $\beta = 0$ were based on the likelihood ratio statistic. In addition, relative risks were calculated by dose category using the lowest category as the baseline, and based on a model with cancer risk given by $\lambda_j \exp(\beta_k)$, where k indexes dose categories. Confidence intervals for these relative risks were based on the Wald statistic.

Because of substantial prior evidence (2, 4, 17) that the relative risk of thyroid cancer declines with age at exposure with little evidence of risk for persons exposed in adulthood, our univariate analyses emphasize three dose metrics: dose received before 1 year of age, dose received before 5 years of age, and dose received before 15 years of age; all three doses include dose received *in utero*. Multivariate analyses that simultaneously evaluate risk of doses received at different ages were also conducted. For these analyses, the age categories were < 1 year, 1-4 years, and 5-14 years.

RESULTS

Table 1 shows the distribution of thyroid cancer cases and person-years, crude thyroid cancer rates, and mean doses to the thyroid by registry. Altogether 18,545 thyroid cancers (13,960 in females and 4,585 in males) were included in the analyses, compared with about 9,400 cases that were included in our previous analyses and had potential for exposure before age 15. Mean doses varied substantially by registry. The mean and median age at thyroid cancer diagnosis was 42 years.

Table 2 shows relative risks by categories of dose and age at time of receiving dose, as well as estimated excess relative risks (ERRs) per Gy. There is little evidence of a positive dose-response when doses received before age 5 years or before age 15 years were analyzed, but a significant positive trend with dose is observed for dose received before one year of age ($p = 0.004$) with an estimated ERR per Gy of 1.8 (95% CI=0.4 to 3.2); this estimate was 2.4 (95% CI = 0.6 to 4.5) when restricted to the calendar period 1973-1994 covered by our previous analysis. The ERR per Gy from multivariate analyses that simultaneously evaluated doses received at different ages were as follows (Table 3): 2.0 (95% CI=0.7 to 3.4) for dose received before one year of age; 0.03 (95% CI=-0.6 to 0.7) for dose received at ages 1-4; and -0.7 (95% CI=-1.3 to -0.1) for dose received at ages 5-14. Thus, only for dose received before one year of age was the estimated ERR per Gy significantly greater than zero. We also conducted a multivariate analysis that included dose received at ages 15 years or older. The ERR per Gy for this age category was -1.0 (95% CI = -3.3 to 1.6).

There are about three times as many cases in females as in males (Table 3). For dose received before age one, the sex difference in the ERR per Gy was not statistically significant ($p = 0.08$), but the estimate for females was smaller and did not quite achieve statistical significance ($p = 0.06$). There was no evidence of sex differences for dose received after one year of age, and none of the sex-specific ERR per Gy differed significantly from zero. The ERR per Gy for dose received under one year for specific calendar year periods (Table 3) were erratic without any clear pattern; when the 1973-1984 and 1985-1994 periods were subdivided, the ERR per Gy alternated between estimates near zero and significantly positive estimates. The ERR per Gy increased significantly with calendar year for dose received between 5 and 14 years of age, but this trend came about because of the strongly negative estimate for the period 1973-1984; when we excluded the 1973-1979 time period, the significance of the trend disappeared.

Influence analyses, in which each of the eight SEER cancer registries were excluded one at a time, were also conducted. Results were generally similar to those based on all registries except that when Iowa was excluded, significant dose-response relationships were found not only for dose received under one year of age, but also for dose received at older ages. With Iowa excluded, the estimates of the ERR per Gy in a multivariate analysis were as follows: 2.2 (95% CI=0.4 to 4.3) for dose received before one year of age; 1.1 (95% CI= 0.01 to 2.3) for dose received at ages 1-4; and 1.6 (95% CI= 0.5 to 2.8) for dose received at ages 5-14. Iowa has the highest doses, but the lowest thyroid cancer rate.

DISCUSSION

In this extension of an earlier study relating thyroid cancer incidence to ^{131}I thyroid doses from radioactive fallout from atmospheric nuclear tests conducted at the Nevada Test Site (5), the suggested association of thyroid cancer risk with dose received before the age of one became statistically significant but, as previously, there was no evidence of dose-response for dose received after one year of age. The current study had two notable improvements over the previous one. First, doses could be assigned more accurately because thyroid cancer incidence data were available by single year age groups whereas in the earlier study data were available only by 5-year age periods. This may account for the change in statistical significance for dose received before one year of age since the earlier estimate of the ERR per Gy had a wider confidence interval [2.4 (95% CI = -0.5 to 5.6)] than the estimate based on current data for the same time period 1973-1994 [2.4 (95% CI = 0.6 to 4.5)]. Second, we had an additional 10 years of follow-up (from 1973-2004 compared with 1973-1994), which doubled the number of cases and thus increased statistical precision. The extra years of follow-up also allowed us to evaluate calendar year period, which is highly correlated with time since exposure, in greater detail.

Although a statistically significant association between ^{131}I thyroid dose and thyroid cancer was observed for exposure before one year of age, the ERR per Gy was lower than that observed in most studies of childhood exposure to external radiation (2, 17) and studies of ^{131}I exposure following the Chernobyl accident (3, 4). For example, the ERR per Gy was 9.1 (95% CI=1.3 to 84.8) for children exposed to ^{131}I at ages 0 to 4 as a result of the Chernobyl accident (4), and was 7.7 (95% CI=2.1 to 28.7) in a pooled analysis of children exposed to external radiation under the age of 15 (2). Furthermore, we found little evidence of an excess risk following exposure after age one whereas when an increased risk was observed in other studies, it generally was seen for exposure up to at least age fifteen though the risk decreased strongly with increasing age at exposure (2, 4, 5, 16, 18). It is possible that our study was able to detect the relatively high risk at very young exposure ages but not the lower risks at older exposure ages. On the other hand, the results from most studies of persons exposed to low-dose environmental ^{131}I , other than Chernobyl, have not

demonstrated statistically significant dose response relationships for thyroid cancer (19-21), partly due to the low statistical power for detecting effects when radiation doses are small and the limited size of the study populations.

The risk for dose received before age one was higher among males compared with females and this difference was marginally significant ($p=0.08$). The question of radiation sensitivity by sex has been studied in many radiation-exposed populations, but the findings have been inconsistent. Among populations exposed to ^{131}I from the Chernobyl accident or nuclear weapons testing, no significant difference by sex was observed (3, 4, 18, 20, 21). In a pooled analysis of five studies of persons exposed to external radiation as children, females had a greater risk than males overall, but the difference was not statistically significant and results varied among the individual studies (2).

Because the SEER cancer registry program began in 1973, no cancer incidence data are available for the first 15-20 years after fallout exposure, yet studies of external radiation have shown that risks are especially high during that time window (22). It is of note, however, that in the last calendar year period, 2000-2004, the ERR for dose received under one year of age was significantly elevated. The finding of a high risk in the most recent calendar year period is somewhat unexpected because several studies have suggested that risk begins to decline after about 20-30 years following exposure (22). Furthermore, migration out of the cancer registry catchment area would be expected to rise over time and therefore increase the likelihood of misclassification of both exposure and disease outcome. This type of misclassification generally reduces the probability to detect a risk and attenuates the point estimate of the risk. In the 1990 and 2000 censuses, approximately 60% of the US population was born in the state of current residence and this percentage was only about 50% for California, New Mexico and Washington, states in which the SEER registries were used in our analyses (www.census.gov/prod/cen2000/doc/sf3.pdf. Internet release date January 31, 2005).

As in all ecologic studies, results from this study must be interpreted with caution. Some of the dose-response is due to a comparison across registries and the extent of fallout may not be the only difference in registry catchment areas. Our result regarding the ERR per Gy for exposure received before age one should be considered in the context of the dose actually received by the study population. The ERR at the mean dose of 25 mGy is estimated to be 0.02 and such small risks are vulnerable to potential, but unknown, confounding. Such confounding may be suggested by the negative dose-response for exposure at ages 5 to 15 years as well as the strong difference in risks between the calendar year periods 1973-84 and 1985-2004 for this age group. Such findings are likely the result of factors other than fallout dose that are related to geography, calendar time, or birth cohort, or, alternatively, dosimetry biases that might depend on geography. The positive risk estimates for doses at ages older than one that emerge when Iowa is excluded might be the result of dosimetry or other biases that differ for Iowa compared with the remaining registries.

Another limitation of the study is the uncertain nature of dosimetry for environmental exposures that occurred decades ago. Because no direct thyroid measurements were available for this study, the dose estimates are highly dependent on assumptions regarding peoples' location at the time of the fallout, their milk and food intake and their thyroid gland size (1). For persons exposed under age one, there is likely less uncertainty in the amount of milk consumed but greater uncertainty in the source of milk (i.e., formula, mother's milk, or fresh cow's milk). In addition, thyroid doses from nuclear tests other than those conducted in Nevada have not been considered in this paper, as they remain to be estimated on a systematic basis by county and by year (23). For the two Pacific coast registries, it is likely that these doses are of comparable magnitude to doses from tests in Nevada. Tests outside

Nevada might also contribute substantially in areas where heavy thunderstorms coincided with the passage of contaminated air masses.

In summary, in this ecologic study, we observed a significant dose response between thyroid cancer incidence and ^{131}I exposure from nuclear weapons testing fallout before age one, but did not find such a dose-response for exposure at older ages. Although the latter finding is contrary to other studies that show elevated risk until at least age 15, this discrepancy can be explained by the likely bias in our findings due to large uncertainties in dose estimates and to our inability to take account of individual differences in other potential confounding factors. Based on studies of persons exposed externally and of those exposed to ^{131}I as a result of the Chernobyl accident, it seems likely that exposure from fallout has increased thyroid cancer incidence. Our study adds support for this increase although other explanations of our findings are possible and the data are not adequate to quantify any possible increase.

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TABLE 1

Numbers of person-years, thyroid cancer cases, thyroid cancer crude rates, and mean thyroid dose by SEER cancer registry, 1973-2004^a

Registry	Person-years × 10 ⁻⁵	Thyroid cancer cases (%)	Crude thyroid cancer rate per 10 ⁵ person-years	Mean thyroid dose in mGy (range)
Atlanta, Georgia	187	1643 (8.9)	8.8	14 (13-15)
Connecticut	369	2767 (14.9)	7.5	31 (26-31)
Detroit, Michigan	381	2954 (15.9)	7.8	22 (21-27)
Iowa	332	2394 (12.9)	7.2	50 (37-71)
New Mexico	158	1510 (8.1)	9.6	16 (4-31)
San Francisco, California	354	2572 (13.9)	7.3	4 (3-6)
Seattle, Washington	371	2937 (15.8)	7.9	8 (4-9)
Utah	184	1768 (9.5)	9.6	42 (15-105)
Total	2336	18,545 (100)	7.9	25 (3-105)

mGy = milligray

^aIncludes birth years 1937-1964 and attained ages under

TABLE 2

Numbers of person-years, thyroid cancer cases, and relative risks (with 95% CI) for thyroid cancer incidence by dose and age that dose was received

Thyroid dose (mGy)	Dose received at <1 y of age			Dose received at <5 y of age			Dose received at <15 y of age		
	Person-years $\times 10^{-5}$	Thyroid cancer cases	Relative Risk (95% CI)	Person-years $\times 10^{-5}$	Thyroid cancer cases	Relative Risk (95% CI)	Person-years $\times 10^{-5}$	Thyroid cancer cases	Relative Risk (95% CI)
0–5 ^a	549 ^b	3940 ^b	1.0	618 ^b	4345 ^b	1.0	602 ^b	4185 ^b	1.0
5–10	149	1153	1.08 (1.00-1.17)	174	1341	0.99 (0.92-1.07)	227	1770	1.00 (0.93-1.07)
10–20	97	760	1.12 (1.03-1.22)	131	1040	1.04 (0.95-1.13)	237	1947	1.02 (0.95-1.09)
20–40	95	672	1.06 (0.96-1.18)	253	1990	1.05 (0.98-1.13)	428	3663	1.08 (1.01-1.14)
40–60	53	388	1.10 (0.97-1.24)	164	1320	1.07 (0.99-1.16)	232	1998	1.12 (1.05-1.20)
60–90	51	415	1.26 (1.11-1.42)	201	1641	1.08 (1.01-1.17)	253	2140	1.08 (1.00-1.15)
90–120	14	117	1.29 (1.07-1.57)	108	841	1.05 (0.96-1.15)	168	1420	1.08 (1.00-1.16)
120–240	6	28	0.76 (0.52-1.11)	56	391	0.96 (0.86-1.08)	174	1319	1.00 (0.92-1.08)
240 +	0.3	4	1.82 (0.68-4.88)	1	20	1.64 (1.06-2.56)	14	103	0.92 (0.75-1.12)
P trend ^c			0.004 (+)			0.27 (+)			>0.5 (–)
ERR per Gy			1.8 (0.5, 3.2)			0.3 (–0.2, 0.9)			–0.05 (–0.4, 0.3)
Total	1016	7477		1707	12,929		2336	18,545	

mGy = milligray; CI = confidence interval

^aReferent group.

^bExcludes cases and person-years in birth years with no potential for exposure at age of interest.

^cTest is based on the 18 dose categories indicated in the text. Direction of trend given in parentheses.

TABLE 3

Numbers of person-years, thyroid cancer cases, and excess relative risks (ERRs) per Gy for thyroid cancer incidence by gender and calendar year based on multivariate analyses

	Person-years ^a × 10 ⁻⁵	Thyroid cancer cases ^a	ERR per Gy (95% CI)		
			Dose received at <1 year of age	Dose received at 1-4 years of age	Dose received at 5-14 years of age
All	2336	18,545	2.0 (0.7 to 3.4)	0.03 (−0.6 to 0.7)	−0.7 (−1.3 to −0.1)
By gender					
Males	1170	4585	4.4 (1.4 to 8.1)	−0.6 (−1.8 to 0.9)	−1.0 (−2.1 to 0.3)
Females	1166	13,690	1.3 (−0.04 to 2.9)	0.2 (−0.5 to 1.0)	−0.6 (−1.3 to 0.1)
P for difference			0.075	0.35	>0.5
By calendar year					
1973-1984	877	3964	4.4 (1.5 to 8.0)	−0.5 (−1.7, 0.8)	−2.4 (−3.3 to −1.3)
1985-1994	746	5554	1.9 (−0.2 to 4.3)	−0.1 (−1.1 to 1.1)	−0.4 (−1.5 to 0.7)
1995-1999	367	3878	−0.7 (−1.9 to 1.1)	−0.2 (−1.3 to 1.2)	0.2 (−1.0 to 1.6)
2000-2004	359	5149	3.9 (1.5 to 6.7)	1.0 (−0.3 to 2.5)	0.1 (−1.1 to 1.5)
P trend ^b			>0.5 (+)	0.23 (+)	0.002 (+)

Gy = gray; C CI = confidence interval; ERR=excess relative risk

^aExcludes cases and person-years in birth years with no potential for exposure before age 15.

^bTrend test is based on calendar year categories 1973-1974, 1975-1979, 1980-1984, 1985-1989, 1990-1994, 1995-1999, 2000-2004. Direction of trend is given in parentheses.