Re: Thyroid Cancer Rates and ^{131}I Doses From Nevada Atmospheric Nuclear Bomb Tests

The sophisticated analysis of thyroid cancer and nuclear fallout by Gilbert et al. *(1)* overlooked some important confounders. Mortality from thyroid cancer varies markedly by ethnicity and region *(2)* and by population density. A strong hint of confounding by these factors is present in their Tables 3 and 4 *(1)*, where many of the calculated risk values were negative. The base value (from persons least exposed) appears to have been too high. The reason may be that

Table 1. Average thyroid cancer death rates (95% confidence intervals) per 100 000 in regional samples of both sexes, age adjusted, 1950–1959

Race	Average rates (95% confidence intervals)*						
	Southeast	South Central	Mountain	West Coast	North Central	Northeast	All†
White	0.45 (0.40–0.50)	0.58 (0.54–0.63)	0.58 (0.49–0.67)	0.67 (0.63–0.71)	0.70 (0.66–0.74)	0.77 (0.74–0.81)	0.68 (0.66–0.70)
Nonwhite	0.45 (0.36–0.54)	0.48 (0.39–0.57)	0.93 (0.41–1.45)	1.15 (0.54–1.76)	0.58 (0.46–0.70)	0.62 (0.53–0.71)	0.61 (0.56–0.66)

*Southeast = Florida, Mississippi, Virginia; South Central = Kansas, Louisiana, Texas; Mountain = Colorado, Montana, New Mexico; West Coast = California, Oregon, Washington; North Central = Iowa, Illinois, Michigan; and Northeast = New Jersey, New York, Pennsylvania.

†The all rates data have been population weighted; the others are a direct average of three rates.

the lowest exposed groups lived mainly in densely populated eastern or coastal areas, in contrast to the largely white, rural population in the western mountain states who received the highest exposures. This reason applies to both their incidence and mortality data.

To check on the potential of the confounders noted above, a sampling of counties and states has been used. Population data from 1954 *(3)* and cancer data *(4)* from 1950 through 1959 were used. The white and nonwhite division was the same as that used by Riggan et al. *(4)*, and included Hispanics with whites. This time frame was the period of greatest fallout but before any fallout-induced cancers would have been manifested. In Table 1, with the use of three states from each of six different regions in the United States, the thyroid cancer death rates among whites are found to be markedly different, rising from 0.45 deaths per 100 000 people in one area (southeast) to 0.77 deaths per 100 000 people in the northeast, with the mountain, south, and central states having lower than average rates. Nonwhite thyroid cancer rates exhibit a similar regional variation but do not appear to be substantially different from white rates.

County populations in 1954 from 10 states (Alabama, Connecticut, Delaware, Illinois, Michigan, North Carolina, Oregon, Pennsylvania, Utah, and Wyoming) were used to obtain six size groups. They were selected for region and population distribution. The thyroid cancer rates from 1950 through 1959 ranged from a low of 0.56 (95% confidence interval [CI] = 0.35–0.77) for the 88 counties with fewer than 10 000 people, to 0.61 (95% CI = 0.57–0.65) for the 394 counties with 10 000–199 999, to 0.66 (95% CI = 0.60–0.71) for 27 counties with 200 000–499 999, and to 0.80 (95% CI = 0.75–0.85) for nine counties with more than 500 000 people.

These data from large county and state samples suggest that ethnicity was probably not an important problem, but they emphasize that region and population density probably were problematic. To be valid, the analyses of Gilbert et al. must consider both of these confounders. The regional problem can probably be solved by limiting the analysis to those regions with the higher exposures and nearby less exposed regions or by matching the higher exposed counties with less exposed counties in regions with similar background rates. The population density problem might be solved by simply excluding all counties with a population of more than 200 000 people in 1954. If the authors can revise their analyses to consider these two confounders, it is likely that their radiation risk calculations will approximate those obtained by other studies *(1)*.

The overall average exposure in 1951 was given as 0.0 Gy. This exposure rate seems strange because there were 12 atmospheric tests that year that had an equivalence of about 112 kilotons *(3)*. This rate contrasts with 0.7 and 0.8 Gy attributed to testing in 1952 and 1958 from smaller kiloton-equivalent yields *(3)*. Large radioiodine releases from Hanford, WA, reactors at about the same time must have given additional exposures to people in some of the counties in Washington, Idaho, Oregon, or Montana, but they were not mentioned.

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References

(1) Gilbert ES, Tarone R, Bouville A, Ron E. Thyroid cancer rates and ^{131}I doses from Nevada atmospheric nuclear bomb tests. J Natl Cancer Inst 1998;90:1654–60.

(2) Collin SL. Etiopathogenesis of thyroid cancer. In: Falk S, editor. Thyroid disease. New York (NY): Raven Press; 1990. p. 434.

(3) National Cancer Institute. Estimated exposures and thyroid doses received by the American people from iodine-131 in fallout following Nevada atmospheric nuclear bomb tests. Bethesda (MD): National Institutes of Health, National Cancer Institute; 1997 Report No.: DHHS Publ No. (NIH) 97-4264 and appendices.

(4) Riggan WB, Van Bruggen J, Acquavella JF, Beaubier J, Mason TJ. United States cancer mortality rates and trends, 1950–1979. Vol 3. Washington (DC): Environmental Protection Agency and National Cancer Institute, EPA-600/1/-83-015a, Government Printing Office; 1983.

Note

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Response

We thank Dr. Archer for his comments. We note, however, that higher doses are not restricted to counties in western states with small populations *(1)*. For example, we calculate an average dose of 3.2 cGy for the north central region (Iowa, Illinois, and Michigan) in Dr. Archer's Table 1, which is not substantially lower than the average dose of 3.8 cGy for the mountain region (Colorado, Montana, and New Mexico). We agree that risk estimates from our study may be biased for several reasons, including especially the errors in doses when studying a mobile population, and we state in the abstract that various problems ". . . preclude making a quantitative estimate of risk due to exposure" *(2)*. We question whether further analyses can overcome the basic limitations of this ecologic study.

With regard to the thyroid dose estimation, the small doses estimated for the 1951 tests are due to the fact that all 1951 tests with relatively large yields (>10 kilotons) were detonated at heights above ground of more than 300 meters. As a consequence, most of the radioactive materials stayed aloft and little was deposited on the ground over the territory of the continental United States. In addition, the 1951 tests were clustered around February 1 and November 1, at times when cows are off pasture in many regions of the United States and, therefore, the ^{131}I transfer from ground contamination to milk is minimal. Finally, the average doses for 1952 and 1958 were 0.7 and 0.0 cGy, not 0.7 and 0.8 Gy, respectively, as stated by Dr. Archer.

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REFERENCES

(1) National Cancer Institute. Estimated exposures and thyroid doses received by the American people from iodine-131 in fallout following Nevada atmospheric nuclear bomb tests, a report from the National Cancer Institute. Washington (DC): U.S. Department of Health and Human Services; 1997.

(2) Gilbert ES, Tarone R, Bouville A, Ron E. Thyroid cancer rates and ^{131}I doses from Nevada atmospheric nuclear bomb tests. J Natl Cancer Inst 1998;90;1654–60.

NOTES

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