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Original Contributions

CANCER NEAR THE THREE MILE ISLAND NUCLEAR PLANT: RADIATION EMISSIONS

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As a public charge, cancers among the 159,684 residents living within a 10-mile (16-km) radius of the Three Mile Island nuclear plant were studied relative to releases of radiation during the March 28, 1979, accident as well as to routine plant emissions. The principal cancers considered were leukemia and childhood malignancies. Estimates of the emissions delivered to small geographic study tracts were derived from mathematical dispersion models which accounted for modifying factors such as wind and terrain; the model of accident emissions was validated by readings from off-site dosimeters. Incident cancers among area residents for the period 1975–1985 (n = 5,493) were identified by a review of the records at all local and regional hospitals; preaccident and postaccident trends in cancer rates were examined. For accident emissions, the authors failed to find definite effects of exposure on the cancer types and population subgroups thought to be most susceptible to radiation. No associations were seen for leukemia in adults or for childhood cancers as a group. For leukemia in children, the odds ratio was raised, but cases were few (n = 4), and the estimate was highly variable. Moreover, rates of childhood leukemia in the Three Mile Island area are low compared with national and regional rates. For exposure to routine emissions, the odds ratios were raised for childhood cancers as a whole and for childhood leukemia, but confidence intervals were wide and included 1.0. For leukemia in adults, there was a negative trend. Trends for two types of cancer ran counter to expectation. Non-Hodgkin's lymphoma showed raised risks relative to both accident and routine emissions; lung cancer (adjusted only indirectly for smoking) showed raised risks relative to accident emissions, routine emissions, and background gamma radiation. Overall, the pattern of results does not provide convincing evidence that radiation releases from the Three Mile Island nuclear facility influenced cancer risk during the limited period of follow-up.

environmental exposure; neoplasms; nuclear reactors; radiation, ionizing

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Abbreviations: CI, confidence interval; OR, odds ratio; Gy, gray; Sv, sievert.

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Editor's note: *For a discussion of this paper and for the authors' response, see pages 413 and 416, respectively.*

In the study reported in this paper, we tested a priori hypotheses that risks of specified cancers may have been raised by exposure to radiation emanating from the Three Mile Island nuclear power plant in Pennsylvania. Early on the morning of March 28, 1979, an accident at the facility triggered releases of radioactivity into the environment. The releases were contained shortly thereafter, and an evacuation advisory issued on March 30 was rescinded on April 9. More than a decade later, cleanup of the damaged reactor continues; a second reactor, shut down for refueling at the time of the accident, resumed operation in October 1985.

Monitoring equipment was limited but, from available measurements of accident releases, dosimetry experts calculated average whole-blood gamma exposures of 0.1–0.25 millisieverts (mSv) for persons within 5 miles (8 km) of Three Mile Island (1). These estimates are a fraction of the average US exposure of 0.8–1 mSv from natural background radiation in the course of a year (2). Official reports predicted that, among the population within 50 miles (80 km) of the Three Mile Island facility, at most one cancer death would occur as a result of the accident (3, 4). Nonetheless, when a survey by area residents found a cluster of cancer deaths, there was concern in the community that accident releases had not been estimated accurately (5).

At the request of the Three Mile Island Public Health Fund, we undertook an investigation of whether or not the pattern of cancer occurrence after the March 28, 1979, accident was related to radiation releases from the plant. We proposed a review of record-based data on cancer incidence and mortality, which could be followed by an individual-level study if the findings so warranted. Routine preaccident releases were examined as potentially confounding, since the plant operated for some years prior to the accident and since the risk of cancer from living near nuclear facilities has been the subject of several investigations (6–17). Recent reports from England, Wales, and Scotland (9–11, 13, 17) have seemed to identify a genuine excess of childhood leukemia near some nuclear installations, although the cause is in doubt since levels of radioactivity are too low to explain the excess in terms of conventional risk models (18, 19). Finally, we proposed to account for background gamma radiation, a source of higher exposure than emissions from the nuclear plant, using data available from a national monitoring program.

To increase the sensitivity of the study given very low doses and a short period of observation, we attempted to describe the spatial distribution of radioactivity across small demographic units. Exposure estimates for accident and routine emissions were derived from mathematical models. Estimates for background gamma radiation were based on environmental measurements. Cancers to be analyzed were specified on the basis of radiosensitivity and induction times and, in testing hypotheses,

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we made efforts to control potential confounding factors to the extent possible in record-based research.

MATERIALS AND METHODS

The area studied comprised all census tracts or blocks with geographic centers in a strict 10-mile (16-km) radius of the Three Mile Island nuclear plant, thus forming an approximate 10-mile (16-km) ring around it (figure 1). The defined study area has a population of approximately 160,000 persons. For purposes of analysis, we created 69 "study tracts" (figure 2); these were built up from census blocks and range in population from 500 to 9,500 persons (average, 2,300). In order to estimate the dispersion of radioactive emissions, the study tracts are subtended by angular projections from the plant, with a minimum width of 10 degrees to accommodate a 5-degree uncertainty in the measurement of wind direction at the reactor.

For each study tract, yearly cancer rates were calculated over the period 1975–1985. The study period provides 4 years of observation during routine plant operations and about 7 years of observation after the accident. For the individual years 1975–1979, population totals by sex and 5-year age groups were derived for each study tract from a straight line interpolation between the numbers recorded by the decennial censuses of 1970 and 1980. If a study tract cut across census units, we apportioned the population relative to the size of the segment. For the years 1981–1985, population totals were extrapolated from 1980 to biannual intercensal estimates at the minor civil division level; since these estimates are not age or sex specific, we maintained the age-sex distribution of the 1980 Census. For time periods that combined several years, we used the average of the values for each of the years included.

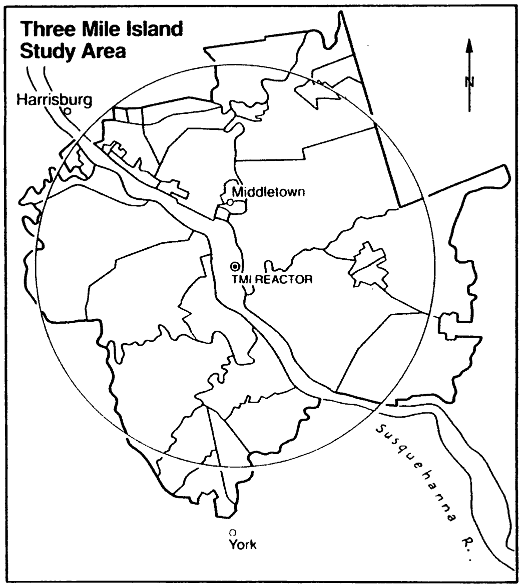

FIGURE 1. Three Mile Island study area showing the main towns, the Three Mile Island (TMI) nuclear plant, and the surrounding area to a 10-mile radius.

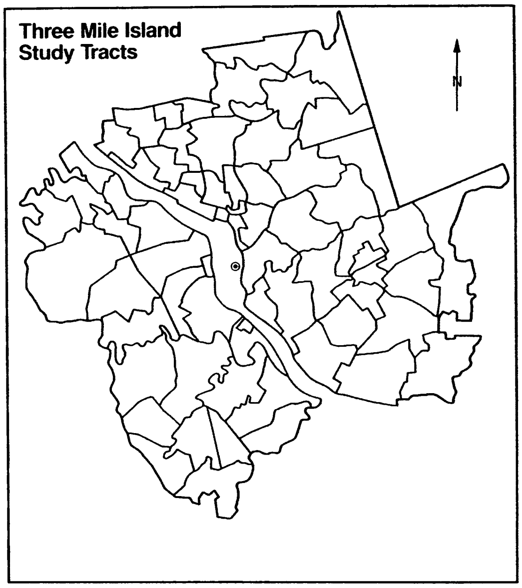

FIGURE 2. Three Mile Island study area subdivided into 69 study tracts (average population, 2,300) for purposes of analysis. ◉, Three Mile Island reactor.

Cancer data

Incident cases of cancer among study area residents during the period January 1, 1975, to December 31, 1985, were sought through a review of hospital records. All 19 hospitals within 30 miles (48 km) of Three Mile Island agreed to let us abstract patient charts (although for some, intercession of the supervising court was required); virtually all charts requested (99 percent) were obtained. We also reviewed the records of six referral hospitals in the nearby cities of Philadelphia and Pittsburgh, Pennsylvania, and Baltimore, Maryland, to increase case ascertainment. A total of 5,493 new cancer cases were diagnosed in study area residents during the 11-year period. All diagnoses were histologically confirmed or otherwise supported by evidence in the medical record (98 percent by pathology, 2 percent by imaging). The primary site was coded according to the *International Classification of Diseases for Oncology* (20). Fatal cancers (n = 3,216) were found through review of Pennsylvania death certificates for the years in question.

Residence at diagnosis was the basis for assigning cancer events to study tracts. When the address of record involved a rural delivery route or a postal box number, the precise location could usually be clarified by means of postal delivery maps. Approximately 4 percent of the cancers could not be assigned with certainty (e.g., when the delivery route passed through two tracts). Such cases were apportioned among all likely study tracts according to the relative population size of each tract (hence, some tracts contain fractions of cases).

Cancer types studied

We selected a priori certain cancer types or groupings of cancers for analysis, because of either short latency periods or sensitivity to low-dose radiation, or both. The primary categories selected were leukemia (excluding chronic lymphocytic leukemia, which is considered to be nonradiogenic (21)) and cancer in children under 15 years of age (the age category 0–24 years was also included to permit comparison with British studies of cancer in young people living near nuclear facilities). We prespecified lymphoma as a type for analysis because of the similarity (i.e., B-cell origin) between non-Hodgkin's lymphoma and certain lymphocytic leukemias. We also examined the grouping "all cancers," for the sake of completeness. Associations with the "all cancer" category were explored through post hoc analysis of two subgroupings, lung cancer and all neoplasms minus lung cancer.

Radiation emissions at Three Mile Island

The accident at Three Mile Island involved releases of radioactive noble gases, primarily xenon, and, to a much lesser extent, iodine; no other isotopes were detected in the air or the soil (22). Thus, exposures from accident emissions were to whole-body gamma radiation and a small component of beta radiation. Shielding by building construction materials reduced indoor gamma exposures by about 25 percent (23). In the absence of high linear-energy-transfer radionuclides, there was little exposure to internal radiation. Most of the accident releases were estimated to have occurred during the evening of the first day and the early morning hours of the second day, when winds were steady to the northwest (24). Because the vent-stack monitor at the reactor went "off scale" and the thermoluminescent dosimeters located outside the plant provided incomplete coverage, there was uncertainty about the magnitude of the releases (25). Published estimates vary but, in every instance, the level of exposure was deemed to be very low, an average of approximately 0.1 mSv, with 1 mSv the projected maximal dose.

No data were available concerning releases during routine operation of the Three Mile Island plant, but such emissions are presumed to be considerably less than the accident emissions. According to the National Council for Radiation Protection and Measurements, the maximal exposure to persons living within 50 miles (80 km) of pressurized water-power reactors of the type at Three Mile Island is 0.006 mSv per year (26).

Exposure models

Because exposures to Three Mile Island plant emissions were low and their absolute magnitude was uncertain, we adopted a strategy designed to delimit the portion of the study area that received nonzero exposures and to assign exposure in relative terms, without specifying absolute values. We used mathematical models to predict exposure patterns for both accident releases and routine emissions, taking into consideration such factors as height of the releases, temperature and stability, wind speed and direction, height and location of receptor points, and the modifying effect of terrain. Both models (accident emissions and routine emissions) consider nighttime releases, when people were more likely to have been at home. Each model estimates the exposure to a study tract relative to an arbitrary scaling parameter chosen for convenience. The relative units of the two models are not directly comparable.

Model 1: accident emissions. Estimates of radioactive releases were derived indirectly from the only monitors within the plant that remained on scale for most of the accident (24). Vent-stack emissions were assumed to vary proportionally with the radioactivity levels recorded inside the plant. Meteorologic data, for 15-minute time segments over the 48 hours from the onset of the accident, came from a weather station maintained by the plant. Terrain contours at 100-foot (30.5-m) vertical intervals were generalized from a cartographer's reduced tracing of the 1/24,000 scale US Geological Survey quadrangles, except for one contour traced at the height of the reactor stack (465 feet (142 m)). A gaussian dispersion model (27), modified to account for terrain and wind shifts (28, 29), projected concentrations of radioactivity to a grid of points selected to reflect the distribution of population. Exposures from the concentrations surrounding each grid point were integrated by using standard methods (30, 31) and were averaged to arrive at summary values for each study tract. Estimates for study tracts are robust to variations in uncertain parameters that affect the release and dispersion meteorology (32). (Details of the modeling are available, on request, from one of the authors (J. B.).)

Figure 3 illustrates the exposure pattern predicted by the model. Although the principal analysis treats each study tract separately, the figure shows study tracts grouped into quartiles by relative exposure, solely for ease of presentation. The highest accident exposures are projected to the north and northwest of the plant; zero exposures were estimated for 20 of 69 study tracts.

Model 2: routine emissions. Dispersion modeling was also used to estimate the relative pattern of emissions associated with routine operation of the plant prior to the accident (figure 4). Releases of radioactivity follow engineering demands rather than a fixed schedule and, thus, are random with respect to season and weather. Hence, the model assumes a constant average release; the distribution of emissions then becomes a function of the average annual wind pattern (again derived from site-specific weather data) and the modulating effects of terrain contours. The pattern projected by the dispersion model locates

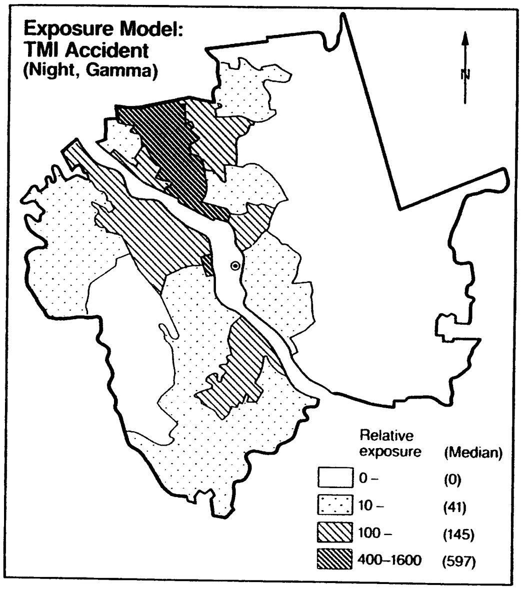

FIGURE 3. The relative distribution of gamma radiation from the March 1979 accident at the Three Mile Island (TMI) nuclear plant, as predicted by mathematical dispersion modeling. (Units given are relative to an arbitrary scaling factor; see the text for discussion.) ◉, Three Mile Island reactor.

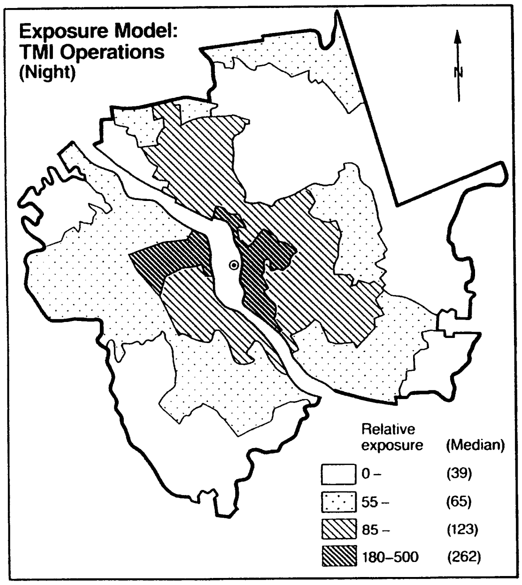

FIGURE 4. The relative distribution of emissions related to the routine operation of the Three Mile Island (TMI) nuclear plant, as predicted by mathematical dispersion modeling. (Units given are relative values and are not directly comparable to the values for the accident model.) ◉, Three Mile Island reactor.

higher exposures close to the plant, particularly between east and west.

Background gamma radiation. Figure 5 shows the distribution of background gamma radiation. Estimates of outdoor gamma ray exposure rates by study tract are based on measurements recorded with scintillation detectors and associated instrumentation in a 1976 airborne radiologic survey that is part of a national program to monitor radioactivity around nuclear facilities (33). The aerial survey did not directly assess the radon component of background radiation. Dose rates ranged from 0.057 to 0.105 μGy/hour (5.7–10.5 μR/hour), with higher exposure levels to the south and southeast of the study area.

Figures 3–5 show quite distinct patterns. Correlations among the three sources of radiation are low, with the greatest overlap between accident and routine emissions (Pearson correlation coefficients: accident and background gamma, $r = -0.27$; accident and routine emissions, $r = 0.37$; and background gamma and routine emissions, $r = -0.28$).

Comparison of accident model predictions with off-site monitors

To test the validity of the accident model, we analyzed the data obtained from all 20 thermoluminescent dosimeters located around the Three Mile Island plant during the accident (32). A best fit to these 20 data points, consistent with the meteorology, gives an emissions pattern in time that can be used to project exposures to each study tract. The agreement between predictions generated from the dosimeters and those made by the model is excellent ($r = 0.92$).

Translating the relative units of exposure models into absolute terms

The fact that exposure patterns projected by the model and data from the thermoluminescent dosimeters compare so well indicates that available monitors were probably adequate to characterize accident releases. Thus, the comparison provides a justification for using official exposure estimates as "best estimates" of the level of radiation corresponding to points along our relative scale. Accordingly, for accident emission exposures of study tracts, the up-

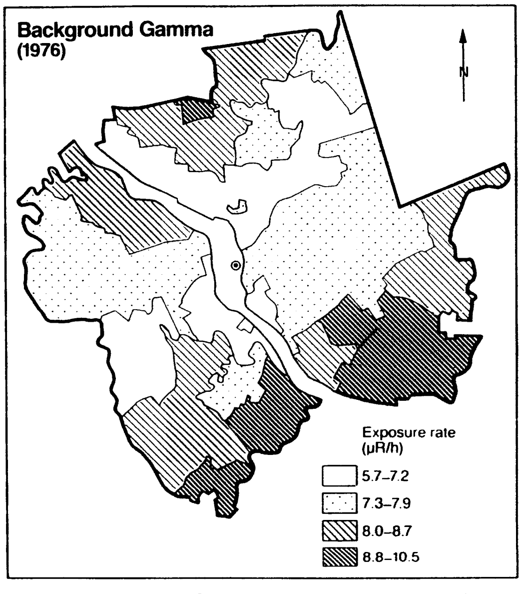

FIGURE 5. The distribution of exposure to background gamma radiation in the Three Mile Island study area as measured in a 1976 aerial survey (33). ◉, Three Mile Island reactor.

per limit of 1,600 relative units (see figure 3 legend) is equivalent to about 1 mSv, similar to the estimated annual exposure from natural background radiation. The median exposure of study tracts in the upper quartile for accident emissions is equivalent to approximately 0.4 mSv (40 percent of annual natural background), and the average exposure over all study tracts is equivalent to about 0.1 mSv (10 percent of annual natural background).

For routine emissions, such an exercise is less well founded. If, however, one sets the upper limit of 500 on the relative scale to be equivalent to the value of 0.006 mSv given by the National Council for Radiation Protection and Measurements, then one could estimate the average exposure to routine emissions over all study tracts at 0.001 mSv, or about 0.1 percent of natural background.

Statistical methods

The major statistical analysis, a logistic regression based on maximum likelihood estimation (34), was an examination of the rates of cancer in relation to radiation exposure, with each source of radiation initially evaluated separately. Odds ratios derived from the logistic regression estimates were calculated contrasting median exposure in the highest quartile to that in the lowest quartile. When associations were noted for one type of exposure, we examined whether or not the association was maintained when other sources of radiation exposure were entered into the model.

The regression analysis was adjusted for sex and age by using indicator variables for age-sex combinations (5-year age intervals under age 15 years and 10-year intervals over age 15 years). Age-sex groupings with zero events were merged with the next age-sex category. Since the population is over 96 percent white, no adjustment was made for race. Data in medical records on smoking habits and occupation were inadequate to permit statistical adjustment. For each of the 69 study tracts, we introduced a measure of urbanization (population per km^2) and two measures of social class (median income and percentage of high school graduates) based on aggregate data; these measures were treated as continuous variables. A preliminary examination of the data for overdispersion in cancer rates yielded little evidence that extravariability was present; thus, the standard errors estimated from the logistic analysis have not been corrected to allow for it.

The logistic regression is the primary analysis upon which interpretations are based. We also computed standardized incidence ratios by quartile of exposure in order to provide estimates of effect close to the observed data as well as a comparison of area rates with national values. The expected numbers of cancers were derived from age-sex-specific rates for whites in the national Surveillance, Epidemiology, and End Results program (1978–1981, Puerto Rico excluded) (35). These national rates are generally comparable with the 1983 cancer registration rates reported for south-central Pennsylvania, although respiratory cancer is an exception, with regional rates substantially lower (36). While this discrepancy affects the absolute values of the standardized incidence ratios for lung cancer, it will not affect internal comparisons across exposure quartiles.

Analysis of accident emissions. For evaluation of accident emissions, the set of analyses described above was performed for both the preaccident period (1975 to March 1979) and the postaccident period, allowing 2-year (1981–1985) and 5-year (1984–1985) latency intervals after the accident. Trends within and between periods were examined. Where indicated, we performed an unconditional logistic regression analysis to control for underlying heterogeneity among the study tracts *prior* to the accident. This analysis, which is tantamount to adjusting for baseline risk, incorporated a parameter for each study tract, an indicator variable for period (pre- or postaccident), and a dose variable coded zero prior to the accident and coded according to its assigned value in the postaccident period; adjacent study tracts with similar relative exposures were grouped as needed to ensure a minimum of

3–5 cancers of the type studied over a given period. A conditional analysis based on a Poisson model gave similar results and is not presented.

Analysis of routine emissions. Data collection for a "baseline" period before start-up of the plant in 1974–1975 proved infeasible since hospital records dating back to that time were usually not stored in retrievable form. Thus, adjustment for baseline risk was not possible in the analysis of routine emissions. Emissions attributable to routine plant operations occurred only in the period before the accident since, after the accident, the plant shut down entirely. Hence, different time intervals are emphasized in the analyses of the accident and routine emissions. By definition, accident effects must arise in the postaccident period, whereas in evaluating routine emissions we looked for trends throughout the 11 years of observation. Data are presented for 1975 to March 1979 (the period of plant operation), April 1979 to 1985 (the period when the plant was not operating), and 1975 to 1985 (all 11 years combined).

RESULTS

For each radiation variable, the distribution of population, density, income, and level of education for study tracts grouped into exposure quartiles is presented (table 1). The proportion of the population in the upper quartiles is relatively small. Estimates of both accident and routine emissions tend to be higher in urbanized areas and in areas with a lower median income; the educational level also varies with the estimated exposure to plant emissions. Our interpretation emphasizes the logistic analysis which adjusts for these sociodemographic differences although, when sample size is small, multivariate estimates must be viewed cautiously.

Results are presented for incident cancers only. Changes in incidence occur earlier than do changes in mortality and are unaffected by survival factors; moreover, resources did not permit validation of death certificate diagnoses. (The analyses of mortality data are available on request from the authors.)

Accident emissions

For leukemia in adults, the odds ratios (ORs) and confidence intervals (CIs) derived from the logistic regression analyses (table 2) show little evidence of increased risk at either 2-year (OR = 1.4; 95 percent CI 0.8–2.6) or 5-year (OR = 1.1; 95 percent CI 0.4–3.1) latency intervals after the accident. Leukemia subtypes were not inves-

TABLE 1

Descriptive data for areas classified by estimated level of exposure to accident and routine emissions from the Three Mile Island nuclear plant, Pennsylvania

Exposure quartiles in relative units*	1980 population†	Density per km^2	Median income ($)	% of high school graduates
Accident				
0–9.9 (0)‡	52,972	200	19,735	66.7
10–99.9 (41)	47,040	260	19,692	65.3
100–399.9 (145)	40,048	664	19,202	70.4
400–1,600.0 (597)	19,624	637	17,988	70.1
Routine				
0–54.9 (39)	66,470	252	19,788	66.7
55–84.9 (65)	53,461	359	19,953	70.0
85–179.9 (123)	32,843	509	18,916	67.9
180–500.0 (262)	6,910	467	17,440	61.4

* Units given represent relative rather than absolute values. The units for accident and routine emissions use different scaling parameters and are not directly comparable (see text for discussion).

† Midyear population estimates; adjustment has been made to the census figures for April 1, 1980.

‡ Numbers in parentheses, median.

TABLE 2

Accident emissions and cancer incidence: standardized incidence ratios and odds ratios† for specific cancer types at different times before and after the accident at the Three Mile Island nuclear plant, Pennsylvania, 1975–1985*

Cancer grouping	Standardized incidence ratios (observed) at the following levels of exposure				Odds ratio	95% confidence interval
	1 (lowest)	2	3	4 (highest)		
			Childhood cancers			
Age, 0–14 years						
1975–1979	1.70 (11)‡	0.23 (1.3)	0.95 (4.7)	0.87 (2)	0.67	0.18–2.47
1981–1985	0.51 (4)	0.94 (6)	0.87 (5)	1.17 (3)	1.06	0.40–2.82
1984–1985	0.63 (2)	0.38 (1)	1.29 (3)	0.00 (0)	0.39	0.01–12.02
Age, 0–24 years						
1975–1979	1.10 (18)	0.10 (1.3)	0.71 (8.7)	1.04 (6)	1.21	0.64–2.29
1981–1985	0.82 (17)	0.79 (13)	0.82 (12)	0.73 (5)	0.995	0.57–1.75
1984–1985	0.95 (8)	0.00 (0)	1.02 (6)	0.37 (1)	0.82	0.26–2.38
			Leukemia§			
Age, 0–14 years						
1975–1979	0.51 (1)	0.00 (0)	0.00 (0)	0.00 (0)		
1981–1985	0.42 (1)	0.00 (0)	1.14 (2)	1.28 (1)	2.28	0.40–12.82
1984–1985	0.00 (0)	0.00 (0)	1.41 (1)	0.00 (0)		
Age, 0–24 years						
1975–1979	0.36 (1)	0.00 (0)	0.00 (0)	0.00 (0)		
1981–1985	0.29 (1)	0.36 (1)	0.80 (2)	0.88 (1)	2.81	0.49–16.19
1984–1985	0.00 (0)	0.00 (0)	0.99 (1)	0.00 (0)		
Age, ≥25 years						
1975–1979	0.68 (7.8)	0.90 (11.2)	0.65 (6)	0.41 (2)	0.59	0.15–2.38
1981–1985	0.87 (14.1)	1.05 (16.3)	0.99 (11.6)	1.16 (7)	1.43	0.77–2.63
1984–1985	0.91 (6)	1.43 (9)	0.85 (4)	1.65 (4)	1.12	0.41–3.08
			Lymphoma			
Non-Hodgkin's lymphoma						
1975–1979	0.71 (16.5)	1.00 (23)	1.11 (19.5)	0.87 (8)	0.95	0.53–1.72
1981–1985	0.74 (22.2)	1.07 (30.9)	1.14 (24.8)	1.14 (13)	1.48	0.99–2.20
1984–1985	0.75 (9.1)	0.76 (8.9)	1.14 (10)	1.31 (6)	2.01	1.15–3.49
Hodgkin's disease						
1975–1979	0.36 (2.4)	1.51 (9)	0.90 (4.6)	2.36 (6)	1.02	0.56–2.78
1981–1985	0.47 (4)	1.45 (11)	1.10 (7)	0.64 (2)	0.85	0.33–2.14
1984–1985	0.58 (2)	1.96 (6)	1.95 (5)	0.79 (1)	1.04	0.37–2.92
			All cancer			
1975–1979	0.72 (538.6)	0.71 (525.5)	0.72 (403.8)	0.85 (254.1)	1.02	0.92–1.13
1981–1985	0.89 (846.9)	0.94 (874.8)	1.01 (707.4)	1.08 (401.8)	1.11	1.03–1.21
1984–1985	0.96 (372.5)	0.93 (351.5)	1.02 (286.5)	1.10 (163.4)	1.14	1.00–1.29

* For calculation of standardized incidence ratios, expectations are based on the Surveillance, Epidemiology, and End Results program (1978–1981) age-sex-specific rates for whites (excluding Puerto Ricans) (35).

† Odds ratios comparing the median exposure in the highest quartile with the median exposure in the lowest quartile are derived from logistic analysis adjusted for age, sex, density, median income, and education. Indicator variables were used for age-sex categories (5-year intervals under age 15 years and 10-year intervals at age 15 years and over); all other covariates were defined as continuous measures.

‡ Numbers in parentheses, number of observed cancers (fractional numbers of observed cancers result from allocation of data across study tracts in cases where address information did not identify the exact location).

§ Excludes chronic lymphocytic leukemia.

tigated because of small numbers. For leukemia in children, there were only four cases observed after the accident, allowing for a 2-year latent period. While the odds ratio for childhood leukemia is elevated (2.3), the confidence interval is very wide, and the lower limit is well below unity (95 percent CI 0.4–12.8). For all childhood cancers, there is no association with accident emissions in the 1981–1985 interval (OR = 1.1; 95 percent CI 0.4–2.8). In very young children (age, 0–9 years), the trend is toward decreased risk with increasing exposure (data not shown).

Non-Hodgkin's lymphoma appears to relate to accident emissions. The odds ratio, assuming a 5-year latency interval, is 2.0 (95 percent CI 1.2–3.5). Inspection of the observed incidence data shows that the regression result reflects both an increased risk at the highest dose level and a decreased risk at the lower dose levels.

The category "all cancers" had a modest relation with predicted accident emissions (OR = 1.1; 95 percent CI 1.0–1.2). On further analysis, the association for this category proved to be entirely accounted for by lung cancer (table 3). Prior to the accident, a gradient in lung cancer by exposure level was also present, and we therefore carried out the analysis described above to correct for risk at baseline. After adjustment for baseline risk, the odds ratio for lung cancer was reduced from 1.8 (95 percent CI 1.5–2.1) to 1.3 (95 percent CI 0.9–1.8).

Routine emissions

Each prespecified cancer category was also examined in relation to exposures from routine plant releases as predicted by the dispersion model. In view of the findings for accident emissions, table 4 subdivides the data on all cancers into lung cancer and all minus lung.

Routine emissions showed no association with leukemia in adults; in fact, the trend is in the opposite direction. For childhood leukemia, the data are sparse. Although odds ratios are above unity, confidence intervals are wide, even when all eight cases over the 11-year period are considered (OR = 2.3; 95 percent CI 0.6–9.7). For all childhood cancers, the association with routine emissions is modest (for 1975–1985, OR = 1.5; 95 percent CI 0.7–3.5).

As in the case of accident emissions, non-

TABLE 3

Three Mile Island accident emissions and all cancer incidence: standardized incidence ratios and odds ratios† for subgroupings of lung cancer and all other cancer, 1975–1985*

Cancer grouping	Standardized incidence ratios (observed) at the following levels of exposure				Odds ratio	95% confidence interval
	1 (lowest)	2	3	4 (highest)		
All minus lung						
1975–1979	0.77 (493.5)‡	0.73 (463.3)	0.75 (353.1)	0.87 (219.1)	0.99	0.88–1.12
1981–1985	0.93 (758.7)	0.93 (737.4)	0.99 (586.9)	0.98 (308)	1.00	0.91–1.10
1984–1985	1.00 (327.8)	0.92 (293.3)	1.01 (240.9)	0.99 (124)	1.02	0.89–1.18
Lung						
1975–1979	0.41 (45.1)	0.56 (63.2)	0.60 (50.7)	0.76 (35)	1.28	0.95–1.72
1981–1985	0.62 (88.2)	0.97 (137.4)	1.14 (120.5)	1.63 (93.9)	1.75	1.47–2.08
1984–1985	0.78 (44.8)	1.02 (58.2)	1.08 (45.6)	1.71 (39.4)	1.72	1.33–2.22

* For calculation of standardized incidence ratios, expectations are based on the Surveillance, Epidemiology, and End Results program (1978–1981) age-sex-specific rates for whites (excluding Puerto Ricans) (35).

† Odds ratios comparing the median exposure in the highest quartile with the median exposure in the lowest quartile are derived from logistic regression analysis adjusted for age, sex, density, median income, and education. Indicator variables were used for age-sex categories (5-year intervals under age 15 years and 10-year intervals at age 15 years and over); all other covariates were defined as continuous measures.

‡ Numbers in parentheses, number of observed cancers (fractional numbers of observed cancers result from allocation of data across study tracts in cases where address information did not identify the exact location).

TABLE 4

Routine emissions and cancer incidence: standardized incidence ratios and odds ratios† for specific cancer types at different times before and after routine operation of the Three Mile Island nuclear plant, 1975–1985*

Cancer grouping	Standardized incidence ratios (observed) at the following levels of exposure				Odds ratio	95% confidence interval
	1 (lowest)	2	3	4 (highest)		
			Childhood cancers			
Age, 0–14 years						
1975–1979	0.56 (4.4)‡	1.40 (9.1)	0.62 (2.4)	2.97 (3)	2.04	0.62–6.73
1979–1985	0.68 (8.3)	1.06 (10.7)	1.40 (9.0)	1.23 (2.0)	1.20	0.37–3.90
1975–1985	0.68 (12.7)	1.23 (19.8)	1.11 (11.4)	1.94 (5.0)	1.52	0.66–3.53
Age, 0–24 years						
1975–1979	0.39 (7.6)	0.97 (15.8)	0.77 (7.6)	1.42 (3)	2.05	0.81–5.23
1979–1985	0.82 (26.3)	0.94 (24.7)	0.84 (14.0)	1.43 (5.0)	1.07	0.45–2.54
1975–1985	0.66 (33.9)	0.96 (40.5)	0.80 (21.6)	1.45 (8.0)	1.38	0.73–2.61
			Leukemia§			
Age, 0–14 years						
1975–1979	0.14 (0.3)	0.34 (0.7)	0.00 (0)	0.00 (0)		
1979–1985	0.00 (0)	0.32 (1)	2.54 (5)	1.97 (1)	2.50	0.61–10.34
1975–1985	0.06 (0.3)	0.34 (1.7)	1.58 (5)	1.25 (1)	2.32	0.56–9.68
Age, 0–24 years						
1975–1979	0.10 (0.3)	0.24 (0.7)	0.00 (0)	0.00 (0)		
1979–1985	0.19 (1.0)	0.45 (2.0)	1.77 (5.0)	1.51 (1.0)	2.33	0.59–9.23
1975–1985	0.16 (1.3)	0.38 (2.7)	1.10 (5.0)	0.96 (1.0)	2.16	0.54–8.58
Age, ≥25 years						
1975–1979	0.91 (15.5)	0.43 (5.5)	0.52 (4)	1.38 (2)	0.42	0.11–3.38
1979–1985	0.91 (26.0)	1.02 (22.1)	1.14 (15.3)	0.64 (1.6)	0.89	0.31–2.52
1975–1985	0.90 (41.5)	0.79 (27.6)	0.89 (19.3)	0.91 (3.6)	0.79	0.31–1.98
			Lymphoma			
Non-Hodgkin's lymphoma						
1975–1979	0.93 (29.7)	0.83 (20.2)	1.03 (14.6)	0.91 (2.5)	1.06	0.40–2.84
1979–1985	0.76 (40.4)	0.91 (37.1)	1.10 (27.2)	1.79 (8.2)	2.13	1.29–3.51
1975–1985	0.81 (70.1)	0.87 (57.4)	1.05 (41.8)	1.48 (10.6)	1.81	1.16–2.82
Hodgkin's disease						
1975–1979	1.06 (9)	1.12 (7.6)	0.69 (2.8)	3.04 (2.6)	1.44	0.37–5.59
1979–1985	1.12 (16.0)	1.05 (12.0)	1.13 (8.0)	1.38 (2.0)	1.12	0.36–3.46
1975–1985	1.08 (25.0)	1.06 (19.6)	0.95 (10.8)	1.99 (4.6)	1.24	0.52–2.96
			All cancer			
All minus lung						
1975–1979	0.74 (640.6)	0.81 (535.5)	0.72 (278.1)	1.01 (73.8)	1.14	0.93–1.39
1979–1985	0.90 (1,313.5)	0.98 (1,088.6)	1.00 (673.9)	1.07 (132.0)	1.10	0.95–1.26
1975–1985	0.83 (1,954.2)	0.91 (1,624.1)	0.88 (950.0)	1.05 (205.8)	1.12	0.996–1.26
Lung						
1975–1979	0.49 (75.8)	0.57 (67.4)	0.64 (42.4)	0.63 (8.3)	1.31	0.78–2.18
1979–1985	0.76 (97.5)	0.96 (193.2)	1.26 (144.3)	1.27 (28.0)	1.55	1.18–2.03
1975–1985	0.65 (273.3)	0.80 (260.6)	1.01 (186.7)	1.04 (36.3)	1.50	1.18–1.91

* For calculation of standardized incidence ratios, expectations are based on the Surveillance, Epidemiology, and End Results program (1978–1981) age-sex-specific rates for whites (excluding Puerto Ricans) (35).

† Odds ratios comparing the median exposure in the highest quartile with the median exposure in the lowest quartile are derived from logistic analysis adjusted for age, sex, density, median income, and education. Indicator variables were used for age-sex categories (5-year intervals under age 15 years and 10-year intervals at age 15 years and over); all other covariates were defined as continuous measures.

‡ Numbers in parentheses, number of observed cancers (fractional numbers of observed cancers result from allocation of data across study tracts in cases where address information did not identify the exact location).

§ Excludes chronic lymphocytic leukemia.

Hodgkin's lymphoma and cancer of the lung show associations with estimated exposure from routine emissions, while the category all minus lung shows little if any relation. For the period 1975–1985, the odds ratio for non-Hodgkin's lymphoma is 1.8 (95 percent CI 1.2–2.8); for lung cancer, the odds ratio is 1.5 (95 percent CI 1.2–1.9). These associations are affected only slightly by adjustment for exposure to accident releases (and vice versa).

Background gamma radiation

Exposures of study area residents to background gamma radiation over the course of a year (not to mention cumulatively) are higher than the average exposures estimated from accident or routine emissions (0.5–1.0 mSv annually from background compared with an average of 0.1 mSv from accident releases over a few days and 0.001 mSv per year from routine emissions). It was thus of interest to know whether the two cancer types related to plant emissions also showed relations with background gamma radiation. For lung cancer, there is a slight trend in risk with background gamma radiation (OR = 1.1; 95 percent CI 0.9–1.4). For non-Hodgkin's lymphoma, no association is seen.

DISCUSSION

We undertook this investigation as a response to public concern, despite the low estimates of Three Mile Island accident releases and the brief interval thereafter. The prior expectation based on estimated releases and conventional radiobiology—that no excess cancer would be found—was confirmed in most if not all respects.

Accident emissions

Leukemia is believed to be sensitive to low-dose ionizing radiation and to have a relatively short latent period (21). In adults in the Three Mile Island area, leukemia shows little association with the exposure predicted by the accident emissions model. Follow-up has been long enough to accommodate at least an early peak, and yet the odds ratio after a 5-year latency interval is barely above unity. In children, the odds ratio was raised substantially, but the estimate of effect was so variable that results must be viewed as indeterminate, particularly in light of the unexplained observation of fewer cases overall than predicted from national or regional rates. For childhood cancers as a group, another focus of the analysis, the trends with exposure in the postaccident period were nil (or in the case of younger children (age, 0–9 years), negative). Thus, we observe no definite associations of accident emissions with the cancer types or population subgroups thought to be most susceptible to radiation carcinogenesis and to be most likely to demonstrate increases in a short period of follow-up. In addition, post hoc analyses of highly radiosensitive sites with longer latent periods, such as the breast and the thyroid, indicate either no association or negative trends with exposure.

We did find an association of accident exposure with non-Hodgkin's lymphoma (OR = 2.0; 95 percent CI 1.2–3.5), but the relation rests on few cases and the result is thus unstable. It would not be surprising, given multiple hypotheses, to find one such association simply by chance. Indeed, whether or not non-Hodgkin's lymphoma is a radiogenic cancer even at very high doses has now been called into question by recent evidence from Japan (37, 38).

An association between lung cancer and predicted accident exposure emerged in post hoc analyses testing a positive finding for the category "all cancers." After adjustment for a gradient in risk prior to the accident (largely contributed by males aged 75 years and older), the postaccident data indicate about a 30 percent increase in lung cancer for residents of the highest exposure quartile compared with the lowest quartile. The association is stronger in males, but also is present in females.

Confounding by smoking cannot be ruled out as an explanation for the lung cancer result, although the adjustment for baseline risk should account to some extent for such factors as smoking, radon, and occupational exposures. To evaluate confounding by smoking in the postaccident period specifically, we examined trends for esophageal and laryngeal cancer which are smoking related but not especially radiosensitive (20). Cancer of the larynx showed an association with accident exposure (OR = 2.1; 95 percent CI 1.2–3.8), while esophageal cancer showed the reverse, with the risk declining with accident exposure (OR = 0.2; 95 percent CI 0.0–2.4). Although results for smoking-related cancers in the postaccident period are conflicting, it is apparent from the preaccident gradient that one or more lung cancer risk factors are operating to produce an exposure pattern very similar to the pathway for the radioactive plume.

Routine emissions

Concerning routine emissions, early studies showed no cancer risk for residents near nuclear facilities or atomic test sites (6–8, 39). Recent studies are suggestive of excess leukemia among children and young adults (9–11, 13, 16, 17, 40) but, in view of the low exposures, radiation is considered an unlikely cause, and alternative explanations are being pursued (19).*

Leukemia and, to a lesser extent, other cancers in children do show raised risks in relation to our models of routine emissions. In all cases, however, confidence intervals are wide, and estimates are compatible with no association as well as with quite strong associations. For all cancers in children under age 15 years, the odds ratio is 1.5 (95 percent CI 0.7–3.5); the odds ratio for younger children (age, 0–9 years) is 1.2 (95 percent CI 0.4–3.7). For leukemia, the odds ratio among children (age, 0–14 years) is 2.3 (95 percent CI 0.6–9.7) and, among children under age 10 years at the time of diagnosis, the odds ratio is 2.7 (95 percent CI 0.6–13.6). The results for leukemia must be placed in the context of a local incidence well below the regional and national incidence. For childhood cancers as a whole, the Three Mile Island rates are similar to national rates. To minimize underascertainment, we directed a special effort at regional referral hospitals for children; this increased our yield of childhood leukemia by one case. It is possible that additional cases would have been found had we included still more distant referral centers (e.g., Sloan-Kettering Cancer Institute in New York) but, at present, the low incidence of childhood leukemia in the Three Mile Island area is unexplained.

Among adults, leukemia was not associated with routine emissions, but lung cancer and non-Hodgkin's lymphoma were. These same cancers were considered in two recent studies of mortality among residents near all nuclear installations in England and Wales (11, 17). The studies used different methodologies; neither found increased risk for lung cancer or non-Hodgkin's lymphoma.

Lung cancer, non-Hodgkin's lymphoma, and Three Mile Island plant emissions

The fact that we observed raised risks for lung cancer and non-Hodgkin's lymphoma with both accident and routine plant emissions deserves comment. If the associations reflect radiation exposure, then the findings from our analyses of plant emissions and background radiation are not easily reconciled. Non-Hodgkin's lymphoma was related to accident and routine emissions but was unrelated to background gamma radiation; moreover, as mentioned above, it is now thought not to be a radiosensitive cancer. The association could be due to some correlated factor that increases the risk of non-Hodgkin's lymphoma. Acquired immunodeficiency syndrome might be such a factor (41), given the time frame, but in this low-risk area, neither the hospital records nor the age, sex, or marital status distributions gave any indication that this was so. Lung cancer is considered

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* Note added in proof: The latest follow-up study, published since this paper was completed, relates the excess cancers in children near the Sellafield nuclear facility in England to preconception radiation exposure of fathers working at the plant (Gardner MJ, et al. Br Med J 1990;300:423–9).

a radiosensitive site (21), and it showed associations with background radiation as well as plant emissions. The odds ratios (1.1 for background, 1.3 for accident emissions, and 1.5 for routine emissions) are, however, inconsistent with the relative magnitudes of the three exposures. In addition, the observation period is short for a cancer of long latency. If radiation emitted by the plant contributed to the trends in lung cancer, it would have to be acting at a late stage in the disease; in this connection, a promoting role for gamma radiation (as opposed to alpha particles) is a possibility, but remains to be established (42). What geographic factors other than radiation could explain these associations is also unclear. Smoking habits are the obvious candidate; if so, smoking frequencies must have been congruent with the quite different geographic distributions of the three radiation exposures.

Limitations of ecologic studies

In relation to the findings presented here, the limitations of ecologic studies need to be considered. Validation of the accident model supports the dispersion modeling approach as a means of predicting the geographic pattern of emissions. Still, migration of the population to and from the area is a problem: Exposed persons who moved away (or who sought care far away) will not be counted; unexposed persons who moved into the area will be counted as exposed. While migration from the area should not bias effect estimates, providing it is independent of disease status, it will reduce statistical power by reducing the sample of exposed persons. Inclusion of persons migrating to the area, however, will cause exposure misclassification and thus weaken estimates of effect (43) (after the accident, clean-up operations and development of new safety procedures at the plant drew an influx of young workers (44)).

When considering potential confounding, we could account on an individual basis only for variations in age and sex. For urbanization and social class, we derived indices on a group basis from available census data. Since accident and routine emissions were correlated with urbanization and lower income, we tested the effectiveness of our adjustments for these factors using cervical cancer as a tracer condition. Cervical cancer has a strong inverse social class gradient (45) and is thought not to relate to radiation (21, 46). The regression analysis adjusting for social indicators eliminated a trend in the standardized incidence ratios for cervical cancer with accident exposure, yielding an association close to zero (OR = 1.0; 95 percent CI 0.7–1.5); the test thus suggests that the procedures based on aggregate data are reasonably sound, at least for categories with large sample sizes. Even aggregate data, however, were unavailable for important cancer determinants such as smoking.

In addition to confounding, extra-Poisson variation can arise in geographic analyses of cancer rates (47). Such overdispersion was not anticipated in our relatively small and homogeneous study area, nor was it indicated on a preliminary examination of the data. If such extravariability were present, its effect would be to widen the reported confidence intervals around the estimated odds ratios.

A final consideration is that this study shares with many of its kind a limitation of numbers, and hence of statistical power, so its results need to be read in context with those of other investigations.

Conclusions

In summary, the possibility that emissions from the Three Mile Island nuclear power plant could have contributed to the observed trends, in lung cancer particularly, must be weighed against 1) the lack of effects on the cancers believed to be most radiosensitive and the indeterminate effects on children; 2) the threat of confounding by factors unmeasured or inadequately controlled; 3) inconsistency within our own data between the findings for plant emissions and background gamma radiation; and 4) the low estimates of radiation exposure and the brief interval since exposure occurred. Pending a demonstration that very low-dose gamma radiation can act as a tumor promoter or the identification of another late-stage carcinogen in the effluent stream, an effect of plant emissions in producing the unusual patterns of lung cancer and non-Hodgkin's lymphoma appears unlikely, and alternative explanations need to be considered. The increased risk that we observed for childhood leukemia and other childhood cancers in relation to routine emissions is compatible with increases reported near some other nuclear installations, but confidence intervals are wide and, for leukemia, the numbers are small and the rates found in the Three Mile Island area low compared with national and regional data.

Studies currently being mounted around other nuclear plants in the United States and elsewhere can be expected to contribute further evidence about the effects of radiation exposures on cancer patterns in populations living nearby.

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