

A Re-Analysis of Cancer Incidence near the Three Mile Island Nuclear Plant

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Introduction

The well-known accident at the Three Mile Island nuclear power plant on the Susquehanna River near Harrisburg, Pennsylvania, USA, began on March 28, 1979. Industry and government reports concluded that the maximum gamma dose to a member of the general population was about 1 mSv and that the accident would not result in detectable health effects [2,11]. Despite these reassurances, many community members reported erythema, hair loss, vomiting, metallic taste, and deaths of pets and livestock at the time of the accident [7]. A local survey reported clusters of excess cancer deaths near TMI during 1979-84 [1]. These reports led the TMI Public Health Fund, an entity that had been created by a court order, to commission an epidemiological study of radiation doses and cancer incidence in the population living within ten miles of TMI.

Results of the epidemiological study were published in 1990 [5] and 1991 [6]. The first report showed some positive associations between estimated radiation doses and cancer incidence, however the authors concluded that these associations were not indicative of an effect of radiation [5]. The second study examined cancer incidence in relation to proximity of residence to TMI as a proxy for accident-induced psychological stress, which had previously been proposed as an explanation for reports of erythema, hair loss and other symptoms. The stress study did not produce conclusive findings [6].

We reanalyzed the TMI cancer incidence data for two reasons. First, some of the

symptoms reported at the time of the accident are consistent with acute responses to high dose radiation, and recently reported cytogenetic studies of persons reporting such symptoms suggest accident doses of 600-900 mGy [8,9]. Because the authors of the previous study did not consider the possibility of serious underestimation of doses, there was a logical inconsistency between the hypothesis they addressed, „that risks of specified cancers may have been raised by exposure to radiation emanating from the Three Mile Island nuclear power plant“ [5, p. 398], and the assumption that doses were too low to cause cancer. This assumption led the authors to conclude that associations which were demonstrated as evidence relevant to the study hypothesis did not support the hypothesis.

Second, the original study suffered from a number of methodological difficulties [5]. Only rare cancers with presumably shorter latency were considered as primary outcomes, greatly limiting statistical power, despite the fact that ionizing radiation is a general mutagen and immune suppressant that can induce most types of cancer [3,4]. It is important to note that increases in cancer incidence following radiation-induced immune system suppression begin months to a few years after exposure [4], well within the time frame of this study. The sensitivity of analyses of one primary outcome, childhood cancers, was reduced by failure to consider birth cohorts in the analysis [5]. Finally, although the investigators collected both pre- and post-accident cancer incidence data, they did not use

those data to correct estimates of accident effect for pre-accident geographic variation in cancer incidence.

Our re-analysis addresses these logical and methodological problems by (1) considering associations between cancer incidence and relative dose as an indication of the coincidence of cancer increases with plume travel, which allows the epidemiological analysis to be interpretable as an indicator of higher doses; (2) focusing on types of cancers that occur with sufficient frequency to increase the statistical power of the analysis; (3) using an analytical model to correct cancer-accident dose associations for pre-accident variation in cancer incidence; and (4) correcting for errors in data used for the previous analyses. We also interpret the results in the context of the strengths and limitations of the design of the study.

Materials and Methods

We obtained data from the TMI Public Health Fund. The previous investigators divided the 10-mile area around TMI into 69 study tracts, enumerated incident cancers in the area during 1975-1985, derived annual age and sex-specific population counts from US Census Bureau data, and estimated relative doses in each tract from dispersion models based on radiation monitors that remained on-scale for most of the accident, weather conditions and local topography [5]. We used the dose estimates from the original study, which were given on a ratio scale from 0 to 1665 units that were not assigned an absolute magnitude (such as Sv or Gy).

We corrected cancer counts for 1981-85 because duplicate records were discovered in the original files. The potential for bias due to a previously reported undercount in enumeration of cases in 1975 was eliminated by excluding that year from the pre-accident period, which was defined in our analyses as 1976 through March, 1979. In order to account for delay between radia-

tion exposure and cancer detection, post-accident periods were defined as 1981-85 and 1984-85. We analyzed data for lung cancer, leukemia, and all cancers combined.

Cancer incidence typically shows geographic variation, so pre-accident differences in cancer incidence between study tracts was to be expected. Higher cancer incidence prior to the accident in areas that were downwind from plumes released during the accident would bias estimates of the accident effect by confounding a pre-existing geographical effect with the accident. Our goal was to estimate the association between the accident dose values and cancer incidence following the accident, adjusting for any differences in cancer rates that existed prior to the accident. Thus, our results define the accident effect as the association between accident dose and post-accident incidence minus any gradient that existed prior to the accident. "Model 1" associations were adjusted for pre-accident incidence, age and sex, and "Model 2" associations were additionally adjusted for population density, median income and percentage of high school graduates. Associations were quantified using a log-linear Poisson regression model, and were expressed as the average log percent change in cancer incidence per unit change in dose. Standard errors of the estimates, given in parentheses after the estimate, can be multiplied by 1.645 to obtain 90% confidence limits around the estimates. To evaluate sensitivity of results to the model form, associations were also quantified using an additive relative risk regression model.

Results

Cancer incidence was higher following the accident in those study tracts estimated to have been more exposed to accident plumes, adjusting for pre-accident variation in cancer incidence. During 1981-85, inci-

dence of all cancers combined increased, on average, 0.020% (0.012) per dose unit with adjustment for age, sex, and pre-accident cancer rates (Model 1). This value increased to 0.034% (0.013) with adjustment for socioeconomic factors (Model 2). Associations were 0.023% (0.014) under Model 1 and 0.035% (0.015) under Model 2 for cancers occurring in 1984-85 (a five-year latency). During 1981-85, in study tracts with estimated doses of 800 units or greater, the ratio of observed cancer cases (N=112) to the number expected based on Model 2 was 1.49, while in study tracts estimated to have received no exposure, the ratio of observed (N=62) to expected cases was 0.67.

Stronger associations were observed for lung cancer incidence rates. When the 69 study tracts were divided into 9 dose groups ranging from zero to 800 dose units and above, the ratio of observed to expected (based on Model 1) lung cancers increased in each successive category, from 0.43 to 2.34, representing an average increase of 0.082% (0.032) per dose unit. This estimate increased to 0.103% (0.035) per dose unit in Model 2. During 1984-85 associations were of similar magnitude.

For leukemia during 1981-85, average increases were 0.116% (0.067) per dose unit during 1981-85 and 0.133% (0.077) per unit during 1984-85 (Model 1). These values were somewhat larger with additional adjustments in Model 2. Depending upon adjustment using Model 1 or Model 2, the ratio of observed to expected leukemias increased from a low of about 0.5 at zero dose to a high of about 4.0 in the highest dose group during 1981-85, and from about 0.4 to about 7.0 during 1984-85.

We also evaluated these associations using an additive relative risk regression model. Findings were similar to those reported above for the multiplicative model.

Discussion

Increases in cancer incidence after the 1979 accident at TMI were greater in areas estimated to have been more exposed to accident plumes, adjusting for pre-accident geographic variation in cancer, age and sex. These associations were stronger, in particular for all cancers and leukemia, with additional adjustment for socioeconomic factors. The magnitude of association was greatest for leukemia, intermediate for lung cancer, and smallest for all cancers combined. Associations tended to be slightly larger at the end of the post-accident measurement period (1984-85) than for the entire period (1981-85).

Our findings differ from the previous report [5] in certain respects. After excluding data for the year 1975, when there was an undercount of cancer cases, pre-accident lung cancer incidence was not higher in study tracts that were more exposed to accident radiation, as was reported previously [5]. This is an important finding because the association reported previously led the authors to speculate that the findings might reflect confounding by smoking [5]. We found no evidence to support this suggestion.

Findings for leukemia incidence among the total population of all ages were not reported previously. This outcome showed the strongest association with radiation dose estimates of the three cancer groups chosen for our analyses.

The previous investigators cited estimates of low doses in arguing that positive associations between dose estimates and cancer incidence were not suggestive of an effect of radiation from the accident [5,6]. Low dose estimates were given by government and industry measurements of radioactivity in air, soil, animals and food [2,11]. However, the court order governing the calculation of the doses used in the epidemiological study prohibited „upper limit or worst case estimates of releases of ra-

dioactivity or population doses ... [unless] such estimates would lead to a mathematical projection of less than 0.01 health effects" [10]. The order further stipulated that „a technical analyst ... designated by counsel for the Pools [nuclear industry insurers] concur on the nature and scope of the [dosimetry] projects“ [10]. These conditions raise doubts about whether the assumption that accident doses were low followed from the physical evidence or from the court order.

By adjusting for pre-accident geographic variation in cancer incidence, our estimates control for unmeasured confounders that are stable over time. However, it is possible that confounding could occur if there was a coincidence of temporal changes in the confounding factor with plume travel. For example, our method would not be able to control confounding if smokers differentially moved into downwind areas after the accident, or if non-smokers differentially moved into upwind areas after the accident. However, we have no reason to believe that such differential dose-associated movements would take place over a relatively short time period within the 10-mile area.

We do, however, have reasons to question the validity of the dose classification. Plume travel estimates were based on limited environmental and meteorological measurements. Dose values were assigned for areas up to several kilometers across, and there could have been substantial within-tract variation in dose under conditions conducive to low dispersion of narrow plumes. Population mobility after the accident was not taken into account, as cases were assigned to study tracts based on residence at date of diagnosis, not date of exposure. To the extent that these problems lead to mixing of exposure groups, they would produce an underestimation of the magnitude of the association of accident doses with cancer incidence.

Conclusion

Our re-analysis of cancer incidence around the TMI nuclear power plant is consistent with the hypothesis that radiation from the accident led to an increase in cancer in areas that were in the pathway of radioactive plumes. This would not be expected to occur over a short period of time in the general population unless doses were far higher than estimated by industry and government authorities. Rather, findings support the allegation that people in the area who reported erythema, hair loss, vomiting, and pet deaths at the time of the accident were not suffering from emotional stress, but rather were exposed to high level radiation.

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