Radiation Protection Dosimetry (2005) Vol. 113, No. 2, pp. 214–217
Advance Access publication 18 January 2005
doi:10.1093/rpd/nch445

SCIENTIFIC NOTE

THREE MILE ISLAND EPIDEMIOLOGIC RADIATION DOSE ASSESSMENT REVISITED: 25 YEARS AFTER THE ACCIDENT

R. William Field
Department of Occupational and Environmental Health, Department of Epidemiology,
College of Public Health, University of Iowa, Iowa City, IA 52242

Received September 6 2004, amended November 24 2004, accepted December 5 2004

Over the past 25 years, public health concerns following the Three Mile Island (TMI) accident prompted several epidemiologic investigations in the vicinity of TMI. One of these studies is ongoing. This commentary suggests that the major source of radiation exposure to the population has been ignored as a potential confounding factor or effect modifying factor in previous and ongoing TMI epidemiologic studies that explore whether or not TMI accidental plant radiation releases caused an increase in lung cancer in the community around TMI. The commentary also documents the observation that the counties around TMI have the highest regional radon potential in the United States and concludes that radon progeny exposure should be included as part of the overall radiation dose assessment in future studies of radiation-induced lung cancer resulting from the TMI accident.

INTRODUCTION

Environmental epidemiologic investigations seek associations between involuntary environmental toxicant exposures and their potential adverse health effects, thus illuminating cause and effect relationships. The questions examined by environmental epidemiology are frequently of great importance; unfortunately the answers provided by many of the studies are subject to large uncertainty. Observing the generally low relative risks resulting from most environmental exposures are often hindered by both the lack of individual level data for the population at risk and inaccurate or incomplete assessment of past environmental exposures. In fact, non-differential misclassification and measurement error of exposure generally bias results toward the null in estimates of relationships between exposure and disease[1,2] even when a significant dose-response relationship exists. Another challenging problem in environmental epidemiology is the proper control of confounding variables. Environmental epidemiologic studies are frequently performed in emotionally charged situations in which the public is anxious, angry, fearful or distrustful of an industry or the government and in cases where groups of citizens are in adversarial litigious situations as a result of their perceived exposure.

Public health concerns following the 1979 Three Mile Island (TMI) accident prompted several teams of researchers to perform epidemiologic investigations in the vicinity of TMI. Within this context, one wonders whether or not the public's perception that radon progeny exposures are of low risk may have been a factor in the failure of previous TMI related studies to adjust for this potentially important confounding factor when assessing the impact that the accidental plant releases may have had on lung cancer incidence in the TMI area.

Corresponding author: bill-field@uiowa.edu

RISK PERCEPTION AND ENVIRONMENTAL EPIDEMIOLOGIC STUDY DESIGNS

Using TMI as a backdrop, Wing[3] described the interactions between science, society and litigation and asserted that scientific explanations are shaped by social concepts, norms and preconceptions. I find myself in agreement that scientific explanations and indeed environmental epidemiologic study designs are shaped partly by social factors and perhaps even by a larger extent by the availability of existing data to assess the retrospective exposure. The following comments are provided to illuminate a factor, which has been generally neglected, probably because of lack of readily available data and possibly in part by social apathy, in the epidemiologic research following the TMI accident. It is well documented that prolonged exposure to radon decay products (radon) causes lung cancer[4–7]. In fact, the US Environmental Protection Agency (EPA) recently modified and extended the approach used by the National Research Council[4] to assess the risk posed by residential radon exposure. Based on their re-analysis, the EPA estimates that ~21,000 radon-related lung cancer deaths occur each year in the United States.[8]

Radon has no sensory reminders to repetitively stimulate us think about it. It is not a dread hazard[9]. Lung cancer caused by radon progeny exposure is not distinguishable histologically from lung cancer due to other causes. The preponderance

of exposure to radon decay products occurs indoors at home and at work and is generally not caused by any industry, so there are no 'villains' to blame for its presence. These factors and others[10,11] reduce the risk perceived by the public and, in turn, researchers and other scientists[12] are not publicly reminded by social outrage to include it as part of radiation risk assessment.

TMI-RELATED EPIDEMIOLOGIC STUDIES

Hatch *et al.*[13] investigated the incidence of cancer for individuals in the 10 mile radius around TMI in regard to both estimated TMI-accident-related radiation exposures and possible radiation exposures resulting from routine operations of the plant. The study area was divided into 69 study tracts and the researchers relied on mathematical models to predict exposure patterns for both accident and routine emissions. Individual risk information was not collected. The researchers found elevated risks for lung cancer for both the routine and estimated accident doses. The researchers further noted that, 'one or more lung cancer risk factors are operating to produce an exposure pattern very similar to the pathway for the radioactive plume'. In concluding that the overall results 'did not provide convincing evidence' that radiation releases from TMI influenced cancer risk during the limited period of follow-up, the authors relied, partly, on a median estimated accident-related dose for the study tracts equal to ~0.4 mSv (40 mrem) with an average estimated individual dose of 0.1 mSv (10 mrem). Background gamma radiation doses, excluding radon, were estimated to range from 0.5 to 1 mSv yr^{-1} (50–100 mrem yr^{-1}) and doses to individuals from routine TMI operations in the area were estimated to be about 0.01 mSv yr^{-1} (1 mrem yr^{-1}). In a reanalysis of the Hatch study, Wing *et al.*[14] used the same dose information provided by Hatch *et al.*[13], but made no use of absolute dose estimates, relying only on relative dose units. Their estimated relative risk estimates for lung cancer mortality were slightly higher than the risk estimates noted by Hatch *et al.*[13].

Talbott and colleagues[15] recently updated their previous findings for a long-term follow-up (1979–1998) cohort mortality study in the vicinity of the TMI area. The researchers relied on whole body gamma dose estimates, for Xenon-133, 133 m, 135, 135 m and Krypton-88 limited to the ten days following the accident, which were modelled by Gur *et al.*[16], to assign likely and maximum gamma dose estimates for the population within a 5-mile radius of TMI. Using a 1976 airborne survey, Talbott *et al.*[15] also assigned members of the TMI cohort, by zip code of residence, to quartiles of 'natural background radiation dose'. The researchers reported that over 75% of the TMI cohort within 5 miles of the plant resided in areas with gamma levels <8.8 μR hr^{-1}. Relative risk estimates showed that neither the maximum gamma dose nor the estimated gamma dose was a significant predictor of lung cancer after adjusting for 'known' confounders.

The use of aggregate data to represent the total post accident-derived radiation exposure history for individuals in the vicinity of TMI is a major limitation of TMI-related epidemiologic investigations. Unfortunately, the recent study by Talbott *et al.*[15] failed to consider the major source of 'natural background radiation exposure' impacting lung cancer risk estimates for the population under study, especially in light of a previously published mortality study[17] that acknowledged, 'higher mean levels of indoor radon exposure have been documented in the TMI geographic area'. Overall, the TMI-related epidemiologic studies have neglected the importance of radon exposure in their adjustments for confounding.

RADON CONCENTRATIONS IN THE VICINITY OF TMI

Assuming that the estimates used by Talbott and colleagues[15] are correct, the natural gamma background dose of <1 mSv yr^{-1} (100 mrem yr^{-1}) and the average estimated individual whole body gamma dose of 0.09 mSv (9 mrem) for the five mile zone around TMI from noble gases (excluding radioiodines) released from TMI, pale in comparison to the average dose equivalent from radon progeny exposure to the public in the vicinity of TMI. As part of the US Environmental Protection Agency/State Radon Cooperative Program, Alexander *et al.*[18] published a summary of screening (short-term) radon measurements that included 55,000 randomly selected homes in 38 states. The survey identified geographic regions within the states with elevated screening levels of radon.

Screening radon measurements are short-term radon measurements, usually performed in the lowest area of the home. They are used to identify areas with high radon potential and should not be considered directly representative of personal radon exposure. Oddly coincidental, the highest regional screening radon concentrations noted for the 38 states were found in the Pennsylvania counties surrounding TMI, including Cumberland, Dauphin, Lancaster, Lebanon, Perry and York Counties (Figure 1). Screening radon measurements in these counties averaged 659 Bq m^{-3} (17.8 pCi L^{-1}). The potential confounding effect of this source of radiation exposure is significant when one considers that a yearly average radon concentration of 150 Bq m^{-3} (4 pCi L^{-1}) imparts an estimated average annual

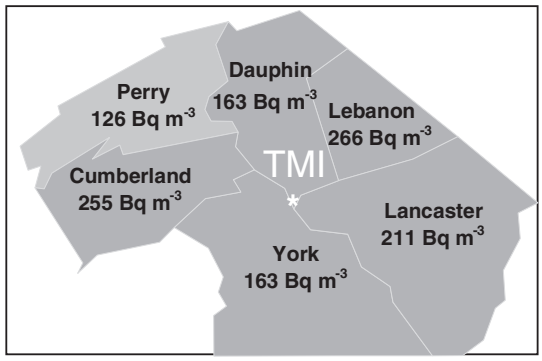

Figure 1. The highest regional screening radon measurements, 666 Bq m^{-3} (18 pCi L^{-1}), of the 38 states surveyed by the US EPA are found in the six counties surrounding TMI. Screening radon measurements are short-term radon measurements, usually performed in the lowest liveable area of the home, used to identify geographic areas with high radon source potential. The figure displays the predicted county average radon concentrations in the main living areas, generally not the basement, of homes for the six counties surrounding TMI (adapted from The High Radon Project http://eetd.lbl.gov/IEP/high-radon/hr.html). Only Perry County is below the US EPA's action level of 150 Bq m^{-3} (4 pCi L^{-1}) for radon.

dose of 200 mSv (20,000 mrem) to the target cells in the bronchial epithelium[8]. Figure 1 depicts the predicted county average radon concentrations in the living areas of homes for the six counties surrounding TMI[19]. Particularly high radon concentrations are known to occur in the confined physiographic region known as the Great Valley Section that extends north of TMI[20]. However, because of the high geologic heterogeneity of radon source material within the 5 mile radius of TMI, it is impossible to predict how the radon concentrations in this area affect risk estimates without a more detailed survey involving residential radon.

CONCLUSIONS

The challenge of adequately reconstructing past radiation exposure makes it very questionable whether or not the various TMI-related epidemiologic studies had sufficient power and scientific rigour to make any claims regarding whether or not the radioactivity released during the TMI accident had a statistically significant impact on the lung cancer mortality experience of this population through 1998. Perhaps, dose assessment may be enhanced as technology improves through the continued development of biological markers or the *in vivo* use of teeth to measure the electronic spin resonance as a surrogate of dose at the level of the individual[21]. Consideration should also be given to performing case-cohort analyses with appropriate matching and enhanced retrospective dose assessment methods that include currently available retrospective assessment of radon progeny exposure[22] and detailed information on lifestyle factors such as smoking[23]. Further thought should also be given to how this population, with its chronically elevated alpha particle exposure to the lung from exposure to radon decay products, may differ from other populations with regard to radiosensitivity or radioadaptation. For example, does the population around TMI have more radon progeny dose-related chromosome aberrations and genomic instability, which would make it more susceptible to cancer induction from noble gas exposure? Obviously, scientific inquiry is never finished, but may proceed at a quicker pace with mutual cooperation.

In summary, the lack of control of confounding by radon decay products in TMI-related epidemiologic studies suggests that the environmental epidemiologic study designs are shaped by social factors. Scientists need to strive to transform their overall scientific objectives, in this case understanding the possible adverse health effects following the TMI accident, from a narrow personal pursuit to a more cooperative vision. While this transformation is difficult under the litigious conditions, in the years following the TMI accident, described by Wing [3] the transformation may be manageable outside the legal arena with mutual cooperation between scientists. Even though there is little outrage or anger regarding radon exposure around TMI, radon progeny exposure should be included as part of the overall dose assessment in studies of radiation-induced lung cancer from the TMI accident, especially since radon progeny exposure in the TMI area produces much higher radiation doses to the lung than the officially reported offsite TMI accident-related releases.

ACKNOWLEDGEMENTS

I thank Drs Charles Lynch, Phil Price, Daniel Strom and Conrad Weiffenbach for their helpful comments on a previous version of this manuscript.

REFERENCES

1. Field, R. W., Smith, B. J., Steck, D. J. and Lynch, C. F. *Residential radon exposure and lung cancer: variation in risk estimates using alternative exposure scenarios*. J. Expo. Anal. Environ. Epidemiol. **12**(3), 197–203 (2002).
2. Kelsey, J. L., Thompson, W. D. and Evans, A. S. *Methods in Observational Epidemiology, Monographs in Epidemiology and Biostatistics*. Vol. 10 (New York: Oxford University Press) (1986).
3. Wing, S. *Objectivity and ethics in environmental health science*. Environ. Health Perspect., **111**, 1809–1818 (2003).

4. National Research Council. *Health effects of exposure to radon, BEIR VI*. Committee on Health Risks of Exposure to Radon (BEIR VI), Board on Radiation Effects Research, Commission on Life Sciences, Washington, DC (National Academy Press) (1998).
5. Field, R. W., Steck, D. J., Smith B. J., Brus, C. P., Fisher, E. L., Neuberger, J. S., Platz, C. E., Robinson, R. A., Woolson, R. F. and Lynch, C. F. *Residential radon gas exposure and lung cancer: the Iowa radon lung cancer study*. Am. J. Epidemiol. **151**(11), 1091–1102 (2000).
6. Darby, S. C. and Hill, D. C. *Health effects of residential radon: a European perspective at the end of 2002*. Radiat. Prot. Dosim. **104**(4), 321–329 (2003).
7. Krewski, D. et al. *Risk of lung cancer in North America associated with residential radon*. Epidemiology (in press).
8. United States Environmental Protein Agency. *EPA assessment of risk's from radon in homes*. United States Environmental Protection Agency, Air and Radiation (6608J), EPA 402-R-03 (2003).
9. Slovic, P. *Perception of risk*. Science **236**(4799), 280–285 (1987).
10. Field, R. W., Kross, B. C. and Vust, L. J. *Radon testing behavior in a sample of individuals with high home radon screening measurements*. Risk Anal. **13**(4), 441–447 (1993).
11. Weinstein, N. D., Klotz, M. L. and Sandman, P. M. *Optimistic biases in public perceptions of the risk from radon*. Am. J. Public Health **78**(7), 796–800 (1988).
12. Likhtarov, I. A. and Kovgan, L. N. *The need for changes in ICRP policy: some examples based on the Chernobyl experience in Ukraine*. J. Radiol. Prot. **22**(2), 163–173 (2003).
13. Hatch, M. C., Wallenstein, S., Beyea, J., Nieves, J. W. and Susser, M. *Cancer rates after the Three Mile Island nuclear accident and proximity of residence to the plant*. Am. J. Public Health **81**(6), 719–724 (1991).
14. Wing. S., Richardson, D., Armstrong, D. and Crawford-Brown, D. *A reevaluation of cancer incidence near the Three Mile Island nuclear plant: the collision of evidence and assumptions*. Environ. Health Perspect. **105**, 52–57 (1997).
15. Talbott, E., Youk, A., McHugh-Pemu, K. and Zborowski, J. *Long-term follow-up of the residents of the Three Mile Island accident area: 1979–1998*. Environ. Health Perspect. **111**, 341–348 (2003).
16. Gur, D., Good, W. F., Tokuhata, G. K., Goldhaber, M. K., Rosen, J. C., Roa, G. R., Herron, J. M., Miller, D. M. and Hollis, R. S. *Radiation dose assignment of individuals residing near the Three Mile Island Nuclear Station*. Proc. PA Acad. Sci. **57**, 99–102 (1983).
17. Talbott, E. O., Youk, A. O., McHugh, K. P., Shire, J. D., Zhang, A., Murphy, B. P. and Engberg, R. A. *Mortality among the residents of the Three Mile Island accident area: 1979–1992*. Environ. Health Perspect. **108**(6), 545–552 (2000).
18. Alexander, B., Rodman, N., White, S.B. and Phillips, J. *Areas of the United States with elevated screening levels of ^{222}Rn*. Health Phys. **66**(1), 50–54 (1994).
19. Price. P. N. *Predictions and maps of county mean indoor radon concentrations in the Mid-Atlantic States*. Health Phys. **72**(6), 893–906 (1997).
20. United States Environmental Protection Agency. *EPA's map of the radon zones: Pennsylvania*. United States Environmental Protection Agency, Air and Radiation (6604J), United States Geological Survey, 402-R-93-058, (1993).
21. International Commission on Radiation Units and Measurements. *Retrospective assessment of exposures to ionising radiation*. ICRU Report 68, J. ICRU **2**(2) (2002).
22. Steck, D. J., Alavanja, M. C., Field, R. W., Parkhurst, M. A., Bates, D. J. and Mahaffey, J. A. *^{210}Po implanted in glass surfaces by long term exposure to indoor radon*. Health Phys. **83**(2), 261–271 (2002).
23. Puskin, J. S. *Smoking as a confounder in ecologic correlations of cancer mortality rates with average county radon levels*. Health Phys. **84**(4), 526–532 (2003).