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DOSIMETRIC CHALLENGES FOR RESIDENTIAL RADON EPIDEMIOLOGY

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Radon concentration alone may not be an adequate surrogate to measure for lung cancer risk in all residential radon epidemiologic lung cancer studies. The dose delivered to the lungs per unit radon exposure can vary significantly with exposure conditions. These dose-effectiveness variations can be comparable to spatial and temporal factor variations in many situations. New technologies that use surface-deposited and implanted radon progeny activities make more accurate dose estimates available for future epidemiologic studies.

The lung cancer risk associated with radon exposure is believed to be proportional to the radiation energy delivered to sensitive lung tissues over extended periods of time (NRC, 1999; UNSCEAR, 2000). Since this energy is difficult to measure directly, current risk estimates are usually based on a loosely correlated surrogate like indoor radon gas concentration or airborne radon progeny activity. Recent work has shown that both of these surrogates introduce significant risk estimate uncertainties from the variation of the dose-effectiveness per unit surrogate under different exposure conditions (Porstendorfer & Reineking, 1999; Porstendorfer, 2001; James et al., 2004). It is widely recognized that limited spatial and temporal sampling of the surrogate can adversely affect the accuracy of exposure estimates. The effects of incomplete sampling and missing radon exposure data on epidemiologic studies have been extensively studied (Lubin et al., 1990, 1995; Baverstam & Swedjemark, 1991; Field et al., 1996, 2002; Darby et al., 1998; Reeves et al., 1998; Gerken et al., 2000; Steck, 2002). These effects are reviewed here only to compare their contributions to risk uncertainty with the effects that surrogate dose variations have on epidemiologic analysis. Dose-effectiveness variations have received little attention to date in radon epidemiology, but emerging measurement techniques have

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reopened the possibility that a better dose surrogate is available for radon-related risk assessment.

DOSE, SURROGATES, AND CONVERSION FACTORS

The primary dose delivered to the lungs depends on the inhaled radon progeny that are able to penetrate the airway and accumulate on the sensitive tissues. The concentrations of lung-deposited radon progeny depend on the physical and behavioral characteristics of the individual being exposed and the size distribution of the radon progeny available to that individual. Nanosize progeny clusters are roughly an order of magnitude more effective in delivering dose to the lungs than aerosol-attached progeny (NRC, 1999; Porstendorfer, 2001; James et al., 2004). Nanosized progeny clusters are more mobile and tend to deposit more readily on surfaces, leading to a loss of breathable dose. Thus, accurate dose rate estimates require measurements of the progeny activity size distribution, not just radon or total radon progeny concentrations. Even simple size distribution measurements that separate the airborne progeny into unattached (cluster mode <5 nm) and all other sizes will improve the dose rate estimate. Better estimates are obtained if three modes—cluster, nucleation (5–50 nm), and accumulation (100–500 nm)—are measured.

While recognizing that individual variation in breathing rate, breathing style, or radiation sensitivity may cause variations in the risk by as much as a factor of 2 (NRC, 1999), we do not incorporate those issues quantitatively in the present analysis. Rather, we focus on dose surrogates that might be measurable with field-grade detectors in an epidemiologic study of indoor radon exposure. In addition, we exclude ^{220}Rn (thoron) progeny from the discussion because the dose contribution from thoron is believed to be much less than the ^{222}Rn (radon) progeny dose in most living spaces (UNSCEAR, 2000).

SURROGATES

Past studies examining the relationship between residential radon and lung cancer have not measured dose for two main reasons: (1) Dose measurements are difficult and (2) only recently have dosimetric models and measurement techniques become sophisticated enough to identify the proper variables to be measured and how to interpret those measurements.

Early epidemiologic studies of miners used the airborne radon progeny potential alpha energy concentration (PAEC) exposure, in units of working level month (WLM), as the risk metric. In some mines, the airborne radon progeny were measured directly; in others, the radon was monitored and radon progeny grab samples helped establish an equilibrium ratio (F) of the actual progeny PAEC to the maximum that could be created by the radon concentration. In other mines, aerosol concentration and ventilation rates were used to estimate F (NRC, 1999).

Although radon progeny, rather than radon, deliver the majority of the energy to the lung, residential radon epidemiologic studies adopted contemporary

radon concentrations as the risk metric. Radon concentrations are easier to measure than progeny, and it was believed that radon correlated well enough with dose to be a good risk surrogate. Sufficient sampling has been done to characterize radon's spatial and temporal variation in some regions. Table 1 summarizes a sample of the spatial and temporal variation in central North America, where three of the epidemiologic studies (Alavanja et al., 1999; Field et al., 2000; Létourneau et al., 1994) included in the North America pooled analysis were performed (Krewski et al., 2005). The largest radon concentration differences in this region occur when a subject moves from outdoors to indoors, from the basement to the first floor, and from house to house (see Table 1). The effects of these differences on the risk metric under different exposure scenarios were significant for the Iowa Radon Lung Cancer Study (IRLCS) (Field et al., 2000). For example, ignoring the radon exposure outside the home reduced the IRLCS radon-exposure lung cancer trend (Field et al., 2002). The dose delivered per unit radon exposure in homes shows variation of a similar magnitude to the radon variation (Wasiolek et al., 1992; Hopke et al., 1995; Porstendorfer, 2001). If one assumes that, to a large degree, dose variations follow radon exposure variations, then good estimates for dose may be obtained from comprehensive spatial and temporal radon gas measurements. However, better dose assessments would require some measure of the airborne progeny, especially the activity size distribution, to calculate the dose rate to radon concentration ratio under the varying exposure conditions. The conditions that affect this ratio include dose reduction through attachment to aerosols, deposition on surfaces, and ventilation (when indoors), and dose enhancement in tight, clean, still rooms.

TABLE 1. Temporal and Spatial Variations of Radon Observed[a] in Central North America

Variation	COV[b] (%)	Mean (deviation) (Bq m^{-3})	Region
Temporal: year to year	25		Minnesota
Spatial: within a house			
Room to room on a floor	10; 11		Iowa; Minnesota
First floor to basement ratio[c]		0.55 (0.25); 0.64 (0.16)	Iowa; Minnesota
First to second floor ratio[c]		1.02 (0.24)	Iowa
House to house[d]	120	90 (2.2)	Iowa
(Mobility weighted Rn in living spaces; 7% basement residency)	85	100 (1.8)	Minnesota
	120	44 (2.2)	Missouri
	150	120 (2.5)	Winnipeg
Workplaces[d]	170	70 (2.7)	Minnesota
Outdoors[d]	40	29 (1.4)	Iowa
	80	19 (1.8)	Minnesota

[a]Alavanja et al. (1999), Field et al. (1998, 2000), Fisher et al. (1998), Létourneau et al. (1994), Steck (1992), and Steck et al. (1999).

[b]Coefficient of variation, expressed as a percentage.

[c]Normal distribution: mean value (standard deviation).

[d]Lognormal distribution: geometric mean value (geometric standard deviation).

CONVERSION FACTORS

We use the term *dose-effectiveness coefficient* (DEC) for the ratio of the annual dose rate (mSv/yr) to the annual average radon concentration (Bq m^{-3}). This conversion factor is more useful for discussions of residential radon studies than the dose conversion factor (DCF), the ratio of the cumulative dose (mSv) to the radon progeny exposure (WLM), which is used in many risk assessments. Using the DCF makes sense for situations where the airborne progeny are measured rather than radon concentration. Unfortunately, most of the dose-effectiveness experiments or residential surveys report the DCF rather than the DEC, since one needs to measure the activity size distribution of the progeny to calculate the dose.

To study dose variations, we incorporated recent microdosimetric developments in a fate and transport model that includes distributions for radon, aerosol concentrations, ventilation rates, and surface deposition rates (Steck & Field, 1999a; Steck, 2002). The model generates a bimodal (cluster and attached modes) progeny size distribution within a room to calculate the dose using models of Porstendorfer (Porstendorfer & Reineking, 1999; Porstendorfer, 2001) and James (James et al., 2004). The Porstendorfer model assigns more effectiveness to the cluster mode than does the James model. Together, the models provide a median and range for the dose estimates under varying environmental situations. We have found the model to be reasonably reliable when compared to measurements in laboratory and home environments. In the laboratory, exposures took place in a room the size of a small bedroom, where radon concentrations around 1 kBq m^{-3} could be maintained while various aerosol sources (tobacco smoke, air fresheners, candles) and sinks (HEPA and ionic air cleaners, fans) were used to alter the activity size distribution. We measured cluster and attached activity concentrations of each progeny using a spectroscopic sampler SARAD EQF3210 (EQF), which was set at a 2-h sampling cycle. Continuous radon concentrations were also measured by a Durridge RAD-7 as well as the EQF. Figure 1 shows that the DEC ranges from roughly 50 to several hundred mSv/y/kBq/m^3 or roughly a factor of 2 about the mean. The range of variation for the DCF in these tests was somewhat smaller than a factor of 2. Hopke reported a coefficient of variation of roughly 50% in the DEC in seven houses studied in northeastern North America (Hopke et al., 1995a). Porstendorfer reports a range of about 2 in DCF for German houses (Porstendorfer, 2001). Yu reports a difference of 30% between urban and marine sited houses in Hong Kong (Yu et al., 2001).

Dose-effectiveness, as well as radon concentration, varies for exposures outside the home. While outdoor concentrations are usually lower than indoor concentrations, the DEC outdoors is generally higher than indoors. If a person spends considerable time outdoors in work or play, the outdoor dose contribution may be significant (Wasiolek & James, 1995; Steck et al., 1999). Workplace DECs also vary considerably, as the spaces occupied range from homelike structures to large buildings with active air movement, aerosols, and filtering (Porstendorfer, 2001).

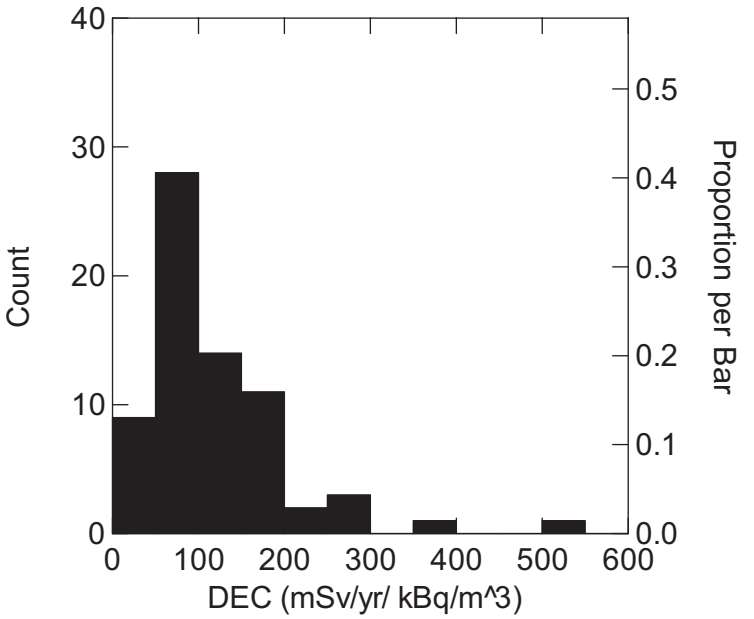

FIGURE 1. Dose-effectiveness coefficients (DEC) measured with varying conditions in a laboratory room, two houses, and a workplace.

We used a Monte Carlo approach to simulate true cumulative dose based on our residential model and individual characteristics (age, mobility, occupancy) of the IRLCS participants and spaces that they occupied. We also used realistic distributions for the major factors that affect dose rates (radon concentration, aerosol attachment, surface deposition, and ventilation rates) as well as an individual's age, smoking, and exposure variation from mobility. In this simulation, dominated by smoking households, the DEC had a coefficient of variation of 30%. In a more representative sample of Iowa homes, where roughly 30% would have an active smoker at home, the DEC variation increased to 50%.

EFFECTS OF SURROGATE CHOICE ON EPIDEMIOLOGIC STUDIES

We extended our Monte Carlo analysis to track an individual's dose and exposure over a lifetime. We also assigned a disease status to explore the effects on different measurement protocols on the dose to surrogate correlation for both cases and controls (Steck, 2000, 2002). We wanted to increase our understanding of possible reasons why the Iowa study (IRLCS) observed a radon–lung cancer association and two studies in nearby Missouri and Winnipeg did not. Those studies had protocols and participants that were somewhat similar to the IRLCS. However, the Iowa study used a more

comprehensive radon measurement protocol to cover every occupied room within the home and the local average values for outdoors and in workplaces. In addition, IRLCS subjects had been living in their current home for 20 yr or more. We also wanted to explore the performance of new surrogates like direct dose rate estimates based on continuous airborne activity size measurements (Porstendorfer, 2001) and indirect dose rate measurements based on ^{210}Po implanted in glass (Steck & Field, 1999a, 1999b). The simulation allowed a comparison of the correlation and misclassification of the surrogates with the true lifetime cumulative dose (Figure 2). The Iowa study's a priori surrogate (COMPLETE_Rn) showed a better correlation and a smaller shift than the Missouri-style surrogate (KITCH_Bed) and a Winnipeg-style surrogate (BASE_1ST Rn). The simulation also suggests that radon progeny dosimeters would make a better surrogate than any of the radon measures. A contemporary dose rate measurement with a 25% instrumental uncertainty performed the best (DOSERATE in Figure 2). Cumulative dose estimates from ^{210}Po implanted in glass surfaces (GLASSDOSE) was the next best surrogate. Figure 2b shows that even in Iowa, where there is a wide range of indoor radon exposures, misclassification due to shifts in exposure categories could occur quite frequently for standard surrogates. In most other North American studies, where the exposure range was compressed, misclassification errors would be more pronounced.

In an analysis of the actual data from the IRLCS, the odds ratio trend decreased for less comprehensive radon exposure measures in a pattern similar to the loss of correlation of the surrogates in the simulation. (Field et al., 2002).

FUTURE DIRECTIONS FOR MEASURABLE DOSE SURROGATES

Based on the simulation study results, the airborne dose rate available in the frequently occupied living spaces is the most logical choice for the best dose surrogate. However, direct dose rate measurements are neither easy nor cost-effective for mass surveys given existing technology. Since issues of spatial and temporal variation are important for the cumulative dose as well as radon exposure, a technique is needed that can be used in all frequently occupied spaces and accurately estimate the cumulative dose. A new integrating radon and radon progeny device, based on electrets, that measures the contemporary bimodal values for the PAEC can give a snapshot of the contemporary dose rate over a few days. However, this device could not reconstruct the past dose rates, especially in critical situations where the environment has been altered, for example, by the cessation of smoking.

For both contemporary and retrospective reconstruction, a technique based on surface-deposited and implanted progeny holds the most promise for epidemiologic studies. The simulation showed that the dose rate and interpreted glass dose estimates correlated well with the cumulative dose. This technique uses measurements of the contemporary radon concentration and

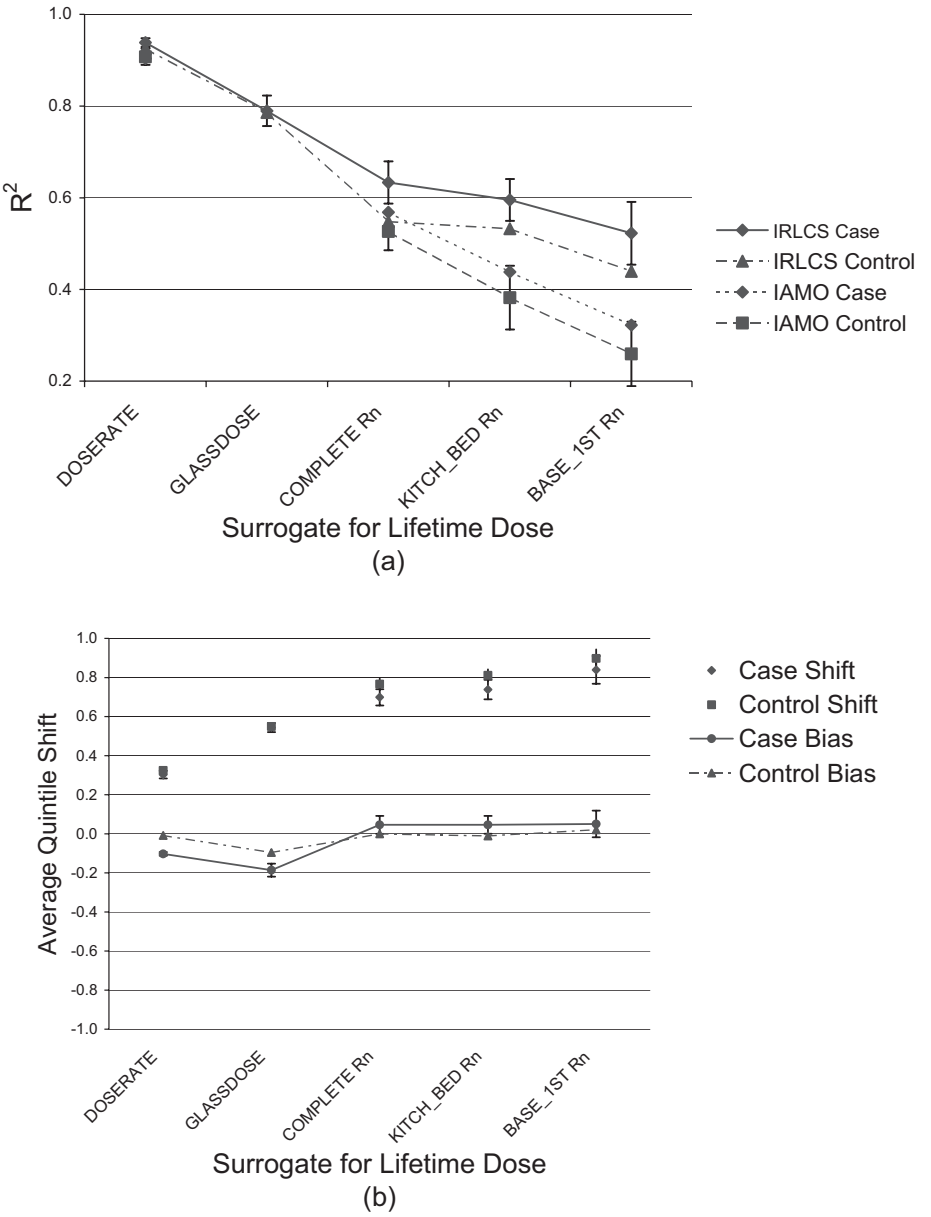

FIGURE 2. (a) Correlation coefficients (R^2) of the true cumulative lifetime dose with two potential surrogates (DOSERATE and GLASSDOSE) and three actual surrogates used in epidemiologic studies in central North America; Iowa (COMPLETE Rn), Missouri (KITCH_BED Rn), Winnipeg (BASE_1STRn). The population characteristics are based on the IRLCS participants. Two residency inclusion criterion for the current residence were analyzed: 20 yr (IRLCS) and 5 yr (IAMO). (b). Shift and bias between the surrogate quintile and cumulative dose quintile per individual. Shift is the average of the number of quintiles moved per individual, and bias is the arithmetic average of the quintile change per individual.

surface deposited ^{218}Po and ^{214}Po to estimate the current dose rate from a fate and transport model (Steck & Field, 1999a, 1999b). The activity measurements are used to characterize the maximum available dose rate, the dose rate lost to surface deposition, and the dose rate modified by aerosol attachment. This technique has been validated in laboratory studies and is now undergoing field testing.

A simpler technique that uses implanted ^{210}Po activity alone has been shown to correlate well with contemporary radon concentrations in large samples of North American homes (Steck & Field, 1999a; Mahaffey et al., 1999). In a smaller sample of homes, implanted ^{210}Po activity was as accurate a predictor of long-term average radon as year-long radon measurements (Steck et al., 2002). Radon exposure estimates based on implanted ^{210}Po alone have been used as a risk metric in two reported residential studies that observed an association with lung cancer (Alavanja et al., 1999; Lagarde et al., 2002). It is interesting to note that the central estimate for the excess odds ratio for these two studies based on implanted polonium was much higher than the radon gas-based estimate.

A more advanced technique is to combine measurements of airborne ^{222}Rn and surface ^{218}Po, ^{214}Po, and ^{210}Po to give contemporary and cumulative dose rate estimates. The contemporary measurements would provide the atmospheric deposition and attachment parameters. These parameters could be used to interpret the ratio of dose to implanted ^{210}Po activity for that room and, hence, a way to interpret the implanted ^{210}Po in terms of cumulative dose. Once a catalog of the parameters for rooms with different deposition environments has been collected, past changes in deposition environment may be included in the retrospective interpretation model. We are currently collecting such a catalog of deposition parameters in central North American homes. We will then be able to interpret the more than 2000 surface deposited and implanted progeny activities that were measured as part of the IRLCS.

CONCLUSIONS

Radon concentration measurements alone may not be sufficient to reconstruct cumulative dose estimates that are accurate enough to accurately assess the dose-response relationship in residential radon epidemiologic studies. New techniques that measure surface-deposited and implanted progeny show good promise to improve radon dosimetry for future radon studies.

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