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Sent: Thursday, May 28, 2015 2:21 AM
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Subject: LU 14-218444-HR-EN Testimony of Katherin Kirkpatrick 2015-05-28 -- Email 7 of 11
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Dear Karla:
Please accept my attached testimony for submission into the record of LU 14-218444-HR-EN on the Mt. Tabor Reservoirs Decommissioning, scheduled for hearing this afternoon at 2:00 p.m.
This batch consists of Exhibit R in support of my legal brief. Kindly send me an electronic receipt when this document is entered.

Thank you,
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Risk Factors for Osteosarcoma in Young People in Cornwall: A Case-Control Study

Risk Factors for Osteosarcoma in Young People in Cornwall: A Case-Control Study

Volume:5

Issue:2

Year: 2006

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Abstract

A case-control study has been carried out in an attempt to identify risk factors that may be implicated in a group of cases of osteosarcoma in young people that have occurred over a nine year period in or near the West Cornish town of Helston. The incidence of osteosarcoma in the study area was substantially in excess of the national rate, but did not significantly exceed that found in the South-West region as a whole (chi2 with continuity correction = 0.003; p 0.956).

Data were obtained by postal questionnaire. In addition, domestic radon levels in the homes of cases and controls were obtained by direct measurement. Statistical analyses included tests of association x2 tests, or Fisher's Exact Test where appropriate) and the calculation of odds ratios for exposure. For continuous variables, Mann- Whitney tests of rank distribution were undertaken.

Much higher levels of domestic radon were found in the houses of cases compared with those of controls (Mann-Whitney rank distribution test: p = 0.000376). Other possible risk factors identified were diphtheria/tetanus/ pertussis immunisation, difficulty coping at school, periods of low mood, and previous accidents. BCG immunisation appeared to have a protective effect. However, logistic regression analysis showed that these were unimportant in comparison with radon, and their role as possible risk factors for osteosarcoma is by no means proved.

The strength of association with radon exposure is remarkable and convincing. Other associations which are weaker, but nonetheless statistically significant, are consistent with previous published research. The study should, though, be repeated on a larger scale in order to replicate these findings.

Key Words: Environmental health; osteosarcoma, radon, risk factors.

Introduction

A case-control study has been carried out in an attempt to identify risk factors that may be implicated in a group of cases of osteosarcoma in young people that have occurred within the past decade in or near the West Cornish town of Helston. Recently, concern has been expressed there about the number of cases (BBC News 2004; Guardian Unlimited, 2004, the "Helston Packet", 29th January and 26th February 2004). A total of seven cases has been identified, which were diagnosed with osteosarcoma in the period 1996 to 2003 inclusive. Data pertaining to six of these cases, three of whom were male and three female, are presented here. In every case, they were aged less than 20 when diagnosed with osteosarcoma. The public perception was that these cases constituted a cluster, and that the incidence of osteosarcoma was in excess of that normally to be expected in that area. There was concern that this might have resulted from

exposure to a common risk factor, which remained unidentified.

Osteosarcoma, though a rare form of cancer, is the fourth most common cancer in people under 20 years of age (Homa et al., 1991), and the most common type of bone cancer in this age group. It is derived from primitive bone-forming mesenchyma (Kramarova and Stiller, 1996). It is bimodally distributed by age, with an initial peak at age 15-19 years (Fraumeni and Boyce, 1982).

Table 1.0 summarises the incidence of osteosarcoma in the study area (i.e. an area of radius 13 miles around Helston). It will be noted that, while the incidence of osteosarcoma was substantially in excess of the national rate, it did not significantly exceed that found in the South-West region as a whole (chi2 with continuity correction = 0.003; p 0.956).

Table 1.0: Incidence of osteosarcoma in people aged <25 in the UK*, the South West* and the Study Area

	Location		
	UK	SW	Study area
No. of cases	732	210	7
Population <25 (100,00s)	112.03	6.94	0.14
Observation	1995-2000	1995-2000	1995-2005
No. of years	6	6	9
Person/years at risk	672.18	41.64	1.26
Annual rate/100,000 population <25	1.09	5.04	5.56
Rate ratio (UK = 1)	1.00	4.62	5.10

* Sources: Office for National Statistics 2000 and Stiller et al. 2004.

Very little is known about the aetiology of osteosarcoma in humans. Following local representations, the South West Cancer Intelligence Service prepared a report on the alleged cluster (South West Cancer Intelligence Service, 2004). This states that the aetiology of osteosarcoma is generally poorly understood. The only aetiological factor that is unequivocally recognised is ionising radiation (Rowland *et al.*, 1983), having first been described in female factory workers after World War One who were exposed to radium and mesothorium (Patterson and Harman, 2001) Similarly, treatment especially of children with external beam radiotherapy is known to be associated with an increased risk of osteosarcoma (Patterson and Harman 2001, Tabone *et al.*, 1999), particularly in those with an inherited susceptibility. However, as the SWCIS report points out, there has been no published scientific evidence to date linking radon exposure to osteosarcoma in young people, in either the South West (Thorne *et al.*, 1996) or nationally (Cartwright, 2002). It should be noted that parts of Cornwall have some of the highest levels of radon intensity in England and Wales (Green at al., 2002), particularly in Kerrier district, where 48% of properties were found to have unsafe levels (Spear, 2004). Henshaw *et al.*, (1990) identified that, at an indoor level of 110Bq/m3, radon may cause 23-43% of cancer, and noted that the existence of radon hot spots had implications for the clustering of childhood cancer in the UK.

Other factors suggested to be related to osteosarcoma include tall stature, previous bone trauma (Fraumeni, 1967, Miller, 1976, Scranton *et al.*, 1975) and viruses (Finkel *et al.*, 1975). Animal studies have demonstrated an excess risk of bone sarcomas among larger breeds of dogs which suggests that a relationship may exist between human bone cancer and a large body size at the time of diagnosis (Operskalski *et al.*, 1987, Benedict *et al.*, 1988). Genetic factors have also been identified in a small percentage of cases. Hereditary factors are involved in some patients (Hansen MF 1991), and there is an increased risk in siblings of patients (Coley, 1970, Schimke *et al.*, 1974). Genetic mutations, e.g. of the p53 gene, and an increased incidence in children with the Li-Fraumeni syndrome have been reported (McIntyre *et al.*, 1994).

A number of antenatal environmental exposures such as infective agents, drugs and radiation are capable of altering the

normal development of an embryo which could contribute to the development of osteosarcoma in young people. Parental occupation is also of interest because parents can bring home chemicals or dusts from their workplace on their clothes, thus exposing their children. Parental chemical exposures may be associated with increased risk of osteosarcoma in children. Schwartzbaum *et al.* (1991) identified a statistically significant odds ratio of 2.6 among 78 childhood osteosarcoma patients for parents who reported that they gardened with fertilisers, herbicides and pesticides in the perinatal period, compared with parents of other childhood cancer patients. A genetic predisposition has also been suggested (Buckley *et al.*, 1998, Birch 1999). Earlier occurrence of osteosarcoma in girls than in boys may be associated with their earlier adolescent growth spurt (Price, 1955). Previous trauma is another factor which may predispose to osteosarcoma (Operskalski *et al.*, 1987). Immune status is another factor that has been implicated, with one study finding a highly significant excess of children with osteosarcoma who had not been immunised (Hartley *et al.*, 1988). Finally, psychological factors have been impugned, with another study finding that children who experienced difficulties at school were particularly at risk (Frentzel-Beyme *et al.*, 2004).

The SWCIS report states that there was no statistically significant difference in incidence between West Cornwall and surrounding areas (South West Cancer Intelligence Service, 2004). However, this assessment was made at PCT level, so any local clusters would have been substantially diluted in a larger population. It concluded that there was no evidence of a single environmental or other risk factor causing osteosarcoma in the West of Cornwall Primary Care Trust area, and that public concern was therefore misplaced. However, the study conducted by the SWCIS was a descriptive study utilizing cancer registry data. It was not an analytical study, and was neither designed to establish causation of this group of cases, nor indeed could it as the requisite data on environmental exposures was unavailable. SWCIS itself has commented that the cancer registry does not hold data on lifestyle factors which may affect cancer risk, duration of residence, or occupational or environmental exposures (South West Cancer Intelligence Service Factsheet 18).

The SWCIS report further asserted that any additional investigation of this group of cases would be unethical, as any study of such a small number would necessarily be underpowered (South West Cancer Intelligence Service 2004). This is incorrect. It is true that type 2 errors are more likely in a study of a small sample, but whether or not it is underpowered depends on the effect size of possible risk factors being examined. Thus, our power and sample size calculations indicate that a matched casecontrol study of six cases, with four controls per case, with an a-value of 0.05 and a power of 0.8, would produce a significant result where the exposure in the control population was 10%, and the odds ratio 15:1. The present study is thus designed to complement and extend the SWCIS study.

Methods

A case-control study was undertaken, in order to identify, where possible, differences in exposure to a range of risk factors between the six cases and a group of matched controls. The specific potential risk factors investigated include radon, antenatal exposures, a family history of cancer or congenital malformation, parental occupation and psychological disturbances. Ethical approval was granted by the Faculty of Applied Sciences Ethics Committee at the University of the West of England, which follows NHS governance procedures.

The cases were identified via third party introductions or from word of mouth and media interest (television, radio and press). A close relationship had developed between locally affected families since the high media interest and as such facilitated the task of case finding. The study area comprised some 531 square miles within a 13-mile radius of Helston. The area extended from Redruth to the North, to the Lizard to the South, and St. Ives to the West.

Four controls were selected per case. Some were neighbourhood controls introduced by cases' families, while others were voluntary participants from local schools who had heard about the study. Cases and controls were matched for age, sex and ethnicity. For both cases and controls, participation was on the basis of informed consent.

There were two sources of information regarding exposures of interest. For most exposures, data were obtained by postal questionnaire. In addition, domestic radon levels in the homes of cases and controls were obtained by direct measurement.

The questionnaire, which was piloted among 25 unaffected families, addressed events during the index pregnancy, past medical and social history of the subject and his or her parents and siblings and other family members. Risk factors examined included length of gestation, place and type of delivery, birth weight, condition at birth, neonatal events (e.g. phototherapy, breastfeeding), previous illnesses, drug use and abuse, previous medical treatments including

radiotherapy, immunisation status, and familial exposure to chemicals.

Domestic radon measurements were made using the Pylon Radon Detector Model AB-5, which is an instrument validated by the NRPB and the United States Environmental Protection Agency. The tool was calibrated for efficiency, and a pilot study undertaken to ensure familiarity with the equipment. In order to ensure comparability of results, families were asked to keep windows and doors closed as much as possible for twentyfour hours prior to, and during, measurements, and to turn off all air exchange systems. Siting of the equipment, well away from outside walls and avoiding draughts, was important, as was avoidance of taking measurements during severe storms or strong winds. Calculations were made using an hourly interval method, and expressed in Bq/m3. All radon measurements were made between 1st September and 1st October 2004. Each assessment was undertaken over an 8 hour period. The first three hourly readings were ignored, allowing the instrument to acclimatise to its surroundings. Cases and controls were assessed sequentially, the sequence being determined by random allocation. The apparatus was set up in a bedroom of the occupants' choosing. Background readings were calibrated after each assessment and programmed into the detector. Because of the possible effect of variations in air pressure on radon measurements, local barometric readings were obtained from Culdrose, Cornwall at 12:00 GMT (at www.metoffice.gov.uk).

Results were calculated using the radon concentration formula:

$$C = \frac{CPM - BG}{S}$$

Where:

C	is the concentration (units depend on sensitivity)
CPM	is the count per interval value expressed in counts per minute
BG	is the background level expressed in counts per minute
S	is the counting sensitivity value

Statistical analyses included tests of association (x2 tests, or Fisher's Exact Test where appropriate) and the calculation of odds ratios for exposure. For continuous variables, such as radon measurements, Mann-Whitney tests of rank distribution were undertaken.

Table 2.0 Domestic radon measurements (Bq/m3)

Case number	Results: Bq/m3					
	Cases	Matched controls				
		A	B	C	D	Average of matched controls
1	260	176	164	120	106	141.5
2	3484	52	136	198	166	138.0
3	234	98	90	220	122	132.5
4	352	126	144	144	162	144.0
5	200	54	40	108	140	85.5
6	250	26	38	40	40	61
All cases average	796.7					117.1

Table 3.0 Domestic Radon levels in relation to the NRPB Intervention Level of 200Bq/m3

	cases	controls	Total
At or above intervention level	6	1	7
Below intervention level	0	23	23
Total	6	24	30

Results

Measured radon levels in the houses of cases and controls are summarised in Table 2.0. There was a marked difference, in relation to the NRPB domestic radon intervention level of 200 Bq/m3, between the homes of cases and of controls (p, by Fisher's Exact Test = 0.0000117). These are summarised in Table 3.0. In order to examine possible meteorological factors that might have influenced radon measurements, possible correlations between radon measurements and wind speed, wind direction, rainfall, humidity, air temperature and atmospheric pressure were examined. The strongest correlation (r = 0.28) was with wind speed, i.e. only 7.8% of variation in radon measurements could be explained by variations in this.

Radon measurements ranged from 26 to 3484 Bq/m3. This latter measurement was very much higher than the second highest radon measurement, which was 352 Bq/m3. Consequently, a Mann-Whitney rank distribution test was conducted in order to avoid the possible distorting effect of this one extreme value. The cases occupied five of the top six ranks, while the remaining case was in rank 7. The probability of this distribution having arisen by chance was 0.000376. Similar tests were undertaken in respect of other continuous variables, but no other significant results were obtained. Clearly, radon levels measured at a particular point in time are not a measure of total exposure. Accordingly, cases and controls were asked about duration of residence in their current addresses, and there were in fact no significant differences between them.

As regards categorical variables, 2x2 contingency tables were examined, and odds ratios and 95% confidence limits calculated. The results in respect of exposures experienced by cases and controls are summarised in Table 4.0.

Cases were much more likely than controls to have had iphtheria/tetanus/pertussis immunisation, and also to have had difficulty coping at school, periods of low mood, or previous accidents. However, BCG immunisation was much more frequent among the controls, and therefore appeared to have a protective effect. A logistic regression analysis was undertaken, which involved construction of a model incorporating those variables which individually were positively associated with the occurrence of osteosarcoma, viz. radon above intervention level, diphtheria/ tetanus/pertussis immunisation, school difficulties, periods of low mood, and previous accidents. The model chi2 for this five variable model was 30.02 ($p < 0.0001$). An identical model chi2 was found for a two variable model comprising radon above intervention level anddiphtheria/tetanus/pertussis immunisation only, ndicating that school difficulties, periods of low mood, and previous accidents had no impact at all on the predictive power of the model. For radon above itervention level alone, chi2 was 24.64 ($p < 0.0001$, without continuity correction).

Other possible risk factors enquired about included factors related to the biological mother's obstetric history, i.e. occupation before, during and immediately after the birth, and exposure to X-rays, medication and trauma during pregnancy. Parental occupations (particularly in horticulture and agriculture) were also enquired about, and exposures to chemicals and other external agents (i.e. dusts, fumes, X-rays, fertilisers, herbicides, pesticides, prescription medication, alcohol and tobacco), and serious illnesses in the extended family. Factors concerning the construction of buildings which might affect levels of domestic radon (presence of double glazing, cavity wall insulation, or draft exclusion, and construction date) showed no variation between cases and controls. There were no significant associations involving any of these factors, or with the existence of serious illnesses in other family members, except for genetic diseases in aunts, which cases reported more frequently than controls (odds ratio = 1.83; 95% confidence interval = 1.14 – 23.83). There would not appear to be any plausible biological mechanism to explain this, and it appears likely that it is simply a chance

result arising as a consequence of multiple hypothesis testing.

Discussion

Summary of main findings

The number of cases identified was markedly higher than that which would have been found if national incidence rates applied, but was not significantly higher than the regional rate. The number of cases found in the Helston area during the study period cannot therefore be regarded as a cluster. Consequently, there is no need to postulate a specific localised exposure which may have been a risk factor for osteosarcoma. There is always a danger, in investigating an alleged cluster, of artificially designating a cluster by *post hoc* rationalisation (the socalled 'Texas sharpshooter' fallacy). We have not done this. Rather, we have investigated a group of cases which occurred in close proximity in space and time. Whether or not these cases constitute a cluster is a matter of semantics, and such labelling is not necessarily helpful in endeavouring to identify possible risk factors.

This study has shown a very strong association between levels of domestic radon and the development of osteosarcoma in the group of cases investigated ($p = 0.000376$). Radon levels throughout the South West of England are higher than in other parts of the country, and this may account for the relatively high incidence rate for osteosarcoma found in the region. Logistic regression analysis indicated that diphtheria/tetanus/pertussis immunisation may be a relatively minor risk factor for osteosarcoma, but this remains a hypothesis requiring further testing on a much larger scale. Other factors that were weakly associated with the development of osteosarcoma included difficulty coping at school, periods of low mood, and previous accidents, but these had no impact on the logistic regression model, so their possible roles as risk factors should also be regarded as hypotheses to be tested in much larger studies. Paradoxically, BCG immunisation appeared to have a protective effect. In conclusion, the association between domestic radon levels and developing osteosarcoma is very strong, and this suggests a causal relationship.

Table 4.0 Comparison of cases and controls in respect of selected exposures

History of:	Cases		Controls		Odds Ratio	95% Confidence Interval
	Yes	No	Category 1	Category 2		
Diphtheria/tetanus/pertussis immunisation	6	0	6	18	18.00	1.787 - 181.316
H. influenzae immunisation	2	4	15	9	0.30	0.045 - 0.982
Poliomyelitis immunisation	4	2	9	15	3.33	0.505 - 22.018
MMR immunisation	4	2	18	6	0.67	0.097 - 4.605
BCG immunisation	2	4	20	4	0.50	0.013 - 0.745
X-ray exposure	3	3	16	8	0.50	0.082 - 3.360
Medication	1	5	3	21	1.40	0.119 - 16.459
Bacterial infections	5	1	21	3	0.71	0.061 - 8.398
Viral infections	5	1	13	11	4.23	0.427 - 41.875
Difficulty coping at school	5	1	2	22	55.00	4.128 - 732.747
Difficulty sleeping	2	4	4	20	2.50	0.336 - 18.629
Periods of low mood	3	3	2	22	11.00	1.271 - 95.181
Swimming	5	1	18	6	1.67	1.161 - 17.258
Accidents	4	2	4	20	10.00	1.342 - 75.514

Strengths and the limitations of this study

The main limitation of this study arises from the small number of cases. Nevertheless, the strength of association with radon exposure is remarkable and convincing. Other associations which are weaker but nonetheless statistically significant are consistent with previous published research. The study should, though, be repeated on a larger scale in order to replicate these findings.

Conformity with existing research literature

The strong association with radon exposure is consistent with previous work implicating ionising irradiation in the aetiology of osteosarcoma (Rowland *et al.* 1983), though this particular finding is new information, for, as the SWCIS report (South West Cancer Intelligence Service, 2004) pointed out, there has been to date no published research evidence for the role of radon as a risk factor for osteosarcoma in young people.

Other factors that may constitute risk factors for osteosarcoma include diphtheria/tetanus/pertussis immunisation, difficulty coping at school, periods of low mood, and previous accidents. BCG immunisation, however, appeared to have a protective effect.

A recent collaborative analysis of data from 13 casecontrol studies of residential radon and lung cancer (Darby *et al.*, 2005), involving over seven thousand cases with approximately two controls per case found a mean domestic radon

concentration in the homes of cases of 97 Bq/m3, and in the homes of controls of 104 Bq/m3. This study has found proportionally much larger differences between cases and controls, suggesting that radon is more unequivocally important as a risk factor for osteosarcoma than for lung cancer.

The indication that immunisation status, particularly the apparent protective effect of BCG immunisation, may affect likelihood of developing osteosarcoma is consistent with previous work suggesting that children who had not been immunised were significantly more at risk of osteosarcoma than others (Hartley *et al.*, 1988). As regards accidents, a possible role for trauma as a risk factor has previously been suggested (Fraumeni, 1967; Miller, 1976). In addition, our finding of the possible role as a risk factor of having had difficulty coping at school is consistent with a previous study indicating that children who experienced such difficulties were particularly at risk (Frentzel-Beyme *et al.*, 2004).

The implications for future research and practice

There is a clear need for the findings of this study to be replicated in larger scale studies, as well as for investigation of the reasons for the demonstrated variations in domestic radon levels, in order to enable effective interventions to be implemented. The risks associated with domestic radon exposure have been so conclusively demonstrated in this study that serious consideration should be given to taking remedial action to reduce the risk at the earliest possible opportunity.

Conclusions

A case-control study of risk factors for osteosarcoma in a group of cases in young people in and around Helston, in Cornwall, indicated a very strong association between domestic radon levels and the development of osteosarcoma. This is consistent with other research, though is the first time that this particular association has been demonstrated in this age group. The strength of the association ($p = 0.000376$, by Mann-Whitney rank distribution test) strongly suggests a causal relationship. Other associations, e.g. with diphtheria/tetanus/pertussis immunisation, were much weaker, and more research is needed in larger scale studies to elucidate their possible role as causal factors.

Acknowledgements

Our sincere thanks go to the following people who contributed to the study in a number of ways:

Dr. Andy Tubb and Dr. Paul von Dorfman for their continual support and guidance, Mrs. Judy Brown and Ms. Alison McEwen for their expertise and time regarding the radon detector equipment and associated analytical data output, Andrew George M.P. for his mediation between us and significant others, and our respective families who gave us the time and space to conduct the investigation to its conclusion.

Our appreciation also goes to the participants and their families who took part, without which this study would not have come to fruition.

Finally, our thanks go to the Chartered Institute of Environmental Health who funded, and encouraged us to complete, the project.

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From: Kate & Chris <samsa@pacifier.com>
Sent: Thursday, May 28, 2015 2:23 AM
To: Council Clerk – Testimony
Cc: Hales, Mayor; Commissioner Fritz; Commissioner Fish; Commissioner Novick; Commissioner Saltzman
Subject: LU 14-218444-HR-EN Testimony of Katherin Kirkpatrick 2015-05-28 -- Email 8 of 11
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Dear Karla:
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Applied Geochemistry 23 (2008) 2676–2685

Contents lists available at ScienceDirect

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Radon transfer from groundwater used in showers to indoor air

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ARTICLE INFO

Article history:
Received 19 December 2007
Accepted 31 May 2008
Available online 7 June 2008

Editorial handling by R. Fuge

ABSTRACT

Estimation of Rn transfer from water to indoor air based on multi-day measurements may underestimate alpha exposure that occurs at short time scales in confined spaces, such as from showering, in houses with high Rn activities in the water supply. In order to examine one such incremental increase in exposure, variations in Rn in water and indoor air in 18 houses with private wells in western North Carolina (USA) were investigated. Radon in well water ranged from 158 to 811 Bq L^{-1} (median 239 Bq L^{-1}). After 20-min showers in bathrooms with closed doors, peak Rn in air increases (above background) ranged from 71 to 4420 Bq m^{-3} (median 1170 Bq m^{-3}). Calculated transfer coefficients at the scale of a 40-min closed bathroom (20-min shower plus 20 min post-shower) are described by a lognormal distribution whose geometric mean exceeds the widely-used $\sim 10^{-4}$ whole-house transfer coefficient by about one order of magnitude. As short-lived decay products grow from shower-derived Rn, short-term alpha energy exposure occurs in bathrooms in addition to the exposure caused by Rn mixed throughout the volume of the house. Due to the increasing ratio of Rn decay products to Rn, alpha energy exposure is greatest several minutes after the shower is turned off. For a 7.2-min shower with 10 min of additional exposure before opening the door, a geometric mean 5.6% increase in exposure over the $\sim 10^{-4}$ whole-house transfer coefficient derived from longer measurement periods was estimated. In addition to Rn activity in water, short-term shower exposure to Rn progeny depends on exposure time, ventilation, attachment and deposition, among other variable factors that characterize individual houses and residents.

1. Introduction

Naturally occurring radon-222 (Rn) in indoor air is a significant cause of lung cancer (Krewski et al., 2006), and houses are an important exposure location due to the large proportion of time spent at home. Radon is believed to be the second-largest cause of lung cancer, after smoking, and may cause an estimated 21,100 lung cancer deaths/a in the United States (Environmental Protection Agency (EPA), 2003). Although direct transfer from soil and rock is the largest aggregate exposure pathway, Rn can degas from water into buildings and constitutes an important secondary source of Rn exposure in groundwater-dependent populations. Although both inhalation and ingestion of Rn-rich water may represent some exposure to the lungs and stomach, respectively, inhalation is believed to be a larger health risk (National Research Council (NRC), 1999). Radon exposure is typically evaluated using a linear, no-threshold model (Samet, 2006) so that all exposures to Rn are potentially significant, and treatment of water supplies containing high Rn activities may be an effective way to reduce Rn exposure in some cases.

Elevated levels of Rn in groundwater are widespread in many areas of the USA (Focazio et al., 2006) and especially in areas underlain by felsic crystalline rocks (Table 1). When high-Rn groundwater is utilized in homes, clothes washing, dishwashing, sink usage and showering can cause 28–98% of dissolved Rn to degas (Partridge et al., 1979), and mixing of this Rn into the volume of the house creates

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doi:10.1016/j.apgeochem.2008.05.021

Table 1
Radon in water activities in selected areas of the United States. Radon activities in Bq L^{-1}. (1 Bq L^{-1} = 26.67 pCi L^{-1})

Region	Rock type	Median or geometric mean ^{222}Rn, range in parentheses (Bq L^{-1})	Reference
Georgia	Granitic gneiss	230 (4.8–31,000)	Dillon et al., 1991
South Carolina	Crystalline rocks	84 (15–2197)	King et al., 1982
North Carolina	Granite	218 (19–1623)*	Loomis, 1987
Virginia	Granite	287 (37–901)	Harris et al., 2006
New Jersey	Granite	107 (<4–463)	Wanty et al., 1991
Pennsylvania	Crystalline rocks	52 (2–1961)	Senior, 1998
Maine	Granite	1208 (22–4514)	Brutsaert et al. 1981
Iowa	Sedimentary rocks	12 (1–87)	Field and Kross, 1998
Area of this study	Meta-intrusive	277 (27–1687)	Campbell, 2006
	Meta-sedimentary	149 (64–529)	Campbell, 2006

* Range is 5th–95th percentiles.

incremental increases in exposure to Rn progeny over the relatively long (multi-day or longer) measurement periods typically employed in residential Rn exposure calculations.

It has been noted previously that this single-cell Rn transfer model of water supply contribution to whole-house average Rn underestimates exposure because occupants are in proximity to water uses and because daily lows in airborne Rn activities occur when residents are not at home (e.g. Gesell and Prichard, 1980). However, the most commonly used basis for exposure estimation is at the scale of the entire house and over relatively long measurement periods (days or longer; NRC, 1999). At those spatial and temporal scales, transfer of Rn into homes is commonly modeled by a transfer coefficient, C_T

$$C_T = \frac{\text{Increment of airborne radon added by water}}{\text{Radon activity in water}} \quad (1)$$

Estimates of C_T at whole-house scales ($C_{T,H}$) have consistently centered on approximately 10^{-4} within a large range of variability (Hess et al., 1982,1990; Nazaroff et al., 1987; NRC, 1999).

Other research has documented the effects of specific water uses, such as showering, at short temporal and spatial scales, before Rn mixes throughout the house (McGregor and Gourgon, 1980; Bernhardt and Hess, 1996; Fitzgerald et al., 1997). Use of Rn-rich water results in short-term exposures to humans that may not be accounted for by multi-day, whole-house measurements. The purpose of this study is to quantify the effect of showering, a routine water use that occurs in enclosed spaces. Details of relevant physical processes, such as effects of shower heads, ventilation, attachment of Rn progeny to particles and deposition of progeny onto surfaces, have been simulated in laboratory (Partridge et al., 1979; Fitzgerald et al., 1997) and modeling (Datye et al., 1997; Nikolopoulos and Vogiannis, 2007) experiments. Variations in these parameters can result in several orders-of-magnitude changes in net transfer of Rn from water to an indoor space and mobility of its decay products. Rather than to control the processes involved (e.g. degassing, attachment, deposition and escape, which were beyond the scope of the field measurements), the objective of this paper is to document net transfer under real-world conditions, in which orders-of-magnitude variations in Rn transfer characteristics are expected between houses (e.g. Hess et al., 1990). By examining typical homes, a distribution of net Rn transfer characteristics may be determined, and the potential exposure to Rn decay products can be estimated.

The selected houses use private wells in fractured crystalline rocks of western North Carolina (Fig. 1), an area known to produce high levels of Rn in indoor air and groundwater. Previous sampling in the study area indicated that Rn in water is strongly associated with rock type. Meta-igneous rocks, including granitic gneiss and granodiorite, exhibit higher median Rn levels than metasedimentary rocks, including garnet-mica schist, mica gneiss, and rocks of the Brevard Fault Zone (Table 1). In contrast to these high Rn activities, U and Ra levels were generally below drinking water standards throughout the study area (Campbell, 2006). Sixteen of the 18 wells selected for this study were included in the previous study, and 15 of those 16 exhibited levels of Rn in water in excess of the EPA-proposed alternate maximum contaminant level of 148 Bq L^{-1} (Campbell, 2006).

2. Methodology

Eighteen houses with private wells were visited between February and August 2006. Most wells are completed in granitic gneiss, and a few are in other rock types including granodiorite, amphibolite, and mica schist (Table 1). At the well head sampling tap (where present), a water sample was collected for ^{222}Rn by submerging a glass vial into a glass beaker receiving a low flow of minimally aerated water, then sealing the submerged vial. Radon-in-water samples were rushed to a commercial laboratory and typically analyzed within 24–36 h of collection by liquid scintillation counting. Radon activities were decay-corrected to the time of collection.

Radon in bathroom air was determined using a Durridge RAD7 alpha counting instrument in 5-min counting intervals, with the sampling point at floor level approximately 1 m from the shower. The instrument operated at a flow rate of 0.7 L min^{-1} through a 0.45 μm inlet filter into a chamber of internal volume ~1 L. The RAD7 was calibrated by the manufacturer in September 2005 (efficiency 0.006027 ± 0.000241 Bq m^{-3} cpm^{-1}) and September 2006 (efficiency 0.006514 ± 0.000261 Bq m^{-3} cpm^{-1}; counting errors are ±2σ). The RAD7 counts alpha decays of ^{218}Po, the daughter of ^{222}Rn, generated only by in situ decay, providing a measurement of ^{222}Rn gas exclusive of Rn progeny. Under static conditions, the RAD7 equilibrates

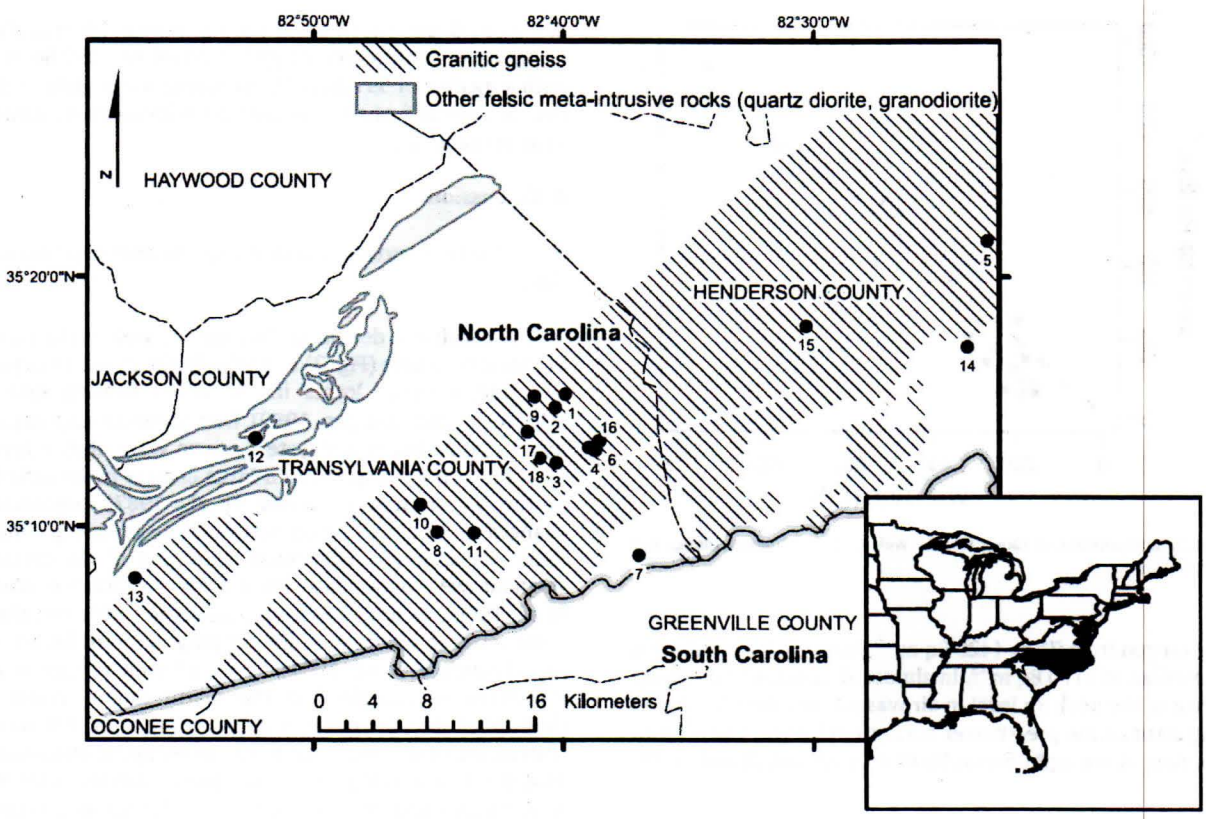

Fig. 1. Map showing the study area (Henderson and Transylvania counties). Geographic layers obtained from www.nationalatlas.gov; geological data modified from North Carolina Geological Survey (1985).

with the room air in approximately 10 min, controlled by the ~3-min half-life of ^{218}Po. Thus, under dynamic conditions, the RAD7 count represents a moving average of the past ~10 min of Rn activities. The RAD7 measured ambient Rn levels for 5–15 min before the water was turned on. Background was established as the highest 5-min interval during the pre-shower period because of the possible isotopic disequilibrium inside the RAD7. For each 5-min counting period, the 2σ counting error calculated by the RAD7 was used, and a standard propagation of error equation (Clesceri et al., 1998) was applied for subtraction of the pre-shower airborne Rn measured in the room.

To conduct the experiment, a typical warm shower was operated for 20 min at approximately the same time that the well water samples were collected, and RAD7 measurement continued with the door closed for at least 30 min after the water was turned off. The shower curtain or stall door was left as open as possible to encourage mixing of air in the room, but closed as necessary to avoid spilling water. Bathroom doors were kept closed and only opened as necessary to enter or leave to monitor the experiments. Ventilation fans, where present, were not used, and windows were kept closed. During some tests, water temperature was measured continuously using a Hobo data logger suspended in the stream of water. Typically after the 20-min shower had ended, warm tap water was collected at the sink or bathtub by submerging a glass vial in a beaker filled with minimally aerated tap water, and warm shower water was collected at that time by holding a glass vial into the shower water and capping without bubbles. Statistical handling of data and construction of a decay simulation were performed using the R software package (R Development Core Team, 2007).

3. Results

Radon in well water ranged from 158 to 811 Bq L^{-1} and had a median of 239 Bq L^{-1}. Radon in warm tap water ranged from 59 to 715 Bq L^{-1}, with a median of 150 Bq L^{-1}, not including one house with a Rn aeration system, in which the indoor tap Rn was less than the laboratory reporting limit of 3 Bq L^{-1}. At most houses, a relatively consistent decline is observed between the cold well water and the warm indoor tap water and thus well water and indoor tap water Rn are well correlated. Linear regression ($R^2 = 0.93$) indicates a slope of 1.05 and an intercept of −102 Bq L^{-1} (Fig. 2). Thus, as well water flows into the household plumbing and hot water system, it experiences an average absolute decline of approximately 100 Bq L^{-1} Rn regardless of the original activity (excluding one house with a Rn treatment system). Radon in warm shower water ranged from 24 to 390 Bq L^{-1}, with a median of 124 Bq L^{-1}. In the experimental showers, peaks of Rn in air were measured that reflect a transition from Rn accumulation in the room as it degasses from water to depletion as it escapes from the room (Fig. 3). Peak recorded levels of Rn in indoor

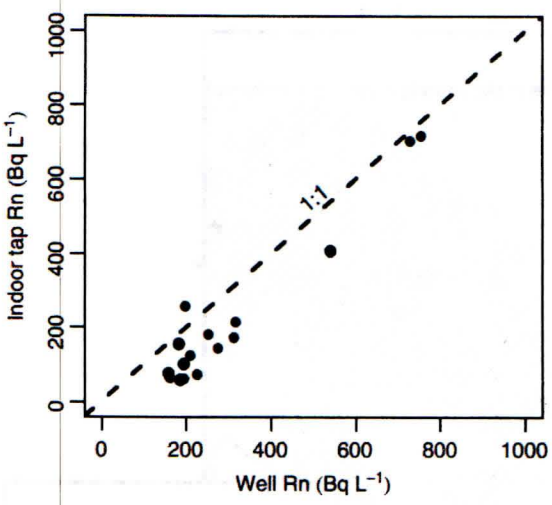

Fig. 2. Comparision of radon in cold well water and warm indoor tap water.

air ranged from 71 to 4420 Bq m^{-3} above background, with a median of 1170 Bq m^{-3}. In almost all cases, ±2σ counting error of the peak Rn level in air was outside the ±2σ counting error of the pre-shower background level. The 40-min average of the eight 5-min Rn in air counting periods comprising a 20-min shower plus the subsequent 20 min, after background subtraction, ranged from 84 to 1990 Bq m^{-3}, with a median of 591 Bq m^{-3}, excluding one slightly negative average and one house with a Rn-in-water treatment system (Table 2).

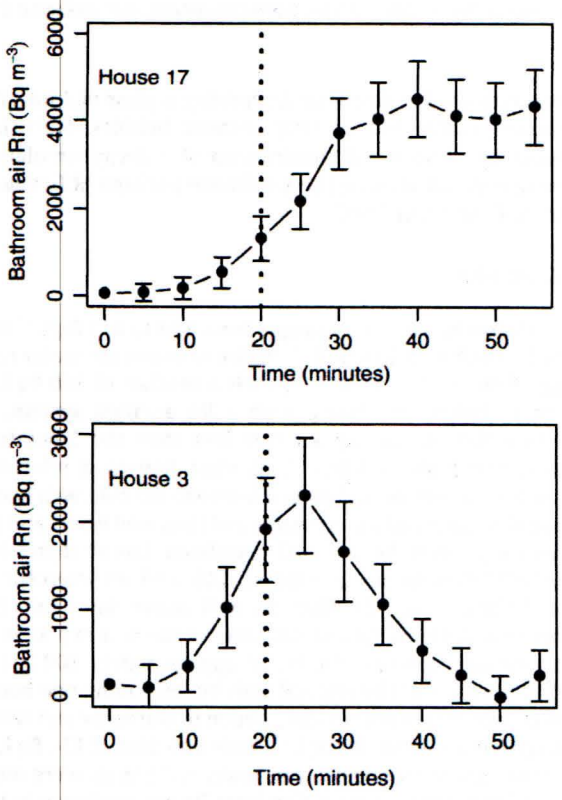

Fig. 3. Results from two shower experiments (prior to background subtraction) illustrating differences in peak size and timing after 20-min showers. Error bars represent ±2σ counting error.

4. Discussion

4.1. Airborne Rn accumulation during and after experimental showers

The consistent decline in Rn from the well to the warm indoor tap sample (Fig. 2) is probably the result of degassing and/or decay losses in the water heating system (McGregor and Gourgon, 1980). Due to the storage capacity of a home hot water system, warm water may require 12–24 h to fully respond to a large change in Rn activity in the water supply (Lowry et al., 1987). Significant changes in Rn activity may be caused by pumping conditions; for example, continuous pumping of an initially stagnant well may cause Rn activity to increase (Cook et al., 1999), or drawdown may shift the well's contributions between fractures of significantly different Rn activities (Lawrence et al., 1991). The cold well sample is an instantaneous sample, but the warm indoor water is time-integrated due to the residence time of the hot water system, and may represent a mix of pumping conditions. That the warm indoor water samples, collected after the well sample and generally after the shower was turned off, exhibit consistently lower Rn activities than the well samples, implies that the difference in Rn activity is mostly due to decay and degassing losses in the plumbing system, rather than large changes in Rn in influent well water. Although shower hardware is expected to release a significant portion of Rn in water, shower water samples exhibited only modestly lower Rn activities, on average, than indoor tap water, and 6 shower samples had higher activity than the corresponding tap water samples (Table 2). Thus, collection methods did not successfully quantify the degree of degassing caused by showers; however, warm indoor tap water contained less Rn than cold well water in most cases.

For Rn carried into the bathroom at a constant rate during the shower, Rn in air should increase steadily until the peak is reached (Fig. 3); thus, the peak value approximates the amount of Rn added to the room by the shower, minus the Rn that escaped the room prior to the peak. Overall, there is no direct relationship between Rn in water and peak Rn activity in air after a shower (Fig. 4). This suggests that other processes, not controlled in these experiments, are significant controls on airborne Rn activities in these bathrooms. These include ventilation (Fitzgerald et al., 1997), differences between shower heads (Partridge et al., 1979) and water flow rates, and mixing into bathrooms of different volumes. Laboratory experiments with shower ventilation indicate that strong ventilation causes peak Rn levels to occur sooner than poorly ventilated rooms, which exhibit later, larger peaks. More ventilation also results in better mixing of Rn throughout the volume of the room (Fitzgerald et al., 1997), so airborne Rn measurements in these rooms should be more representative

Table 2
Radon results for water and air

House number	Rock type	^{222}Rn at well (Bq L^{-1})	Temp. (°C)	^{222}Rn at indoor tap (Bq L^{-1})	Temp. (°C)	^{222}Rn in shower water (Bq L^{-1})	Temp. (°C)	Shower air peak Rn (Bq m^{-3})	40-min avg air Rn increase (Bq m^{-3})	Time to peak from start of shower (min)	Experiment water temperature range (°C)	40-min $C_{T,B}$
1	Granitic gneiss	316	13.1	215[a]	14.0	229	14.1	550 ± 293	271	45	16–21[f]	0.00086
2	Granitic gneiss	728	13.3	700[c]	23.1	382	23.1	603 ± 465	244	40		0.00033
3	Granitic gneiss	540	13.5	406[c]	30.9	176	30.9	2090 ± 705	909	25		0.00168
4	Granitic gneiss	557	13.2			245	46.0	923 ± 589	510	25		0.00092
5	Granitic gneiss	252	13.8	180[c]	38.2	150	38.2	1350 ± 690	754	30	28–34	0.00299
6	Granitic gneiss	186	13.5	59[c]	42.4	24	42.4	284 ± 363	84	25	52	0.00045
7	Garnet-mica schist	195	13.0	62[c]	38.8	104	38.8	71 ± 301	[d]	20	47–51	
8	Granitic gneiss	754	14.4	715	35.9	390	36.7	2170 ± 794	872	35	34–36	0.00116
9	Granitic gneiss	194	15.2	100	36.5	80	39.3	535 ± 404	192[e]	35	30-34	0.00099
10	Granitic gneiss	158	17.2	76	34.3	94	33.4	678 ± 434	129	55	27–40	0.00082
11	Granitic gneiss	312	15.7	172	25.3	155	31.7	1380 ± 553	697	35		0.00223
12	Quartz diorite/granodiorite	162	16.2	63	31.4	76	30.1	710 ± 439	390	25		0.00241
13	Amphibolite	274	14.4	144	36.9	110	36.2	2000 ± 688	871	40		0.00318
14	Porphyroblastic gneiss	198	14.9	256	22.5	320	22.8	1530 ± 560	568	25	25–30	0.00287
15	Granitic gneiss	811	15.9	<3[b]	42.0		42.0	106 ± 287	[d]	40	31–38	
16	Granitic gneiss	182	13.6	156	33.7	137	33.5	1170 ± 546	617	25	32–34	0.00339
17	Granitic gneiss	225	14.7	73	30.3	139	27.5	4420 ± 897	1990	40	30–44	0.00884
18	Granitic gneiss	209	15.3	123	41.5	98	40.2	1310 ± 621	736	30	22–29	0.00352
	Minimum	158	13.0	59	14.0	24	14.1	71	84	20		0.00033
	Maximum	811	17.2	715	46.0	390	46.0	4420	1990	55		0.00884
	Median	239	14.4	150	35.1	124	34.9	1170	592	33		0.00196
	Geo. mean	295		148		122		928	467			0.00165

Counting errors are ±2σ.

[a] Sample collected at kitchen sink.

[b] Rn in water aeration system in place. This house excluded from median and geometric mean calculations for all except well water.

[c] One temperature measurement made while sampling indoor tap and shower water. Temperature is assumed to be same for both samples.

[d] Negative average; excluded from transfer coefficient calculations.

[e] Room opened prematurely during experiment.

[f] Experiment used cold water; all other experiments used warm water.

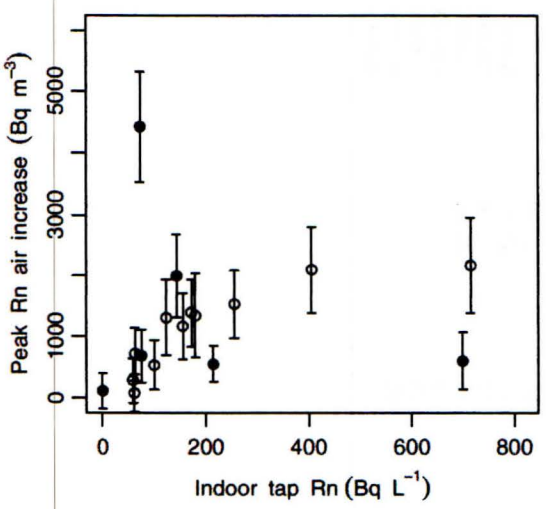

Fig. 4. Shower results for the peak value of Rn in air during the experiment in relation to the Rn level of indoor tap water. Open circles represent peaks observed within 35 min of the start of showers; filled circles represent peaks occurring later.

of the Rn levels in the room air. Thus, the timing of the 5-min counting interval in which the peak was observed provides an indirect way to assess differences in ventilation mode (Figs 3 and 4). Fig. 4 shows that several of the later-peaking rooms (later than 35 min after the showers started; solid circles) recovered higher levels of Rn than the earlier-peaking rooms (open circles), consistent with the ventilation effects described by Fitzgerald et al. (1997). However, two of the later peaks (rooms 2 and 8) document lower-than-expected airborne Rn. This under-recovery of Rn may be explained by (1) the lack of mixing in the poorly ventilated rooms, which should lead to less representative RAD7 measurements; and (2) the characteristics of the rooms of those data points. Room 2 was poorly partitioned off from the rest of a small home as configured in the experiment, possibly suggesting that Rn was mixing throughout the house rather than only the bathroom. Room 8 is an unusually large bathroom.

Obtaining a specific Rn mass balance for each room is impractical because of the methodology of Rn measurement in the bathroom in which RAD7 measurements of the decay of ^{218}Po represent a moving average of the past ~10 min of measurement and counting error is large. In addition, there is no systematic information on critical factors such as Rn escape rate, ventilation, variations of Rn activity in well water during continuous pumping, shower flow rates, variations in the tendency of shower hardware to aerate water, variations in shower water temperature during experiments, and hot water system residence time. In sum, this study aims to document Rn degassing from showers under real-world conditions, whereas a specific mass-balance evaluation of the fate of Rn in a closed room is most practical in a controlled laboratory environment (e.g. Fitzgerald et al., 1997).

Although the experimental design was intended to examine typical homes under realistic conditions, two aspects of the experiments as conducted deviated from expected real-world conditions: the sampling point was at floor level, not at breathing height; and it was necessary to open the door to enter and leave the room during the experiments. Studies in multi-story buildings (Hess et al., 1982; Fisher et al., 1998) and within individual rooms (Zhang et al., 1993) indicate that overall Rn concentrations tend to be higher at lower levels, closer to the soil Rn source. However, vertical Rn gradients within rooms may be difficult to detect outside counting error (Put and de Meijer, 1988; Malanca et al., 1995). Also, airborne Rn may be lower in areas of rooms affected by air leaks such as windows (Abu-Jarad, 1982). In bathrooms while showers are being used, the largest Rn source is near the ceiling, not the floor, and temperature gradients are present (e.g. Fitzgerald et al., 1997), introduced not only by hot water usage but also induced by air-conditioning vents, ventilation fan ducts and leaks around windows and under the door. Thus, the accumulation of Rn and its progeny may be less effective near these leaks, and overall, Rn levels should not be expected to be uniformly mixed throughout a bathroom under these conditions. For example, placement of two Rn monitors in different locations in a bathroom during and after a shower resulted in Rn measurements that differed by a factor of more than two (636 vs. 270 Bq m^{-3}; Fitzgerald et al., 1997). This effect is also indicated in the present results by the median 12-min delay between the end of the shower and the peak Rn measurement. Although complicated by ^{222}Rn–^{218}Po disequilibrium between the room and the RAD7, the range of delays (Table 2) indicates the importance of ventilation in influencing the rate of Rn mixing from the shower into the rest of the room. Placing the sampling point on the floor could cause underestimation of peak Rn activity if the sampling point were in an air current exchanging under the door or if a large proportion of the Rn at breathing height escaped via leaks higher in the room, preventing some Rn from being measured near the floor. The significance of any of these leaks differs with the ventilation characteristics of each room, which are expected to vary between rooms, as indicated by the differing shapes of Rn accumulation curves (Fig. 3). If Rn activity differs between floor level and breathing height, this may result in a slight underestimation from real-world conditions.

The second departure from expected real-world conditions was the need to enter and leave the room during the experiments. In general, each bathroom was entered to start the shower at the end of the background counting period, to stop the shower 20 min later, and a third time after the shower was turned off in order to collect water samples from the bathroom. Although the door was opened as little as necessary to enter and leave the room, this effect may have allowed additional Rn to escape from the room than would have occurred under conditions of normal home occupancy. Here again, the results may undercount Rn in bathroom air for the period of time after the water is turned off.

4.2. Comparison to whole-house radon transfer coefficients

In order to compare short-term Rn transfer in the bathroom to longer-term exposure at the scale of the whole house, the results have been converted to transfer

coefficients over the volume of the bathroom, $C_{T,B}$, using the average airborne Rn increase over background during the 20-min shower plus the first 20-min post-shower, divided by the Rn activity in well water as indicated by Eq. (1) (Table 2). Because these transfer coefficients incorporate the average of 40 min of airborne Rn measurement, they are time-integrated in contrast to the peak values of airborne Rn reported in Fig. 4, which are averaged over only 5 min. Well water Rn activity was used rather than warm indoor water activity in order to compare $C_{T,B}$ to $C_{T,H}$ for the same source of water that supplies the entire house. This treats the hot water system as a Rn dilution mechanism similar to ventilation. Use of the warm indoor tap water data would have led to larger estimates of $C_{T,B}$. The approach necessitated removal of two data points: one with a slightly negative, within-counting error negative transfer and another from a house with a Rn-in-water treatment system and no significant airborne Rn increase caused by the shower. In order to generalize across bathrooms, incorporating ventilation and other uncertainty into a probability distribution, it was assumed that the transfer coefficient is lognormally distributed. This is consistent with the lognormal models used for whole-house Rn transfer from water to air (e.g. NRC, 1999). Examination of ln $C_{T,B}$ on a normal quantile–quantile plot (Dalgaard, 2002) also suggests that this assumption is reasonable given the sample size.

The resulting $C_{T,B}$ values (geometric mean = 1.65×10^{-3}, geometric standard deviation = 0.86) are about one order of magnitude higher than whole-house transfer coefficients (Fig. 5). These high transfer coefficients are the result of high airborne Rn, small spatial scale and short temporal scale. Because a transfer coefficient value is influenced by the spatial and temporal scale of measurement, Rn transfer experiments that do not simulate actual exposure conditions may not accurately quantify exposure. Whole-house transfer coefficients vary because of large variations in house volume, construction methods, and ventilation. The considerable variability observed in $C_{T,B}$ is less than the variability in $C_{T,H}$ determined by a recent data synthesis (geometric mean = 0.55×10^{-4}, geometric standard deviation = 3.5; NRC, 1999).

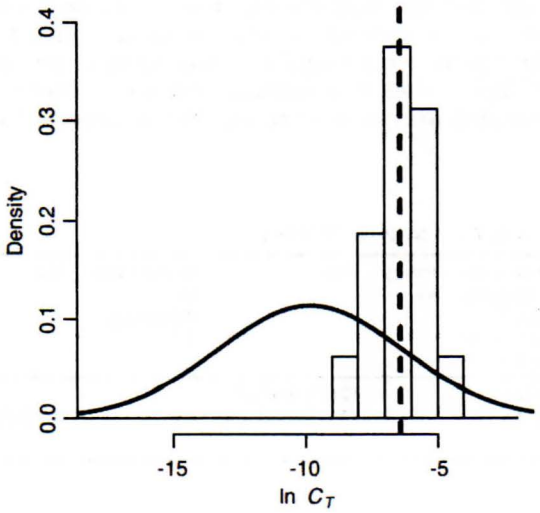

Fig. 5. Histogram of ln $C_{T,B}$ (this study) in relation to density of ln $C_{T,H}$ (NRC, 1999).

4.3. Implications for exposure

Because the health risk from Rn is posed by inhalation of its short-lived alpha-emitting decay products, comparison of transfer coefficients alone does not provide information about alpha energy exposure because high-Rn tap water contains much lower activities of Rn decay products (Bernhardt and Hess, 1996; Fitzgerald et al., 1997). Therefore, decay time is needed for exposure to be significant, that is, for the equilibrium factor (F; i.e. the alpha energy-weighted activity ratio of airborne Rn progeny to Rn; NRC, 1991) to increase from near zero. Here F is referred to at the scale of the entire house as F_H and at the bathroom scale as F_B. The ingrowth of Rn decay products, depicted in Fig. 6 for a hypothetical sealed room and neglecting escape, attachment, and deposition, indicates that F_B of nuclides delivered by the shower is relatively low during the actual use of shower water, and F_B continues to increase after the water has been turned off. Thus, the highest airborne Rn (at the end of water usage) will not coincide with the highest exposure (after the water is turned off). F_B depends directly on deposition rates to surfaces and indirectly on attachment rates to aerosols, which may differ between the Rn decay products ^{218}Po, ^{214}Pb and ^{214}Bi (Nikolopoulos and Vogiannis, 2007). Thus, actual F_B is expected to be lower than depicted in Fig. 6. Published field measurements (calculated from Fig. 6 of Fitzgerald et al., 1997) indicate a F_B range from 0.06 just before the end of a 15-min shower to 0.24 about 28 min after the shower was turned off, reaching values of 0.33–0.43 beyond 1 h. In a laboratory with manipulated ventilation, F_B followed similar trends and increased to 0.41–0.69 after more than 1 h (Fitzgerald et al., 1997). In longer-term measurements corresponding with multi-day exposure periods, F_H at assumed steady state is generally in the range 0.4–0.5 (NRC, 1991; EPA, 2003). Using an average value of F, the equilibrium equivalent concentration

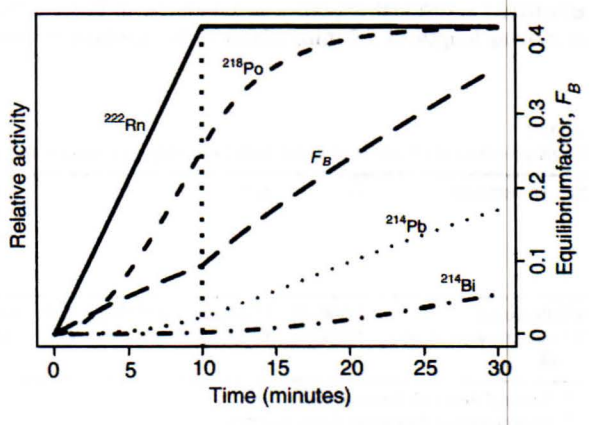

Fig. 6. Relative activities of radon-222 and decay products in a hypothetical sealed room, neglecting effects of ventilation, attachment to aerosols, and deposition onto surfaces.

(International Commission on Radiation Protection (ICRP), 1994) of airborne Rn decay products can be estimated for showering and for whole-house conditions

$$\text{Equilibrium equivalent concentration}(\text{Bq m}^{-3}) = C_T \times F_{\text{average}} \times {}^{222}\text{Rn}_{\text{water}}\text{activity}(\text{Bq m}^{-3}) \quad (2)$$

If exposure time is known, then the alpha energy contribution from each source may be estimated by multiplying equilibrium equivalent concentration by exposure time and the factor 0.00556 μJ m^{-3} per Bq m^{-3} of equilibrium equivalent concentration (ICRP, 1994). Although shower exposure varies in a linear fashion with Rn in water activity (Fitzgerald et al., 1997), C_T and F vary in a non-linear fashion with shower length and time prior to opening the bathroom door. If the door is opened quickly after the water is turned off, Rn and its progeny will escape from the room while F_B remains low, even though Rn activity will be near its peak. On the other hand, a long period of exposure before the door is opened (~1 h) would allow F_B to approach average steady-state conditions. From data published elsewhere (Fitzgerald et al., 1997), an estimated average F_B between 0.05 and 0.15 (here estimated at 0.1) is consistent with a ~10-min shower followed by ~10 additional min before opening the door. It must be emphasized that variability in shower length adds additional uncertainty to exposure estimation, not considered here, in addition to uncertainty related to ventilation and the reactivity of Rn progeny ions. Finally, this simple model only provides estimates of airborne Rn progeny exposure. Parameters necessary for estimating actual dose to the lungs, including aerosol particle size and humidity, are beyond the scope of these field measurements, so the data do not provide a basis for dosimetric calculations (e.g. Fitzgerald et al., 1997).

Nevertheless, it is possible to compare the estimates to the expected whole-house Rn progeny exposure derived from water using $C_{T,H}$ of 0.55×10^{-4} (NRC, 1999), F_H of 0.4 (EPA, 2003), and 70% of time spent at home (EPA, 2003) and treating average inferred whole-house conditions as a background that underlies the entire period of occupancy, including showering. Using an average of 0.98 showers per person per day (Wilkes et al., 2005), estimated additional exposure is presented in Table 3 for a shower length of 7.2 min, which is the median of two geometric mean shower lengths for United States residents (6.8 and 11.3 min; Wilkes et al., 2005) and an Australian median estimate (7.2 min; Burmaster, 1998), and 10 additional minutes before opening the door. Thus, the 40-min. $C_{T,B}$ values (Table 2) were converted into 17.2-min. transfer coefficients. This was accomplished by (1) assuming that the occupant would be exposed to the maximum level of Rn at the time the water was turned off; (2) modeling the increase of Rn as linear using the median rate of increase during the 20-min shower; and (3) modeling Rn escape from the room as exponential (Hess et al., 1982). The escape constant was determined from the median of the median Rn escape obtained from each room (36% Rn reduction per 5 min). To convert to the 17.2-min scenario using this non-linear approach, the 40-min transfer coefficients were divided by 2.26. Applying this transfer coefficient to the occupancy and equilibrium assumptions described above suggests that the above scenario could result in a geometric mean of approximately 5.6% increased exposure to Rn progeny derived from water relative to the whole-house transfer coefficient (Table 3). This is generally consistent with the 8–9% estimated increase from showering in one experimental house (Datye et al., 1997). Per unit time (Eq. (2)), showering provides 3.3 times as much alpha exposure, on average, from water-derived Rn decay products than overall average conditions in the house. The large two-sigma interval produced by these calculations (Table 3) indicates the significant individual characteristics of homes that influence Rn exposure, described above, as well as the long tail in the upper quantiles of the lognormal distribution.

5. Conclusions

During and after showers, significant airborne Rn elevation is seen in bathrooms receiving water with Rn activities above the alternative maximum contaminant level of 148 Bq L^{-1} (4000 pCi L^{-1}) proposed by the EPA. However, at the one house using a treatment system for Rn in water, there was no significant increase of Rn in bathroom air. Although most water supplies containing Rn activity over 148 Bq L^{-1} exhibited a significant increase in airborne Rn associated with shower use, the level of airborne Rn in

Table 3
Exposure estimation from Rn in water from showering and house-scale exposure. Point estimates are geometric means

Scale of exposure	C_T	±2σ	F	Annual exposure time (h)	Annual alpha energy contribution per Bq L^{-1} Rn in water (μJ h m^{-3})	±2σ	Increase due to showering (%)	±2σ
Whole-house	5.50E-05	(5.02E-08–6.03E-02)[a]	0.4[b]	6132[c]	0.750	(7.99E-04–712)		
7.2 min shower + 10 min off	7.28E-04	(1.35E-04–3.94E-03)	0.1	103[d]	0.042	(7.71E-03–0.226)	5.6	(0.03–965)[e]

[a] National Research Council, 1999.
[b] Environmental Protection Agency, 2003.
[c] 70% of time spent at home (EPA, 2003).
[d] Based on 7.2 min in shower and 10 additional minutes before opening door.
[e] Assuming that bathrooms and their corresponding houses are at the same quantiles on the $C_{T,B}$ and $C_{T,H}$ distributions.

bathrooms is not strongly related to Rn in water activity due to variability in ventilation characteristics between houses, room size and water flow rates. Relative to the contribution from water to whole-house Rn, estimated by the $\sim 10^{-4}$ transfer coefficient, it is suggested that showering contributes a few percent of additional Rn progeny exposure, and is dependent on uncertain estimates of exposure time (shower length and after-shower length in a closed bathroom) and F_B. Relative to Rn-from-water exposure generated at longer temporal and larger spatial scales, short-term shower exposure is highly individualized not only because of variations between houses, but also due to variations in shower length and the time of day that showers are taken, that is the length of time that water used for showering has been stored in pipes, the water heater, or the well bore.

Only one of several common household exposures in which occupants are close to water uses is documented. For example, dishwashing and laundry may also generate additional incremental increases in Rn progeny exposure over that suggested by $C_{T,H}$ that will similarly depend on exposure time, location, ventilation, room volume and water volume. However, it should be emphasized that in spite of these upward-revised estimates of Rn progeny exposure due to water, the findings do not substantively affect existing interpretations that the largest source of Rn progeny exposure for the overall population is from Rn drawn directly into houses from rock and soil rather than Rn from water supplies (NRC, 1999; EPA, 2003). The findings are of greatest relevance to (1) residents for whom most Rn exposure comes from water (i.e. high Rn-from-water contribution, low Rn-from-soil contribution; Folger et al., 1994), and (2) residents exposed to somewhat elevated indoor Rn, such as levels close to the EPA action level of 148 Bq m^{-3} (4 pCi L^{-1}). In the second case, considering short-term Rn exposure from water in addition to whole-house Rn-from-water exposure may cause such residents to exceed a nominal exposure level such as that suggested by the EPA action level.

Acknowledgments

This study was supported by Duke Cancer Research; the North Carolina Department of Environment and Natural Resources, Division of Water Quality, Aquifer Protection Section; and a Geological Society of America student Research Grant. This work was partly supported by US Department of Agriculture Cooperative State Research Education and Extension Service Grant NCW-2006-03956. We thank Bill Field, Derek Lane-Smith, and Daniel Steck for discussions that significantly improved the quality of this manuscript. We also thank Richard Wanty and one anonymous reviewer for their helpful comments, and Ron Fuge for editorial handling.

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