Assessment of health risk due to exposure of radon and its daughter products in the lower atmosphere

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Radon concentrations were measured in the lower atmosphere in Garhwal Himalaya, India by using LR-115 type II, plastic track detector. The annual geometrical mean values of radon were found to be 104 Bq/m^3 and 123 Bq/m^3 inside the cemented and mud houses, respectively. The mean value of inhalation dose rate due to indoor radon was found to vary from 4.2 mSv/y to 5.2 mSv/y, for cemented and mud houses respectively. The radon values were observed higher in mud houses which were constructed with the soil and local stone obtained from the area. The observed values were higher than the international recommended values. Based on the available data, the annual effective dose and the risk factors due to environmental exposure of radon and its daughter products have been calculated by using different dosimetric models. The estimated risk of lifetime lung cancer due to environmental radon exposure for a total population of study area, starting at age 1 was calculated as 0.0068 or 0.68%. The mean relative loss of life expectance was estimated as 0.26% for chronic exposure at the measured radon level.

EXPOSURE of persons to high concentrations of radon and its short-lived progeny for a long period leads to pathological effects like the respiratory functional changes and the occurrence of lung cancer[1]. Jacobi[2] indicates that inhalation of short-lived radon daughters seems to be the most important component of the radiation exposure of the population from natural sources. According to an estimate, radon gas may be the major source of public radiation exposure, perhaps accounting for between 5 and 20% of all lung cancer deaths[3]. Recently, the United States Environmental Protection Agency (EPA) estimated that 20,000 deaths occur annually from radon-induced lung cancer[4]. Based on updated dosimetric models[5], this estimate can be revised downward by approximately 25%. Radon decay products rather than radon itself are the active species responsible for the health hazard. In addition, a large body of human epidemiological data on occupational exposure to radon and its progeny, at the resulting elevated lung cancer risk, yielded quite consistent risk factors down to doses encountered in some dwellings[6].

The lung dose equivalent due to inhalation of the short-lived radon decay products cannot be measured directly, and so, must be inferred from the application of a dosimetric model along with measurement of the inhaled activity. Dosimetric modelling of radon decay products exposure has presented special problems to the investigators in that the dose estimated depends upon the assumptions used in determining the activity deposition in various regions of the lung. Recent improvements in dosimetric modelling, combined with a growing volume of measured data for various exposure conditions, have advanced lung modelling to a point where its use has become widely acceptable. Due to the lack of hard epidemiological data, most of the current risk estimates for environmental exposures to radon have been obtained using dosimetric modelling[7]. In this paper, the dose and the risk factors due to environmental exposure of radon and its progeny in Tehri Garhwal have been calculated based on the measured data by using different dosimetric models.

Radon in indoor and outdoor atmosphere has been measured by using LR-115 plastic track detector. The detector films attached on the glass slide are suspended inside the room at a height of about two meters from ground floor. After an exposure time of three months, films are removed and etched in 2.5 N NaOH solution for 2 h at 60°C in constant temperature bath. The films are then scanned under an optical microscope for track density measurements. The calibration constant 3.12×10^{-2} tracks cm^{-2} d^{-1} = 1 Bq m^{-3}, determined by Ramola *et al.*[8] was used to express radon activity in terms of Bq/m^{-3}. This exposure cycle has been extended on a time integrated four quarterly cycle to cover all the four seasons of a calendar year to evaluate the annual indoor radon levels.

Table 1 gives the results of radon levels measured in the lower atmosphere in Tehri Garhwal. The resulting concentration of short-lived radon daughters, expressed in terms of the equilibrium-equivalent radon concentration (EEC_{Rn}), is related to the activity concentration, A_{Rn}, of radon by the relation[9]:

$$EEC_{Rn} = FA_{Rn},$$

where F is an equilibrium factor. Based on recommendations of ICRP, the equilibrium factor for radon daughters in indoor air is in the range 0.3 to 0.6, which is assumed to be 0.45 for the mid point of the range of reported values. The mean equilibrium factor in outdoor air is 0.7 and somewhat higher than indoor air[7].

The estimated dose due to radon and its daughters is calculated by the dose conversion factor[9].

$$1 \text{ Bq h/m}^3 \; EEC_{Rn} = 1.0 \times 10^{-5} \text{ mSv}.$$

The inhalation dose rate due to indoor radon is found to vary from 2.2 mSv/y to 7.2 mSv/y and 2.6 mSv/y to 9.7 mSv/y in cemented and mud houses respectively, with mean value of total dose being 4.2 mSv/y and 5.2 mSv/y (Table 1) respectively.

The mean residence probability of individuals living in Tehri Garhwal was calculated based on the questionnaire filled by the residents of the surveyed houses. The calculated residence probabilities were found 0.60, 0.22 and 0.18 in private homes, other buildings and outdoor respectively, which correspond to mean residence times of 5200, 2000 and 1560 h each year in these areas. On the basis of this data a single residence model, which yields an annual equilibrium-equivalent exposure (E) in Bq h/m^3, is formulated as:

$$E = 5200\ h \times C_{in} + 2000\ h \times C_{ie} + 1560\ h \times C_o,$$

where C_{in}, C_{ie} and C_o are time average mean values of equilibrium-equivalent concentration of radon in air of private homes, other buildings and outdoor air respectively. The estimated mean annual exposure to radon daughters from the different residential areas are listed in Table 2. The calculated annual exposure for indoor radon values were higher than the ICRP recommended levels[9]. These values may vary for individuals, depending on the life-style of the inhabitants and the exposure time indoor. It is expected to be higher for house-bound women, particularly in mud houses where they spend most of time in indoor activities.

At attempt is made to calculate the lung cancer risk due to exposure of radon in the houses of Tehri Garhwal. The calculation is based on a model developed by Harley and Pasternack[10]. The following assumptions were made in the model: 1, It is assumed that no lung cancers occur before age 40. 2, It is assumed that no radiation-induced lung cancer occurs within 5 years of exposure, i.e. there is a 5-year latent period. 3, Repair mechanisms are assumed, such that the lung cancer probability from a given exposure decreases exponentially with time (after 5 year latent period), with a 20 year half time. 4, The contributions to life time risk are ignored after the person reaches age 85. 5, It is assumed that the risk per WLM is 40% greater for the general population than for miners, due to the combined effects of differences in average breathing rates, average lung sizes, aerosol particles sizes and in the fraction of unattached radon daughters (greater in the general environment than in mines due to lower dust levels).

Table 1. Calculated annual dose due to exposure of radon and its daughters in the houses of Tehri Garhwal

Type of house	Number of measurements	Annual mean % of radon conc. (Bq/m^3)	Annual dose (mSv/y)		
			Minimum	Maximum	Mean
Cemented houses	57	104 ± 6.2	2.2	7.1	4.2
Mud houses	42	123 ± 6.9	2.6	9.7	5.2

Table 2. Estimated mean annual exposure to radon daughters in indoor and outdoor environments

Contribution from residence	Equivalent-equilibrium exposure (Bq h/m^3) × 10^3	Potential alpha energy exposure	
		mJh/m^3	WLM
Cemented houses			
Indoors at home	267	1.47	0.43
Indoors elsewhere	103	0.57	0.16
Outdoors	49	0.27	0.078
Total	419	2.31	0.668
Mud houses			
Indoors at home	339	1.86	0.54
Indoors elsewhere	127	0.70	0.20
Outdoors	49	0.27	0.078
Total	515	2.83	0.818
Mean value	467	2.57	0.743
ICRP recommendations			
Indoors at home	90	0.51	0.14
Indoors elsewhere	23	0.13	0.036
Outdoors	4	0.002	0.0064
Total	120	0.66	0.19

Conversion factor: 1 Bq h/m^3 = 5.52 × 10^{-6} mJh/m^3 = 1.60 × 10^{-6} WLM.

The exponential factor given in assumption (3), corresponds to the assumption of a 'decrease in rate of risk expression due to repair, cell death or unspecified mechanisms', with a 20 year half time[11]. With this choice of half time, the risk estimates match the results of the studies of miners, where it is found[11] that the incidence of lung tumours is higher when first exposure to radon daughters occurs at 40 or 50 years of age, compared to first exposure at age 20. Since lung tumours do not usually appear before age 40, exposure at the earlier age allows more time for decay of the risk. However, the model could be equivalently adjusted to match the observations by assuming that the vulnerability is greater if exposure occurs at a later age[10].

With these assumptions, the probability of lung cancer induction for a person between the ages of 40 and 85 is taken to be:

$$A(t, t_o) = 1.4 \times 10^{-6} \times N(t_o) \times P(t, t_o) \times e^{-T \ln 2/20},$$

where $A(t, t_o)$ = the probability of lung cancer induction at age t, due to an exposure to N WLM occurring at age t_o; $N(t_o)$ = the number of WLM of exposure at age t_o, where exposure is taken to be in a typical indoor atmosphere; $P(t, t_o)$ = the probability that a person alive at age t_o will still be alive at age t; $T = t - t_o$ = the time interval since the exposure.

Based on the above model, the estimated risk of lifetime lung cancer due to environmental exposure to radon in Tehri Garhwal for different age groups is given in Table 3. The probability of lung cancer induction was higher for the persons living in mud houses.

It is seen from Table 3 that the total calculated risk of radon-induced lung cancer for a mean annual exposure of 0.74 WLM per year, for the total population of study area, starting at age 1 is 0.0068, or 0.68%. In other words, a person who is exposed to 0.74 WLM per year for every year of his life has a 0.68% chance of suffering a radon-induced lung cancer. Almost all lung cancers are fatal, so this can be taken to represent the approximate lung cancer mortality rate.

The relative lifetime risk of lung cancer from inhaled radon daughters and the air concentration in the study area was calculated based on the ICRP model[9]:

$$R_r/R_o = 0.001\ (\text{Bq/m}^3)^{-1}\ (6 \times C_{in} + 1.5\ C_{ie} + 1.3\ C_o),$$

where C_{in}, C_{ie} and C_o are the annual mean values of ^{222}Rn daughters concentrations in indoor air at home, in indoor air elsewhere (e.g. the working place) and in outdoor air, respectively. Radon values are expressed in terms of equilibrium-equivalent radon concentration in Bq/m^3.

The relative lifetime risk of lung cancer in the study area was calculated as 0.43 and 0.53 per Bq/m^3 for people living in cemented and mud houses. The risk is calculated higher for the people living in mud houses. However, the average value of lung cancer risk is 0.48 for population living in the area. This indicates that the lung cancer risk for a population exposed throughout an average lifetime to the calculated mean equilibrium-equivalent radon concentration is about half of the normal lung cancer risk (without radon daughter being exposed).

The health detriment by lung cancer from inhaled radon daughters can be expressed in terms of the attributable loss of life expectancy. On the basis of the relative excess risk coefficients, the attributable loss of the life expectancy, average over both sexes, is calculated as[9]

$$L_r = 1.4 \times 10^{-4}\ E\ \text{(days)},$$

where L_r, is the attributable loss of life expectancy and E is the annual radon exposure in Bq h/m^3.

Based on this model, the attributable loss of life expectancy was calculated to be 59 days and 72 days for the population living in cemented houses and mud houses respectively. An average value of attributable loss of life expectancy was calculated to be 66 days for the total population in the study area, which is comparable to the value (16 days) calculated by ICRP[9] for an exposure of 15 Bq/m^3 for the reference population living in different parts of the world. Compared with a mean life expectancy, L_o, of about 70 years for the total population in the study area, the mean relative loss of life expectancy is calculated as $L_l/L_o = 0.0026$ or 0.26% for chronic exposure at the measured radon level.

From the studies of miners, it is clear that an exposure to high concentration of radon daughters leads to an increased incidence of lung cancer. The study has now focused on the normal indoor environment and efforts are being made to use the experiences for miners to estimate the incidence of radon-induced lung cancer among general population. Such estimates cannot be very precise, in part because the miners studies do not lead to well-determined relationship between the rate of cancer induction and the exposure levels. In particular,

Table 3. Estimated lifetime lung cancer risk due to radon exposure at different ages

Age at first exposure	Probability of lung cancer induction × 10^4		
	Cemented houses	Mud houses	Mean
1	61	74	68
10	61	74	68
20	51	63	57
30	51	63	57
40	30	37	33
50	18	22	20
60	8.7	11	10
70	2.6	3.1	2.8

there are disparities in the results of studies for different groups of miners. These disparities may arise from the difficulties in obtaining accurate crucial data[11], such as the year-by-year exposure history over the working lifetime of the individual miners.

Further, there are problems in using results obtained for miners at relatively high radon exposure levels for the prediction of the effects of radon at much lower levels encountered in the normal indoor environment. Here arises the problem of extrapolating from large dose level to small dose level. The generally adopted solution is to assume linearity although this assumption may overestimate or underestimate the true risk.

The difficulty with estimating risks at lower radon level is that epidemiological data do not provide a direct evidence of health effect at the moderate exposure involved, in particular among the general public. On the other hand, the current understanding of dose–response relationship, especially for the alpha radiation from radon decay products, suggests the prudent course of presuming that the proportionately small lung cancer risks estimated for these exposures actually occur. A complication is that, based on the epidemiological evidence, it is thought that the risk from radon is to a greater or lesser degree synergistic with that of smoking, so that most of the risk estimated for radon exposure of the general public occurs among smokers, who even without radon incur much greater risk (25% or greater chance of dying from smoking one and half pack of cigarettes per day) knowingly and voluntarily. It is thus ironic that exposure at 150 Bq/m^3 of radon is often said to be equivalent in risk to smoking half a pack of cigarettes per day, a factor of ten overestimate for those who do not smoke. Still health risk estimates provide one of the main bases for determining what radon concentration might be deemed excessive and hence for focusing research and control efforts.

Finally, the calculated values of dose, lung cancer risk and attributable loss of life expectancy for the present study are totally based on the available models used for similar study at different places in the world. These values are recorded above international recommended level. As such these values cannot be taken as the reference but may be used as guidelines to initiate further studies, especially on real radon-induced lung cancer incidence and epidemiological studies to formulate a new model for this area.

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ACKNOWLEDGEMENTS. The authors (R.C.R. & R.B.S.R.) thank BRNS, DAE, Govt of India for financial assistance.

Received 1 July 1997; revised accepted 25 September 1997

Single strand conformation polymorphism profiles with biotinylated PCR products to detect mutations in *rpoB* gene of *Mycobacterium tuberculosis*

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A fragment of the *rpoB* gene, including the region shown to be involved in rifampicin resistance, was amplified from 15 rifampicin-resistant and 6 rifampicin-sensitive clinical isolates of *Mycobacterium tuberculosis* by the polymerase chain reaction (PCR). One of the primers, employed in PCR, was biotinylated. The biotinylated strand of the PCR product was separated from the unbiotinylated strand using streptavidin magnetic beads. Both the strands were subjected to single strand conformation polymorphism in polyacrylamide gel electrophoresis. The DNA bands were silver stained to study their migration pattern. A shift in the migration of either of the strands of the test strain compared to the strands from a control rifampicin-sensitive strain was considered as indicative of resistance. This strategy was found to ease the visualization of shift in the migration of the strands in 17 of 21 samples and thereby detection of mutations.

SINGLE strand conformation polymorphism (SSCP) is one of the usual procedures to detect mutations[1,2]. It is based on the fact that separated strands of DNA adopt a folded