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Radon and Energy Efficient Homes

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RADON AND ENERGY EFFICIENT HOMES

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ABSTRACT

Radon and its daughters in indoor air are presently responsible for dose equivalents of about 31 mSv/year (3 rem/year) to parts of the respiratory tract. Linear extrapolation from the dose response values of uranium miners heavily exposed to radon and its decay products would suggest that almost all lung cancers in the non-smoking population are caused by environmental ^{222}Rn. Using epidemiological data on the types of lung cancer found in non-smokers of the general public as compared to the miners, a smaller effect of low level radon exposure is assumed, which would result in a lung cancer mortality rate due to radon of about 10 deaths per year and million or 25 % of the non-smoker rate.

Higher indoor radon concentrations in energy effficient homes mostly caused by reduced air exchange rates will lead to a several fold increase of the lung cancer incidence from radon. Based on the above assumption, about 100 additional lung cancer deaths/year-million will result both from an increase in radionuclide concentrations in indoor air and a concomitant rise in effectiveness of radiation to cause cancer with higher exposure levels.

Possibilities to reduce indoor radon levels in existing buildings and costs involved are discussed.

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1 INTRODUCTION

Conservation is rightly considered the most important single measure preventing both the fast depletion of our fossil fuel reserves and the increasing pollution of our environment. Although the remark " the cleanest energy is energy conserved " still holds, some popular conservation efforts to save heating energy in dwellings may add a substantial radioactive burden to the general population.

In this report, the literature on radon and its daughters in the indoor environment is reviewed. Dose equivalents to exposed tissues of the respiratory tract for people living in a concrete/ brick dwelling are estimated for conventional and energy efficient homes. Like most risk assessments of low level radiation, the final effort to quantify the effect of radon by giving a death rate from lung cancer attributable to indoor air has to rely on extrapolation from high exposure data. Nevertheless, the large risk anticipated calls for further studies in this field and may also lead to the conclusion, that the slight, but much feared burden due to man made radioactivity could be more than compensated for by controlling environmental radioactivity.

2. FORMATION AND MEASUREMENT OF RADON AND ITS DAUGHTERS

From the 4 naturally occuring radioactive chains of ^{238}uranium, ^{235}uranium, ^{232}thorium and ^{237}neptunium, only the first 3 survived into our times in considerable amounts. Both uranium nuclides and thorium decay in several steps involving α and β radiation to radon (^{222}radon), actinon (^{219}radon) and thoron (^{220}radon) respectively Figure 1 shows the decay scheme for the most important family, ^{238}U. All 3 radon isotopes produce short-lived daughters wich

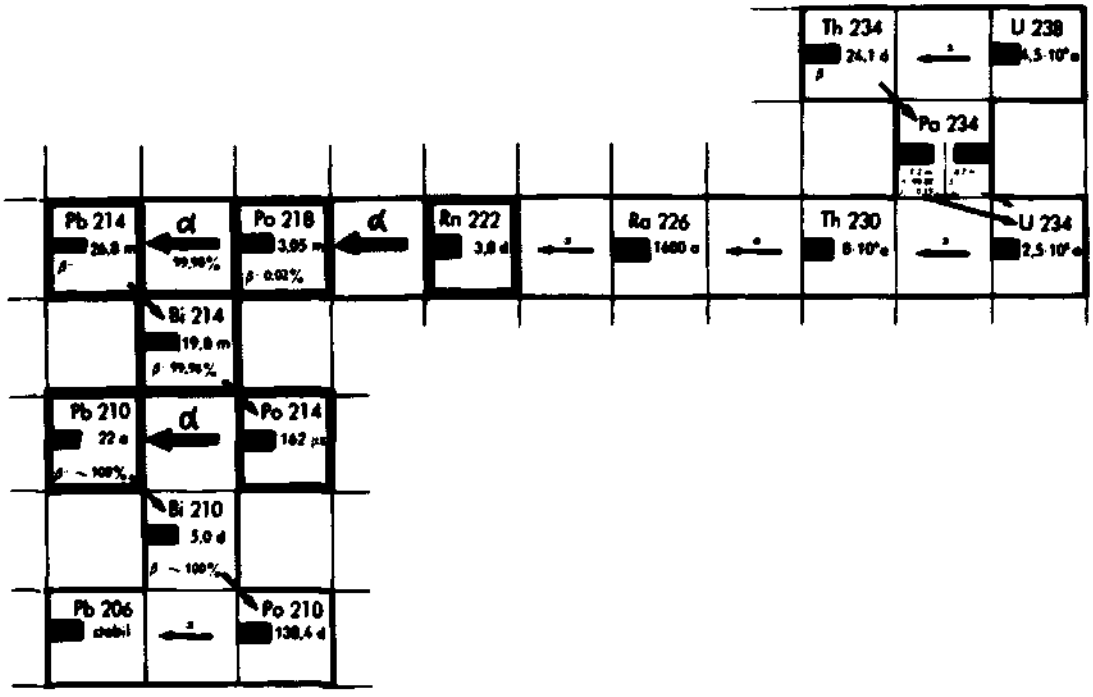

Fig. 1: Decay scheme of ^{238}uranium (modified from 1)

subsequently undergo 2 more α decays and 2 β decays before they turn into relatively long-lived or stable lead isotopes. The energies involved are in the range of 4 - 9 MeV for α, the β energies range from .1 to 2.3 MeV.

Measurements of radon are routinely performed for the purpose of radiation protection in uranium mines, for uranium exploration and more recently for earthquake prediction. Some early methods developed

for determining the air concentration of ^{222}rador consist in collecting the airborne daughters RaA (^{218}Po), RaB (^{214}Pb) and RaC (^{214}Bi) on a filter and subsequently counting the deposited activity (2, 3, 4). There are also methods developed to measure radon but not its daughters already formed before the radon enters the detection space by placing a permeable membrane in front of a cup containing a plastic track detector at the closed end. (5). This technique has the additional advantage that it also excludes the short-lived ^{220}Rn and ^{219}Rn isotopes from reaching the counting area (6).

3. RADON IN THE ENVIRONMENT

Uranium and thorium are major sources of radioactivity in the ground. Typical concentrations are 1 - 10 ppm for both Th and U in sandstone, shale or limestone (7). Granite contains from 9 - 12 ppm uranium and 36 - 44 ppm thorium (8). The corresponding activities range from .02 to .15 Bq/g (.5 - 4 pCi/g). The external dose from both nuclides and their decay products if they remain in the ground during decay is about 5.4 mGy/year-Bq/g (20 mrad/year-pCi/g) at a distance of 1 meter above ground and, ingeneral contributes a significant fraction to the natural background radiation.

Apart from special cases in thorium rich areas (9,10), the concentration of ^{222}Rn in air normally greatly exceeds that of thoron and actinon. This is due mainly to the long half live of ^{222}Rn relative to ^{219}Rn and ^{220}Rn. The long half live of the highly water soluble parent nuclide ^{226}Ra may also increase the ^{222}Rn concentration in the biosphere due to transport of Ra to the surface through aquifers (Table 1). The fraction of radon atoms reaching the soil particle surface before undergoing decay is called the emanating fraction of that source. The ability of radon to survive migration is obviously a function of the half live and the permeability of the source matrix. Radon-222 may travel several meters through soil, whereas thoron and actinon will diffuse a few cm and a few mm, respectively. In view of this

Table 1: Half lives of radon isotopes and its parent radium nuclides

^{222}Rn (radon)	3.8 days	^{226}Ra	1600 a
^{220}Rn (thoron)	55.6 s	^{224}Ra	3.64 d
^{219}Rn (actinon)	3.9 s	^{223}Ra	11.4 d

fact, the following estimations deal only with ^{222}Rn. Release estimates of ^{222}Rn for the USA from various natural and anthropogenic sources are shown in Table 2. Radon exists in ground water

Table 2: Estimated release of ^{222}Rn in the United States in 1978 (modified from 11)

source	Bq/year	kCi/year
soil + evapotranspiration	$47'000 \times 10^{14}$	12'880
uranium industry	148×10^{14}	400
phosphate fertilizer	17.76×10^{14}	48
natural gas: domestic uses	$.11 \times 10^{14}$	.3
industrial uses	4.07×10^{14}	11
building materials, potable water	5.60×10^{14}	15
coal fired power plants	$.19 \times 10^{14}$	.5

at concentrations which are typically much greater than its immediate precursor ^{226}Ra and its daughters. This is due to the high mobility of the noble gas and the chemically active nature of the daughters leading to a fast plate out on surfaces of the aquifer. Measurements made in the USA showed that about 50 % of the water samples contained less than 74 Bq/l (2pCi/l) but 5 % contained more than 370 Bq/l with 2 % of the values from Maine and New Hampshire exceeding 3.7 kBq/l (12).

Radon also diffuses with natural gas into the wells and is released from unvented stoves and heaters at a rate of .74 Bq (20

pCi)/l natural gas.

Most construction materials besides wood, glass and steel contain uranium, thorium and radium. Typical values range from 1 to 10 ppm uranium and thorium but can be much higher in granite, alum shale and gypsum boards, a by-product of phosphate mining.

4. RADON IN DWELLINGS

4.1. Sources

The main sources leading to a build up of radon in dwellings are building materials, diffusion from the soil via cracks, joints, sewers and loose fitting pipes into the basement, tap waters from aquifers, unvented natural gas stoves and space heaters.

4.2. Present indoor concentrations: mean and extreme values

Data on radon levels in buildings were reviewed in the 1977 UNSCEAR Report (13). As a representative value, 37 Bq/m^3 (1 pCi/l) was adopted as the average ^{222}Rn concentration indoors. The equilibrium factor (ratio of Rn daughter concentration to their concentration in radioactive decay equilibrium with Rn gas) was assumed to be .5. This corresponds to a concentration of .005 WL* (14). The houses in this and other surveys were located in Florida, Tennessee, Boston, New York and New Jersey, Canada (17), Sweden (16,19), Norway and Great Britain. Higher values of 74 Bq/m^3 (2pCi/l) were reported in Norwegian houses with concrete walls (15), more than 370 Bq/m^3 (10 pCi/l) in one third of the houses built on lands reclaimed from phosphate mining (18) and in Yougoslavia (peak values of 740 Bq/m^3 or 20 pCi/l) (41).

Although soils with high radium concentrations like those containing uranium tailings are responsible for the highest values reported in litera-

* WL: working level, the radiation level of 100 pCi/l radon in equilibrium with its daughters. 1 WL = 1000 mWL

ture, given the small number of homes affected, widely used construction materials with radium activities only slightly above average tend to have a bigger impact on the general public. The widespread use of concrete and bricks in dwelling construction in moderate and cold climate areas in Europe increases indoor levels to 48 - 75 Bq/m^3 (1.3-2 pCi/l)(15). Between 1930 and 1975, alum shale based aerated concrete was a popular building material in Sweden. This resulted in thousands of homes having average radon concentrations of 270 Bq/m^3 (7.3 pCi/l) with peak values reported at 500 Bq/m^3 (13.5 pCi/l)(18).

After construction of a dwelling the amount of radon and its precursors in soil, building materials, natural gas and tap water has to be taken as constants. However, the emanating fraction of radon from the soil near the basement may vary to a great extent depending on the pathways present through pipe fittings, floor drains, joints and the porosity of the basement walls and floor.

4.3. Estimation of radon levels in energy efficient homes

The steady increase in energy costs lead to major efforts to decrease consumption. Since space heating is responsible for more than 50 % of our energy consumption, insulation of buildings in order to conserve energy is probably the largest conservation program now undertaken in many nations. This measure may lower the air exchange rate considerably and results in trapping radon and its daughters for extended times inside buildings. Theoretical and experimental data suggest that radon levels are inversely proportional to the ventilation rate, the number of air changes per hour (20). Typical values for conventional houses are .75 to 1 air changes per hour whereas a home with energy saving windows and door frames may undergo only .3 - .1 air changes/hr (14, 19). Therefore, insulation would increase radon levels by a factor of 2 to 10. If the relatively low proportion of wood in Central European building materials is taken into account, an estimate of 55 Bq/m^3 (1.5 pCi/l) radon in conventional houses and of 166 Bq/m^3 (4.5 pCi/l) in energy efficient homes seems appropiate.

5. DOSIMETRY OF RADON AND RADON DAUGHTERS IN THE LUNG

The critical organ receiving most of the short ranged α radiation from radon and radon daughter decay is the respiratory tract (21). Up to 1951, evaluations of the hazard associated with radon neglected that the radiation dose due to decay products of radon present in the air is likely to exceed the dose due to radon itself and to disintegration products formed while the radon is in the lung. The charged radon daughters bind immediately to dust particles. The isotopes and their decay energies in the chain from ^{222}Rn to ^{210}Pb are listed in Figure 1. For internal radiation dosimetry, only α radiation is commonly considered. The longer range and lower biological effectivness of γ and β components result in negligible dose equivalents from this radiation to the lung tissue (21). Because of its long half live of 22.3 years, ^{210}Pb (RaD) is considered stable. Those 2 simplifications lead to potential α energies per atom deposited of 13.7 MeV for ^{218}Po and 7.7 MeV for ^{214}Pb and ^{214}Bi. The half live of ^{214}Po (163.7 μsec) is so short, that it can only decay in the lung if its precursor ^{214}Bi is inhaled.

5.1. Deposition and retention models

The Task Group Lung Model of the ICRP published 1966 (22) is used for most calculations of dose from radioactive materials in the respiratory tract. The new deposition and retention model of ICRP Publication 30 (23) resulted in many changes in values of deposition and clearance parameters. Figure 2 shows deposition of particles dependent on their size in the following 3 regions of the respiratory system: nasal passage (N-P), trachea and pulmonary tree (T-B), pulmonary parenchyma (P). Figure 4 shows on the right the 3 regions subdivided into compartments associated with a particular pathway of clearance (a -j). The table on the left gives half lives of clearances and fractions cleared by

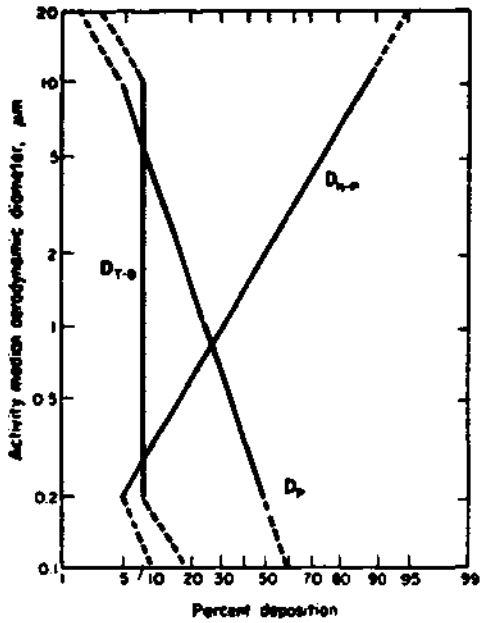

Fig. 2: Deposition of dust in the respiratory system. The percentage of activity or mass of an aerosol which is deposited in the N-P, T-B and P regions is given in relation to the Activity Median Aerodynamic Diameter (AMAD) of the aerosol distribution. The model is intended for use with aerosol distributions with AMADs between 0.2 and 10 μm and with geometric standard deviations of less than 4.5. Provisional estimates of deposition further extending the size range are given by the dashed lines. For an unusual distribution with an AMAD of greater than 20 μm, complete deposition in N-P can be assumed. The model does not apply to aerosols with AMADs of less than 0.1 μm. from (23)

a specific pathway for 3 different classes of materials. These classes are based on retention of the material in the pulmonary region which is dependent on the chemical form, the solubility

		Class					
		D		W		Y	
Region	Compartment	T day	F	T day	F	T day	F
N–P	a	0.01	0.5	0.01	0.1	0.01	0.01
(D_{N-P} = 0.30)	b	0.01	0.5	0.40	0.9	0.40	0.99
T–B	c	0.01	0.95	0.01	0.5	0.01	0.01
(D_{T-B} = 0.08)	d	0.2	0.05	0.2	0.5	0.2	0.99
	e	0.5	0.8	50	0.15	500	0.05
P	f	n.a.	n.a.	1.0	0.4	1.0	0.4
(D_P = 0.25)	g	n.a.	n.a.	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1000	0.9
	j	n.a.	n.a.	n.a.	n.a.	∞	0.1

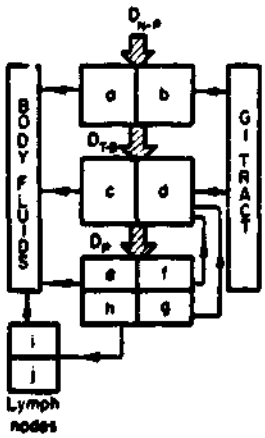

Fig. 3: Mathematical model used to describe clearance from the respiratory system. The values for the removal half-times, T_{a-i}, and compartmental fractions, F_{a-i}, are given in the tabular portion of the figure for each of the three classes of retained materials. The values given for D_{N-P}, D_{T-B} and D_P (left column) are the regional depositions for an aerosol with an AMAD of 1 μm. The schematic drawing identifies the various clearance pathways from compartments a-i in the four respiratory regions, N-P, T-B, P and L.
n.a. = not applicable. from (23)

of the nuclides. Due to the fast decay of the radon daughters, the activity deposited will always behave like a Class D compound. Pathways f, g and h with h-dependent i and j are too slow to contribute to the clearance in this situation and are not considered here. The long mucus transit time in the respiratory and terminal bronchiols compared to larger bronchiols results in the the decay of almost all radon daughters before moving to the higher bronchial regions. Therefore, Altshuler recommended for radon dosimetry to treat the respiratory and terminal brochiols with the alveoli (pulmonary region). Removal of the radon daughters deposited in the epithelium of the trachea and the bronchial tree is accomplished by absorption across the walls and ciliary transport into the gastro-intestinal tract (24).

Biological half lives for radon daughters are in the range of 10 minutes to 5 hours in the T-B region and 6 - 60 hours in the pulmonary region (21,22,24,28). Such a rapid clearance is evidently only possible if the adsorbed daughters leave the the dust particles they were originally attached to. Thus the radon daughters seem to be removed easily from the dust in liquid and to behave like soluble substances. Figure 3 provides biological half lives of 12 hours in the pulmonary region, 14.4 minutes for 95% of T-B (c) and 4.8 hours for d (5% of the T-B deposition).

The first daughter of ^{222}Rn, ^{218}Po (RaA) exists only 10 - 50 seconds as a free, unattached ion. The fraction of free ^{218}Po is in the range of 1 - 10% in representative environmental air (28,25). The dust particles collecting the radioactivity are assumed to have an activity median aerodynamic diameter (AMAD) of 0.1 µm. Figure 2 gives only a provisional estimate of fraction of deposition for this size particles of .6 for the pulmonary region and .1 for the tracheo-bronchial region. A recent dose calculation by McPherson (21) is based on work of Altshuler (24) and assumes that, if free ions are included, deposition fractions for ^{218}Po are .47 for the P region and .07 for the T-B region. For the polonium daughters, the values are .5 (P) and .01 (T-B). Table 3 gives estimates of the α energy in the lung tissue for the radon daughters using fraction values of .6 (T-B) and .33 (P)

Table 3: α energies from radon daughters to regions of the respiratory tract

Rn daughter	respiratory region	absorbed α energy in MeV per Bq (pCi) deposited	absorbed α energy in MeV per Bq (pCi) inhaled
^{218}Po	P	3'475 (128.6)	2'086 (77.2)
	T-B	1'608 (59.5)	54 (2)
^{214}Pb	P	16'757 (620)	10'054 (372)
	T-B	2'546 (94.2)	85 (3.1)
^{214}Bi	P	12'757 (472)	7'654 (283)
	T-B	5'865 (217)	195 (7.2)

respectively and Mc Pherson's equations (21).

5.2. Calculation of dose equivalent factors

Due to the short range of α particles in tissue (47 µm for ^{218}Po and 71 µm for ^{214}Po), only a small mass of tissue in the tracheobronchial region is affected. A total surface area of 417 cm^2 yields 2 g and 3 g of tissue irradiated by ^{218}Po and ^{214}Bi, repectively. The much larger surface of the pulmonary region results in 540 g tissue affected by the α radiation in this compartment. To calculate the average dose over the depth of α penetration, the values from table 3, right column, are converted into Sv/Bq (rem/pCi) by dividing the energy deposited by the mass of the tissue affected and by multipying with a quality factor of 10. Table 4 lists the committed dose equivalent factors for ^{222}Rn and its daughters. The value for ^{222}Rn shows clearly that the dose resulting from inhalation of the radon gas is negligible compared to the dose resulting from inhalation of daugthers bound to particles in the indoor air. However, ^{222}Rn is the only nuclide considered here giving a considerable dose to other organs. The partial absorption in the lung results in a dose equivalent factor for the whole body of $.23 \cdot 10^{-9}$ Sv/Bq inhaled (.82 rem/mCi)(26).

Very often, exposure values are given in working level months (WLM), the unit commonly used in uranium mining. One WLM equals

Table 4: Committed dose equivalent factors for ^{222}Rn and its daughters (contribution of daughters formed after deposition is included)

nuclide	Sv/Bq inhaled (rem/mCi)	
	tracheo-bronchial region	pulmonary region
^{222}Rn	$.97 \times 10^{-9}$ (3.6)	$.25 \times 10^{-9}$ (.91)
^{218}Po	39.00×10^{-9} (113.0)	6.2×10^{-9} (23.00)
^{214}Pb	45.00×10^{-9} (167.0)	29.8×10^{-9} (110.00)
^{214}Bi	104.00×10^{-9} (387.0)	22.7×10^{-9} (84.00)

the exposure for 170 hours to a concentration of 1 working level (WL), where 1 WL is defined as any combination of short lived decay products of ^{222}Rn per liter of air that will result in the emission of 1.3×10^5 MeV of α energy during complete decay (27). 1 WL corresponds to 3.7 Bq (100 pCi) ^{222}Rn/l in equilibrium with its daughters.

5.3. Dose calculations

In addition to the committed dose equivalent factors, dose is directly proportional to radon concentration, radon daughter equilibrium factor, breathing rate and fraction of time spent indoors. The following assumptions are used for the calculations of the dose equivalent per year to the respiratory tract:

- indoor ^{222}Rn concentration in Bq/m^3 (pCi/l)

^{222}Rn in soil, building material	air exchange rate (hr^{-1})	
	.75	.25 (energy efficent home)
average	56 (1.5) A	167 (4.5) B
4 x average	222 (6.0) C	666 (18.0) D

- radon daughter equilibrium factor: .5 (14)

- amount of air inhaled indoors per year (reference man) (42)

activity	fraction of time	l/min	m^3/year
resting, sleeping	.5	7.5	1'971
light activity	.3	25	3'942
totally spent indoors	.8	total inhalation	5'913

Bq ^{222}Rn inhaled per year (mCi):

A	3.3×10^5	(.0089)
B	9.9×10^5	(.0266)
C	13.1×10^5	(.0355)
D	39.4×10^5	(.1064)

Dose equivalent H in mSv/year (rem/year):

	tracheo-bronchial r.		pulmonary region	
A	31	(3.1)	9.8	(.98)
B	93	(9.3)	29.2	(2.9)
C	124	(12.4)	38.9	(3.9)
D	373	(37.3)	116.6	(11.6)

Values reported in the literature are mostly in WLM/year. A recent estimate of a OECD workshop on radon assumed that an indoor concentration of .8 pCi/l results in .004 WL. The resulting exposure in .2 WLM/year (27). Since most publications (24,28,29) assume a dose of 1 rad/WLM to the basal cells of the bronchial epithelium, an exposure of .2 WLM/year results in a dose equivalent of 20 mSv (2 rem)/year to this tissue. This value is in good agreement with the above estimate which is based on a stone/brick house having normally higher radon concentration than a wooden house.

6. RISK ESTIMATE FROM EXPOSURE TO ENVIRONMENTAL ^{222}RADON AND ITS DAUGHTERS

Environmental ^{222}Rn and its decay products deliver quite large dose equivalents to parts of the respiratory system. For risk estimates however, it has to be kept in mind that the risk to transform a cell at a given dose is proportional to the number

of cells irradiated. Here, the tissue mass involved is quite small and consists of cells with low mitotic activity.

6.1. Epidemiological data from uranium mining

The intensively reviewed uranium miner data gives risk estimates for high exposures to radon and its daughters (27,29). Besides the still unresolved problem of extrapolating from the risk at high exposures to a prediction of effects at low level radiation, the miner data has several additional shortcomings. At the beginning of operation in the 1940s, the exposures were sometimes underreported, the aerosol characteristics in the mines are quite different from indoor air and the presence of additional carcinogens in mine air plus the previous history of many miners in lung disease prone occupations like hard-rock and coal mining complicates the evaluation of risk further. To make things even more difficult, in thorium rich mines ^{220}Rn (thoron) and its daughters may give higher contributions than ^{222}Rn plus decay products to the overall burden (30). Figure 4 taken from Cohen's report shows the range of results received from U.S., Canadian, Czechoslovakian and Swedish data on miners (31). The line drawn by Archer (32)

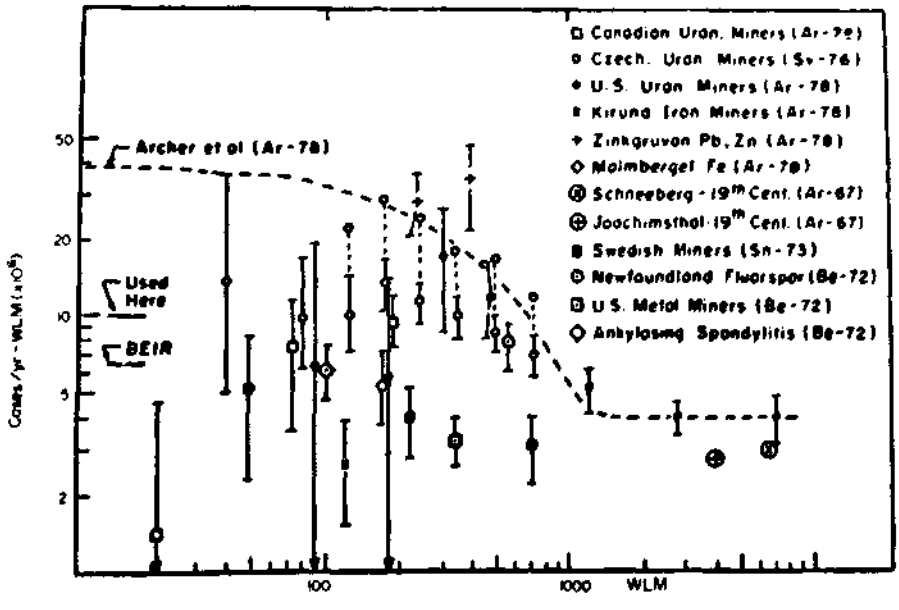

Fig. 4: Lung cancer mortality/year-WLM versus radon exposure in WLM for various groups (from 31).

represents the most conservative interpretation of the data possible. A linear relationship resulting in about 6.5 cases/year-WLM-million is used in the BEIR report (29). On the other hand, an estimate of zero risk for very low levels of exposure cannot be excluded.

6.2. Other epidemiological data

Lung cancer incidence before smoking became widespread gives a life time risk of roughly .1 % for lung cancer (United States, 1930). If a mean life time exposure to radon and its daughters of 12 WLM (60 x .2 WLM/year) would be responsible for all lung cancers in non-smokers, an upper limit for the risk factor at low exposure levels of 10^{-4} per WLM can be drawn (29).Cohen points out that 70 % of the radon induced lung cancers in uranium miners were of the small cell undifferentiated type but only 20 % of the lung cancers occuring early in this century belonged to this category (31). He concludes that only 2/7 of the latter are possibly caused by radon exposure. Epidemiological data also allows us to reject the risk estimate of Archer et al (32) for low level exposure to radon. Their extrapolation predicts a lug cancer rate from radon alone which is about twice as high than the rate of 45 x 10^{-6}/year found for non-smokers (33).

A new model by Harley and Pasternack (34), also based on uranium miner data, takes into account that 1) lung cancer incidence below the age of 40 is very low, 2) a latency period of at least 7 years is commonly observed and 3) age at exposure is critical. Using a risk coefficient of 50 x 10^{-6}/year-WLM, the model also predicts a lung cancer risk which is for early ages bigger than the risk reported for non-smokers.

A case-control study performed in Sweden comparing the type of homes of persons who had died from malignant tumors of the lung with the homes of a control group indicates that the risk of dying from cancer of the respiratory tract is 5 times higher living in a stone home as compared to the risk encountered in a wooden house (43). Although this surprisingly large effect in

a rural study merits further examination, it has to be kept in mind that confounding factors like smoking habits and socio-economic status can influence such studies in an unpredictable way.

From all the data available, a life time risk factor of 3×10^{-5} per WLM for low levels of exposure seems appropiate. With this value, about 30 % of the lung cancer incidence in non-smokers would be attributable to radon. For cumulative exposures above 40 WLM data from Swedish and Czechoslovakian miners suggest a risk factor of 10^{-4}. Using the conversion .1 Sv/WLM (10 rem/WLM or 1 rad/WLM), the cumulative dose equivalent for the basal cells of the T-B region and the lung cancer life time risk for the 4 exposure levels A - D are shown in Table 5. If the values for

Table 5: Yearly and life time exposure, life time risk for lung cancer and cancer cases attributable to radon at different exposure levels.

exposure level	WLM /year	WLM (Sv) in life time	life time risk	lung cancer cases attributable to radon/year-million people
A 56 Bq/m^3	.37	22 (2.2)	.07% *	15
B 167	1.12	67 (6.7)	.67% **	112
C 222	1.5	90 (9)	.9 % **	150
D 666	4.5	270 (27)	2.7 % **	450

* risk factor of 3×10^{-5}/WLM

** risk factor of 10^{-4}/WLM

A and B from the last column are compared to the age adjusted death rates from malignant neoplasms of the lung (35,36) which are 34/year-million for non-smokers and 590/year-million for smokers in this specific US-study and 200 for the whole population per year and million in Norway, it becomes evident that due to the high incidence of lung cancer caused by smoking, the effect of converting the majority of homes from A (air exchange .75 hr^{-1}) to B (.25 hr^{-1}) will, at todays trend of reducing smoking, go undetected. However, the prediction of an annual toll of an extra 100 deaths per million population from lung cancer merits carefull examination of the problem.

A threshold value for lung cancer induced by radon and its daughters could exist at a low level. However, the elevated levels of the energy efficient house produce life time exposures near the values for some groups of uranium miners having well documented risks for lung neoplasms in the order of 5 cases/year-WLM-million (44). Also, theories predicting a threshold for radiation effects cover mostly low LET radiation but not high LET α radiation.

Converting an stone home in a high radon area to an energy efficient house (D) results in an additional health risk comparable to the risk encountered by a smoker smoking ½ - 1 pack of cigarets per day (35).

Several reports exist on the influence of age, sex and mouth breathing instead of nose breathing. However, these effects are small compared to the uncertainities in the response estimates and are not considered here.

7. RECOMMENDATIONS FOR REDUCING RADON LEVELS IN ENERGY EFFICIENT HOMES

If for existing buildings, soil and building material concentrations are taken as constants, there are only two low cost ways to reduce exposure to radon and its daughters considerably. The first solution is to reduce emanation of radon (release from soil and walls), the second using the outgoing air to heat the incoming air in a heat exchanger, thus achieving similar energy savings at an unchanged or even elevated air exchange rate.

7.1. ^{222}Rn diffusion barriers

Radon emanating from building materials and soil can be controlled by surface coating and sealing of cracks in the basement floor and walls. Several recent quantitative assessments (37,38,39) of the reductions achievable with different sealants show that a sheath(thickness .2 mm) of polyamide of polyethylene reduces emanation by at least 97 %. The much easier applicable paints only reduce emanation by 32 - 67 % (latex paint) and 47 - 87 %

(epoxy paint)(38). A four layer coating of epoxy resins with various additives was foud to impede radon satisfactorly (37). Although the trapping of radon daughters in the wall increases γ ray exposure by about 25 %, the overall effect on indoor exposure is clearly beneficial. Moeller (39) calculated that the use of surface sealants (epoxy paint) in basement areas would result in a reduction of lung dose equivalents at a cost as low as US$ 3000 per person-Sv (US$ 30/person-rem). This value compares favorably to the value of US$ 100'000 per person-Sv (US$ 1000/person-rem) adopted 1975 by the US Nuclear Regulatory Commission to determine the cost-effectiveness of techniques for reducing routine radionuclide release from power plants (39,40).

7.2. Heat exchangers

At a cost of about US$ 500 - 1000 per single family home, heat exchanger trasferring most of the thermal energy above ambient level of the outgoing air to the fresh incoming air can be installed. The small amount of energy needed to run the electric fan of such a system ends up heating the indoor air, thus preventing energy loss.

The higher air exchange rates achievable at low heating costs with a heat exchanger would also have beneficial impact on several other indoor pollutants reaching critical levels in poorly ventilated houses. The most commonly found toxicants considered sufficiently serious to warrant action to limit their concentration are asbestos fibers, CO and NO_2 from unvented combustion in gas stoves and heaters, aldehydes and tobacco smoke.

7.3. New buildings

The radium content of materials widely used in the construction of homes and public buildings should be assessed to prevent a repetition of the Swedish experience with aerated concrete from alum shale. Constrution on potentially dangerous grounds, i.e. on tailings from phosphate or uranium mining, has to be regulated.

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