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Indoor Exposure to Radon and its Health Effects

(invited lecture on Int. Symposium on Indoor Environment, 25-27 Sept. 1997, Ustroń, Poland)

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Abstract

Radon (Rn-222) is a noble radioactive gas which originates during U-238 series decay. As a noble gas it is not reacting with soils and building materials and therefore is showing large mobility due to its half-life of 3.82 days. It decays through alpha emission and is producing other radioactive isotopes (Po-218, Bi-214 etc.) which are solid.

The migration of radon and its decay products can be in unattached form or attached to aerosols. The size of aerosol particles is important for adhesion coefficient value and for inhalation probability by human respiratory system. The unattached radon is penetrating more easily into lung space and there it may decay into radioactive and alpha emitting solid isotopes. The emitted alpha particle can damage sensitive cells. An alpha particle that penetrates the epithelial cells can deposit enough energy in a cell to kill or transform it. The transformed cell, alone or through interaction with some other agent, has the potential to develop eventually into a lung cancer.

The data on risk of a lung cancer occurence for high and medium concentrations of radon in the air will also be presented.

Radiation from radon forms in a country of moderate climate about 60% of total absorbed radiation dose. Therefore it is important to study its health effect.

Radon (Rn-222) originates in U-238 series decays. As a noble radioactive gas it is not reacting with elements of rocks, soils or building walls therefore preserving its mobility. Also in thorium series (Th-232) there exists a gaseous Rn-220 isotope, called also thoron, which is less important due to its shorter half-life time (55 s). This shorter half-life time makes migration on larger distances less probable.

Both Rn-222 and Rn-220 are radioactive and when decaying are emitting alpha particles. In Fig.1 is shown the decay scheme of Rn-222 and its shortliving decay products. The decay products which are solids can be attached to aerosols, which when they are breathed are deposited on the interior surfaces of the lung. Two of Rn-222 daughters, polonium-218 (RaA) and polonium-214 (RaC') emit alpha particles and are the source of radiation damage when they decay in the lung.

For a radioactive chain the amount of each chain member will be adjusted until reaching a balance called „secular equilibrium". In secular equilibrium the decay rate $N_i\lambda_i$ should be equal in consecutive members of the radioactive series. Here the equilibrium factor of a value other than 1 will describe the deviation from the state of secular equilibrium. In Fig.2 is shown the time schedule for reaching secular equilibrium between radon and its shortlived daughters.

In the radon dosimetry, however, a different equilibrium factor is used which is denoted F. It is connected not only with decay constants but also with the „potential" alpha energy of decay products. Thus we are obliged to define the equivalent concentration C_{eq} of decay products (having equal alpha particle energy) compared to parent radon. It is calculated as (Porstendoerfer, 1993):

$$C_{eq} = 0.105C_{RaA} + 0.516C_{RaB} + 0.379C_{RaC} + 6x10^{-8}\, C_{RaC'}$$

where the coefficients for particular term are depending also on its alpha energy and alpha decay probability. And now:

$$F = C_{eq}/C_{Rn}$$

Departures from the equilibrium occur when removal processes alter the relative abundances of radon and its daughters in the air. For indoor radon the most important of those processes are ventilation and the plateout of radon daughters on surfaces within the house.

The experimental distribution of F values for ventilation air in a mine is shown in Fig.3 (Skowronek, 1992). We see here that low F values are the dominant ones.

The radon and thoron (Rn-220) decay products undergo in the atmosphere a two-step process, which is schematically presented in Fig.4. When Rn-222 decays there emerge the nuclei of decay products; they are surrounding themselves very fast (<1s) with gas and water vapour molecules present in the air; forming molecule clusters of 0.5 to 5 nm diameter. During next 1-100 seconds these clusters are attaching to the aerosols present in the air. In this way originate radioactive aerosols containing radon daughters. The probability $\beta(d)$ of attachment of radon decay product to aerosol depends on aerosol size d. This effect is a diffusion effect; its theory was given by Arendt, Kalman, 1926 and Baust, 1967. This theory quite well describes the experimental data and is shown in Fig.5.

The desorption of the cluster from aerosol surface is possible in consequence of radioactive decays with alpha particle emission.

As the result of the above enumerated processes we have in the air both unattached decay products and attached to aerosols.

Equilibrium coefficient F as well as the fraction of free decay products f_p are depending on aerosol attachment rate X or aerosol concentration Z (both these quantities are interdependent - low aerosol concentration means high attachment rate). The fraction of free decay products is defined as the ratio (f - means free):

$$f_p = C^f_{eq}/C_{eq}$$

Experimentally this looks as shown in Fig.6.

To obtain a realistic estimate of inhaled dose it is necessary to know also the granulometric distribution of aerosol particles Z(d) in the air at place of measurement. These size distributions of aerosols are strongly dependent on atmospheric conditions; observed were also bimodal distributions (two peaked). At a given place the size distribution of aerosols can undergo fast changes as may be seen in Fig.7 (Chuan, 1976). Here is presented the aerosol mass distribution C ($\mu g/m^3$) measured outside buildings at Costa Mesa (CA) about 11 km from ocean shore. We see that this distribution is changing largely with changing temperature and wind.

The aerosols are inhaled into the lung and the smallest of them are penetrating the alveoles. The percentage of aerosols penetrating into alveoler region depends on aerosol diameter. The data from AEC - Los Alamos and BMRC - British Medical Research Council (Fig.8) are a little different, but anyway it is easily seen that aerosols of the size greater than 10 μm are not penetrating into the alveols.

It is obvious that the radon decay products are not attached to every aerosol particle. The size distribution of active (with attached decay product) aerosol will be the convolution of attachment rate $\beta(d)$ and size distribution of the aerosols.

Deposition in the lung of the fraction of the radon daughters attached to the aerosol is the result of diffusion, impaction and sedimentation. Diffusive deposition is the result of random Brownian motion of small particles which bring them into contact with the surface of the lung. Deposition by impact occurs when a particle can not negotiate an abrupt direction change of an air streamline so that it strikes the duct wall. Sedimentative deposition is the result of gravity causing it to settle out on airway wall.

The maximum deposition probability lays in the region of aerosol sizes $0.1 \div 0.25$ μm.

The ions which comprise the unattached fraction of radon daughters deposit by simple diffusion to the walls of respiratory tract. For a typical value of breathing rate (750 dm^3/h) about 65% of the inhaled unattached fraction is removed (George, 1969). Of the remainder, the deposition probability per area unit is much greater in the larger upper airways.

The studies of miners have shown that the lung cancer incidence from radon is delayed some years after the exposure. The latent period seems to vary inversely with age at first exposure, with amount of cigarette smoking and with total exposure and/or exposure rate. That is, the shortest latent periods are found among those men who are elderly at start of mining, who smoke heavily and who have the intense exposures. The latent period has a large range of about 7 to 50 years. Mean values are usually considered to be between 20 and 30 years. In none of the studies so far has there been any significant appearance of lung cancers before age of 40 (NCRP Report 78-1984b).

The question arises how dangerous is radon as carcinogenic agent. It is well known that high concentrations of radon encountered in mines cause important increases in cancer deaths. Table 1 shows the data for US uranium miners. It is well seen that for low equivalent radon concentrations ~ 600 Bq/m^3 no excess of lung cancers is observed. For higher exposures it is increasingly rising the lung cancer risk. E.g. for equiv. radon concentrations of 27 kBq/m^3 the incidence of lung cancers has increased 11 times.

For lower radon concentrations we have less clear data. A 1991 report of Karolinska Hospital in Stockholm has a survey population 1360 persons. In period 1980-84 these

persons have a diagnosed lung cancer. During the survey 90% of them died. They were of 37-74 years of age and 86% were cigarette smokers. The houses in which they have lived longer than 2 years got radon concentration measured. The values rise up to 6780 Bq/m^3 with an average of 106.5 Bq/m^3. In Table 2 are shown the values of relative risk of cancer incidence compared to incidence rate of lung cancer for nonsmokers and living in houses with radon concentrations < 50 Bq/m^3. We can see from this table that for all persons there exists a rise in lung cancer incidence with rising radon concentration; for nonsmokers, however, it is probably not significant. All cancerogenous effects of radon are smaller than the effect caused by smoking. For smokers there is an increase of cancer incidence rate of more than 10 times. Also there exist in the smokers group an evident rise on lung cancer incidence with rising radon concentration at home. This evidence supports the concept that the risks from exposure to radon and cigarette smoke produce multiplicative risks. Smoking and high radon concentration are thus synergic.

Fig.9 shows the possible overlap of radon and smoking related lung cancer deaths (Data of US EPA - 1987).

The problem whether the low radon concentrations are harmful is unsettled. Some researchers (Cohen) argue that there is even a counterevidence. The data of Cohen shows Fig.10. Here we have for 1610 US counties the lung cancer mortality plotted against average indoor concentration of radon. The regression line is falling in the range of radon concentrations $0 \div 6$ pCi/l (222 Bq/m^3). On the other hand ICRP is not ready to abandon the linear risk-exposure relation (shown in the figure as Theory), by which even small doses are harmful. Urgently new large studies are needed here.

And to end my took a pinch of Attic salt: we are talking on average radon concentration. It is therefore good to look at continuous measurement results in Kraków and the daily variation of radon concentration. They are shown in Fig.11.

I would like to thank Ms Jadwiga Mazur and Mr Grzegorz Tracz for helping in preparation of the manuscript.

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Table 1
Lung cancer risk for US uranium miners due to radon exposure.

(1) WLM Interval	(2) WLM Midpoint	Radon at home* equiv. F=0.5 [kBq/m^3]	(3) Person Years	(4) Lung cancers Observed	(5) Lung cancers Expected
0-119	60	0.58	5183	3	3.96
120-239	180	1.75	3308	7	2.24
240-359	300	2.91	2891	9	2.24
360-599	480	4.66	4171	19	3.33
600-839	720	6.99	3294	9	2.62
840-1799	1320	12.81	6591	40	5.38
1800-3719	2760	26.79	5690	49	4.56
> 3719	7000 (est.)	67.96	1068	23	0.91
ALL	1180	11.46	32196	159	25.24

Notes:
(1) Specifies range of cumulative exposures over working lifetime.
(2) Mid-point of interval from (1).
(3) The number of person-years is based on the number of years at risk. For each person, the years-at-risk is taken to equal the time interval starting 10 years after the beginning of mining and continuing to the end of the study period (Sept. 30, 1974).
(4) Observed lung cancers include those during the time interval starting 10 years after the beginning of mining and continuing to the end of the study period (as above).
(5) Expected lung cancers are based on „age- and year-specific rates for white males in Colorado, Utah, New Mexico and Arizona".
* Nat. Acad. Sci. BEIR III Report 1980, calculated for 20 y av. working time.

Table 2
The risk of lung cancer incidence as function of radon concentration (Karolinska Hospital, Stockholm).

Radon concentration [Bq/m^3] relative risk coefficient	< 50	50-80	80-140	140-400	> 400
all	1	1.1	1.0	1.3	1.8
nonsmokers	1	1.1	1.0	1.5	1.2
former smokers	2.6	2.4	3.2	4.5	1.1
<10 cigarettes/day	6.2	6.0	6.1	7.3	25.1
>10 cigarettes/day	12.6	11.6	11.8	15.0	32.5

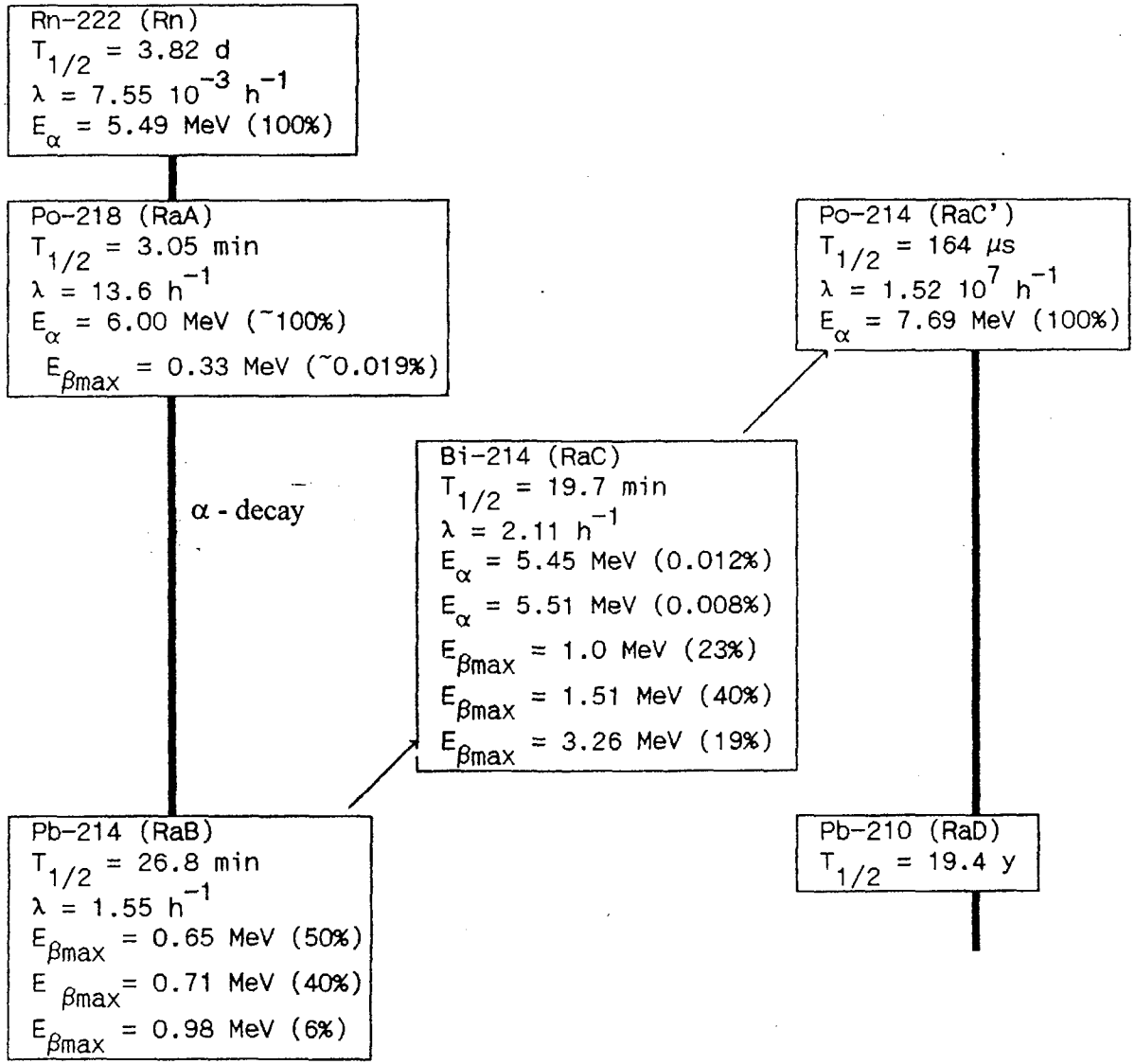

Fig.1 Decay scheme of Rn-222 and its daughters.

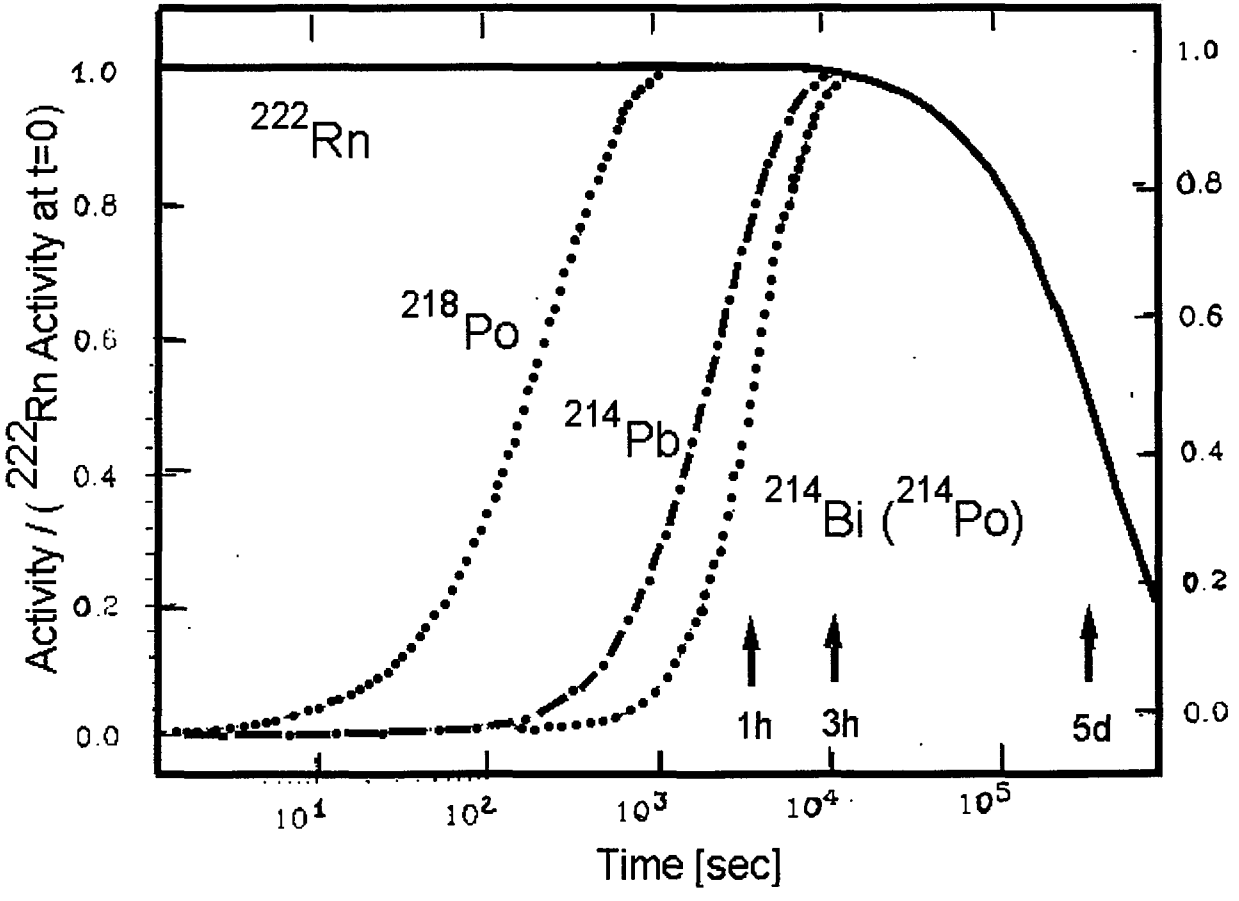

Fig.2 ^{222}Rn daughter build-up.

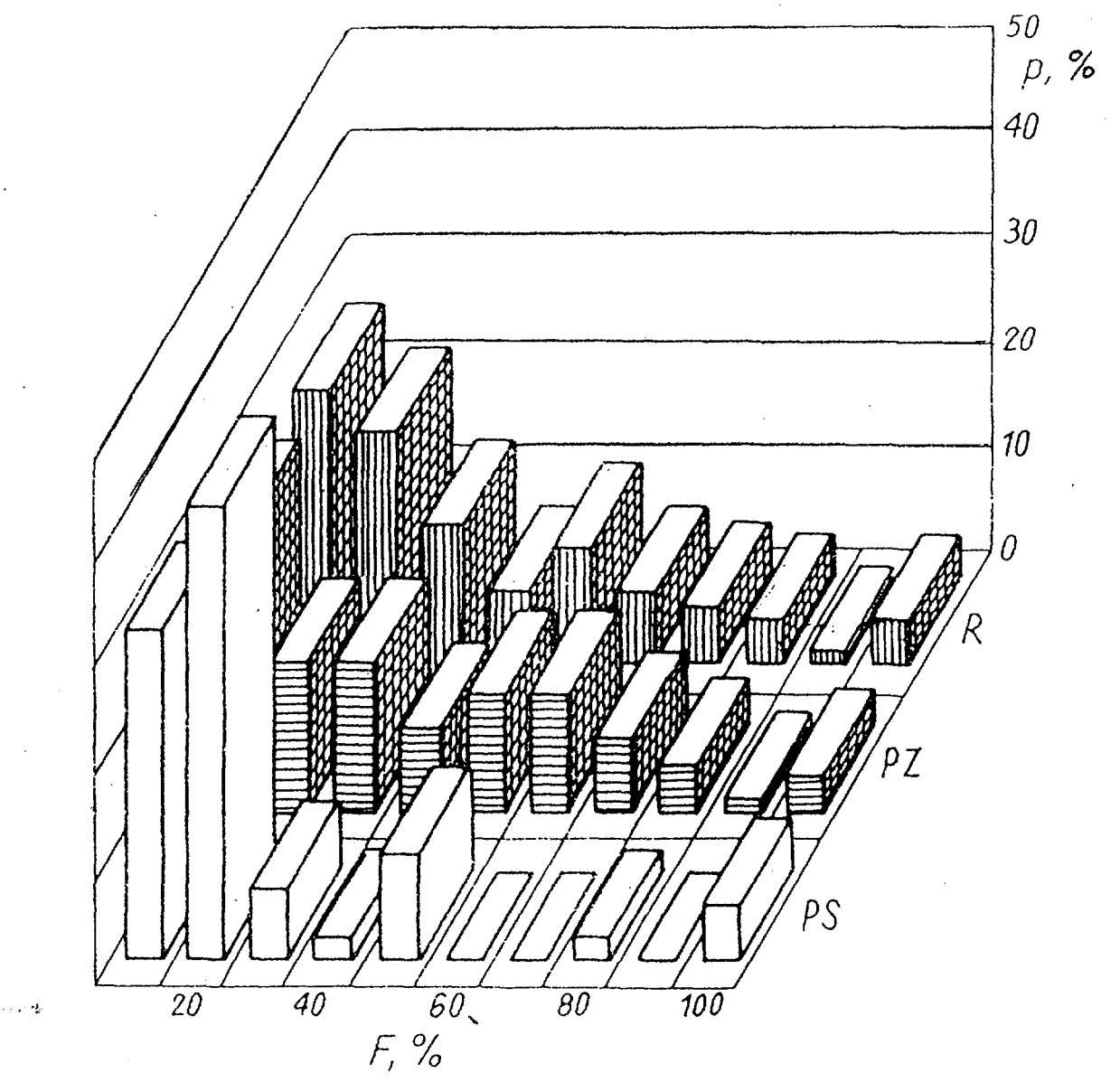

Fig.3 Equilibrium factor F measured in different air ventilation streams in a mine (Skowronek 1992)

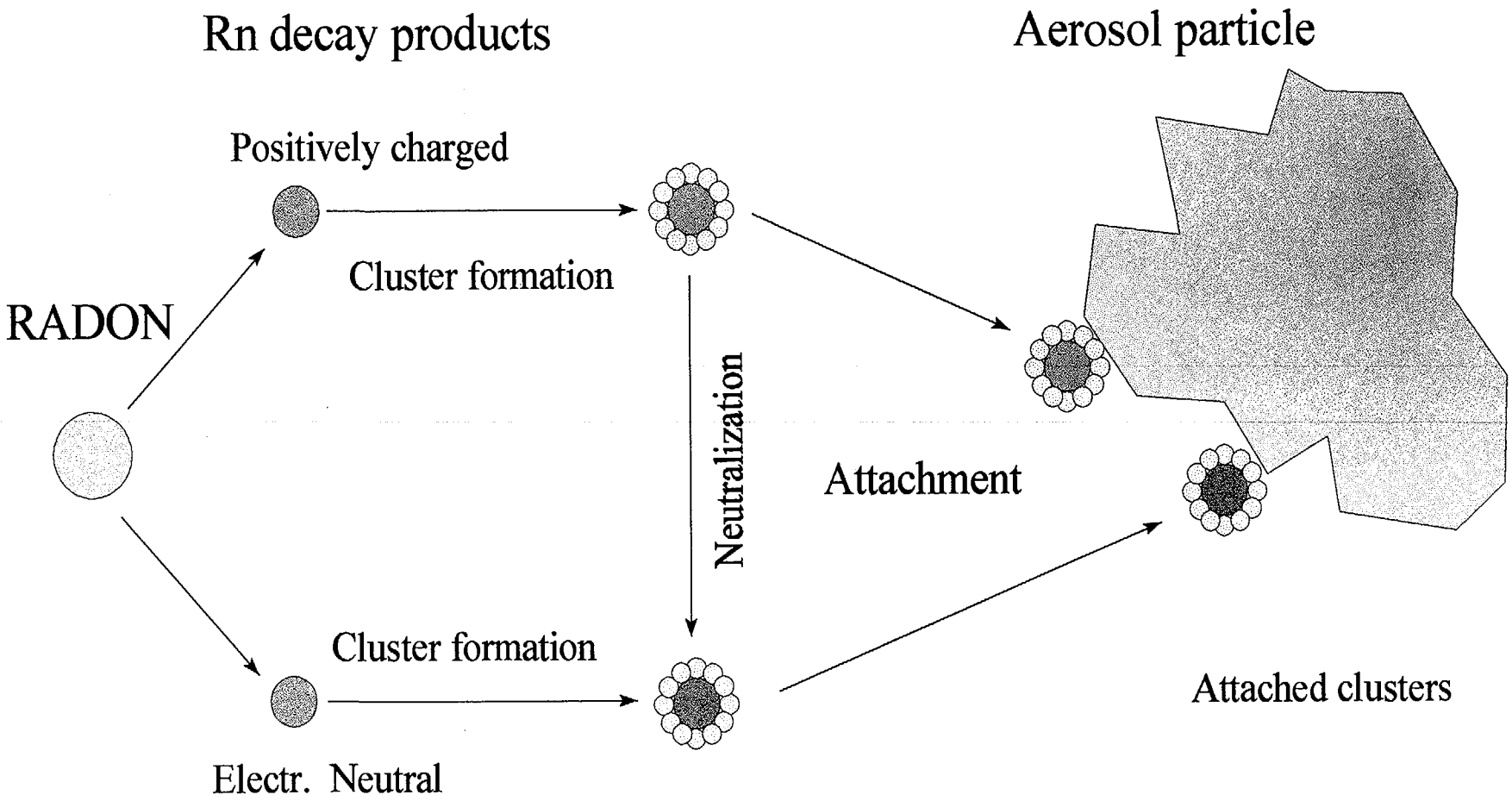

Fig.4. Basic processes of decay products of radon and thoron occurring in the atmosphere

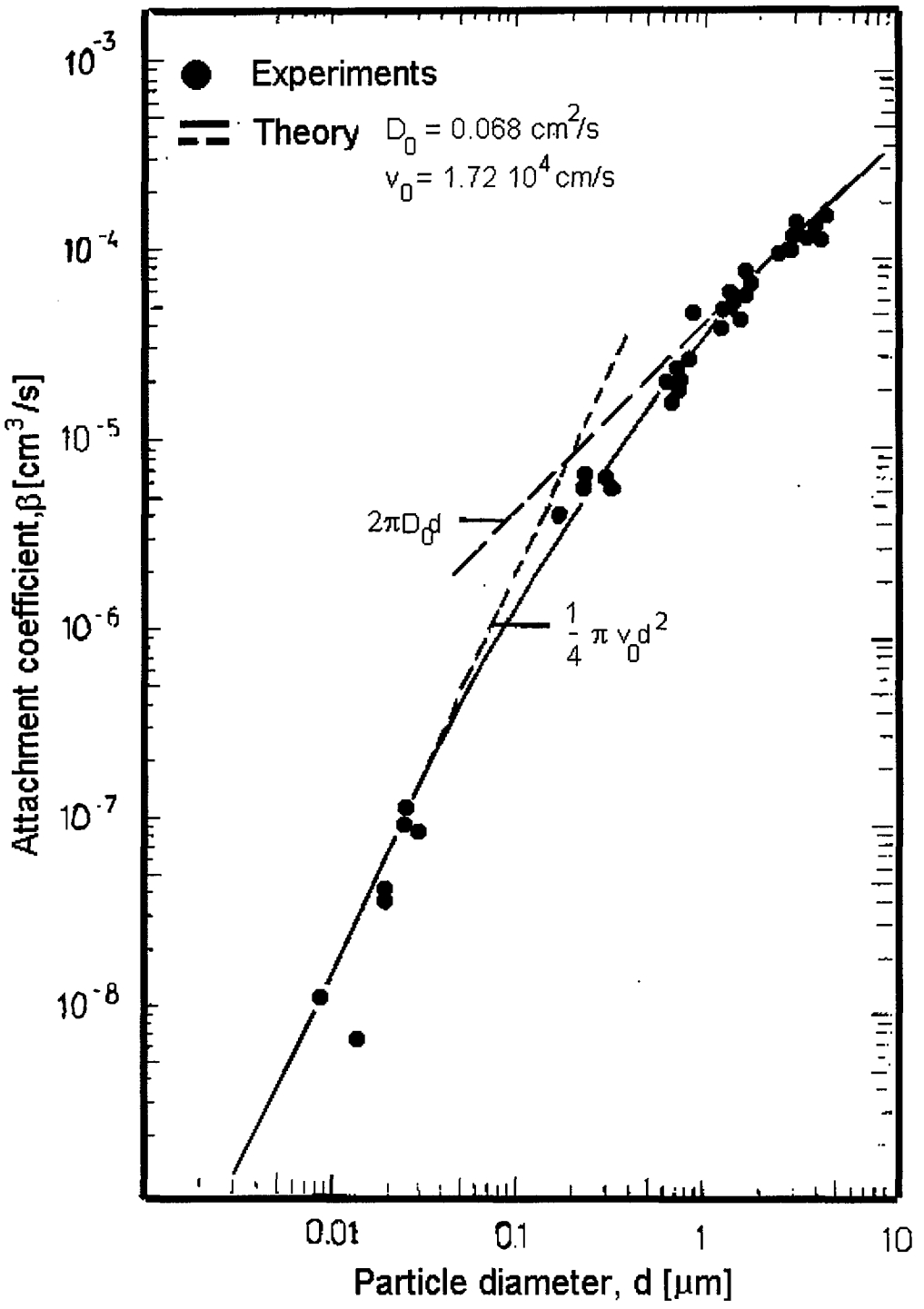

Fig.5 The attachment coefficient of the radon/thoron daughters as a function of aerosol size.

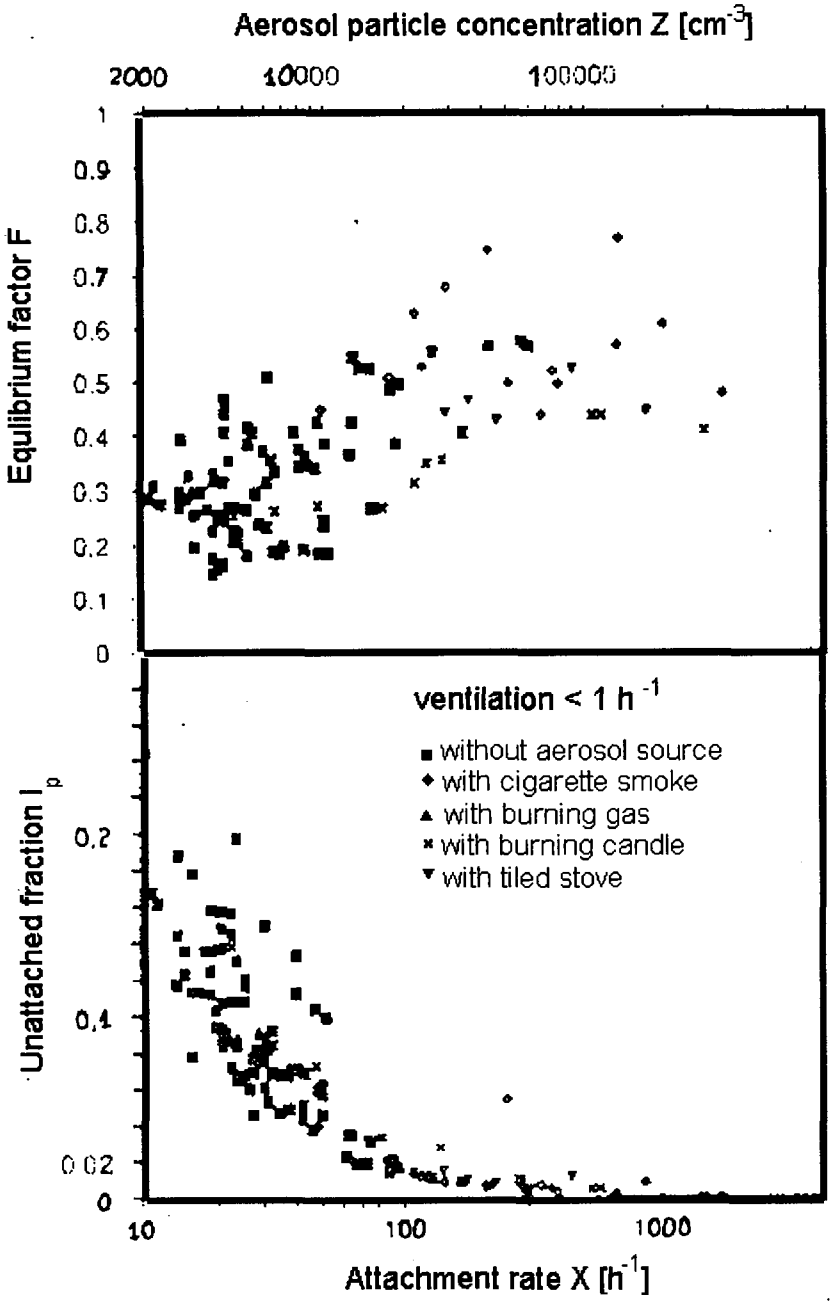

Fig.6 The unattached fraction (f_p) and the equilibrium factor (F) in rooms with different aerosol sources in function of the attachment rate (x) and aerosol particle concentration (z)

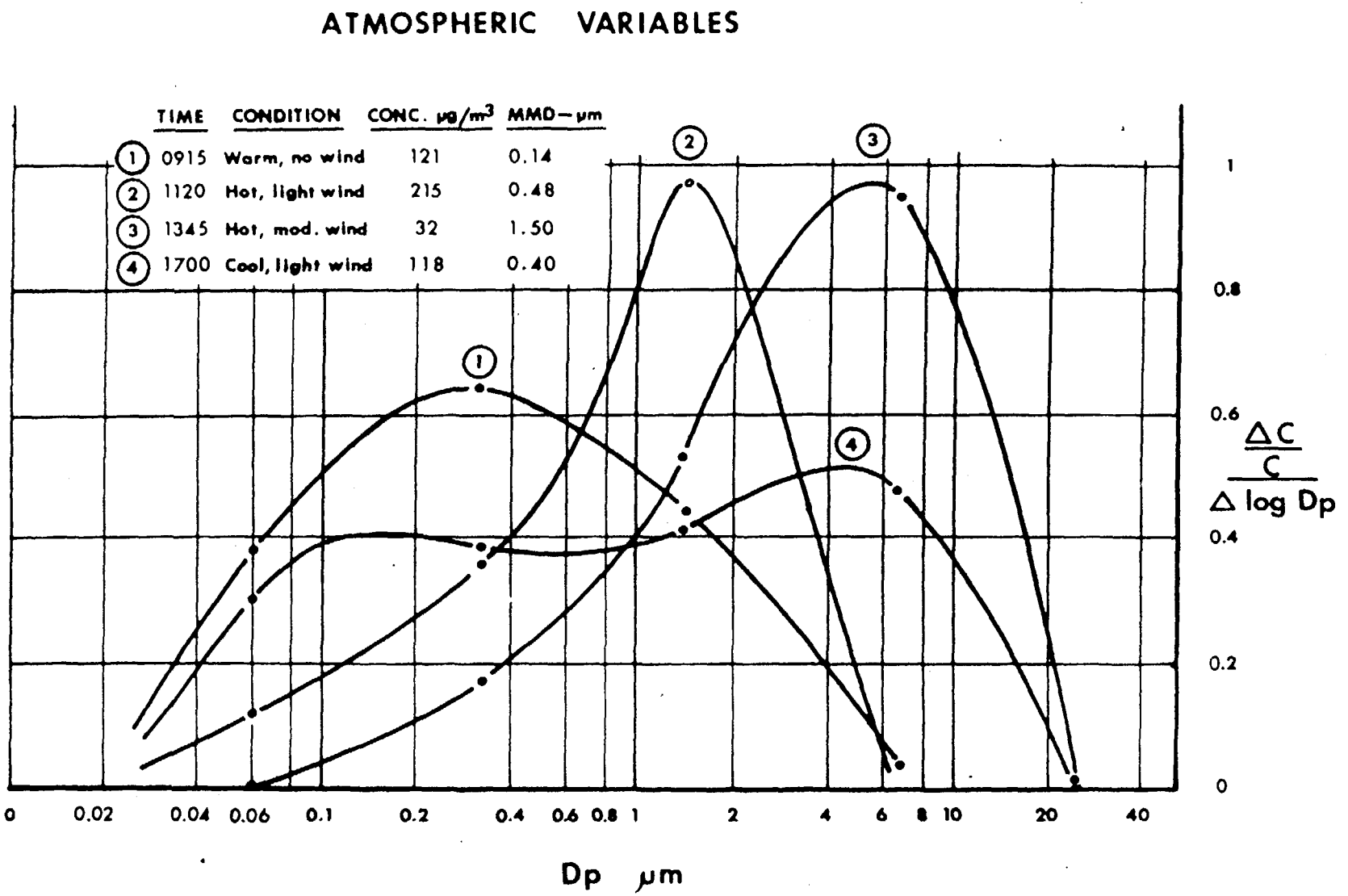

Fig.7 Aerosol mass distribution at different atmospheric conditions.

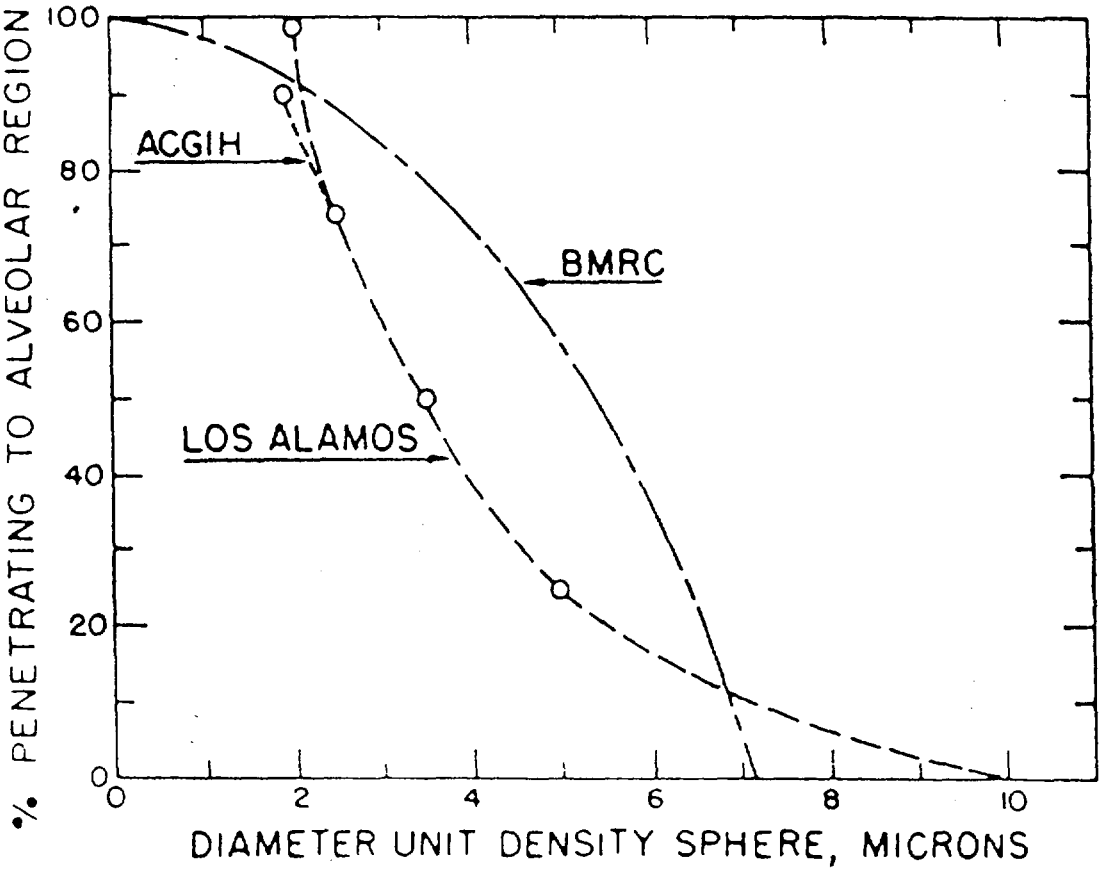

Fig.8 Percentage of aerosols penetrating into alveolar region as a function of their diameter.

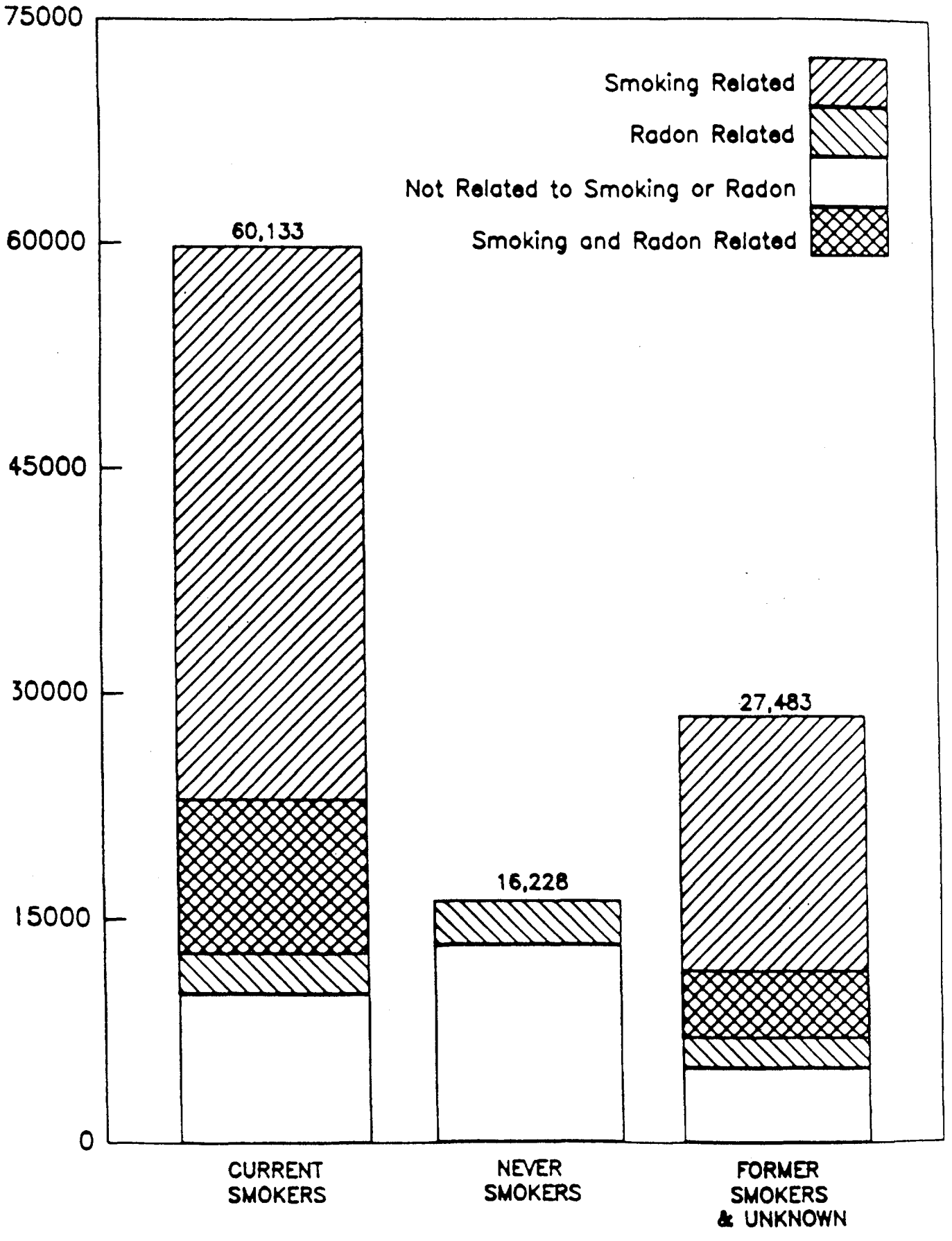

Fig.9 Possible overlap of radon and smoking related lung cancer deaths.

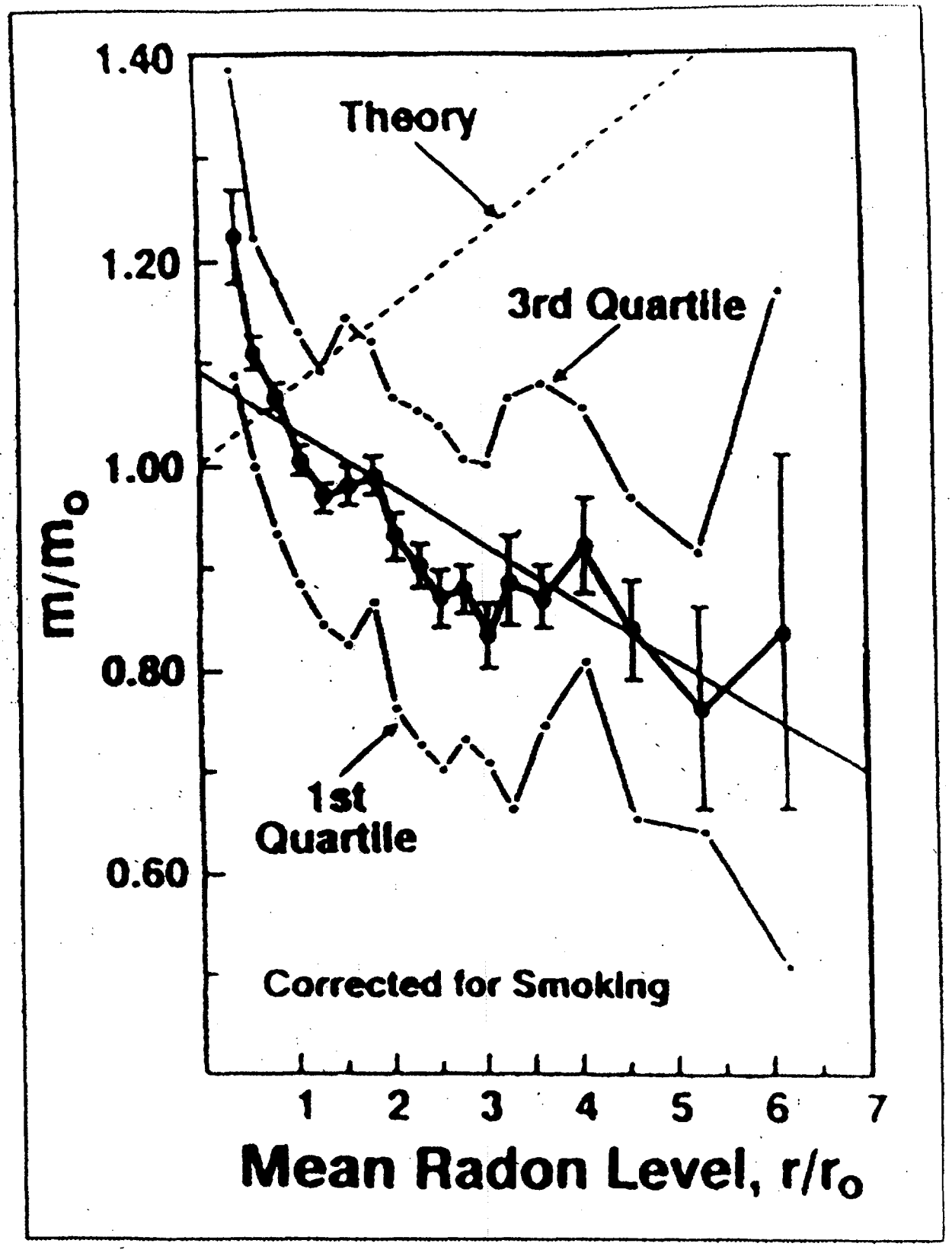

Fig.10 Lung cancer mortality rates vs. mean radon level r_0 = 1 pCi/L = 37 Bq/m^3, in homes for 1610 U.S. counties.

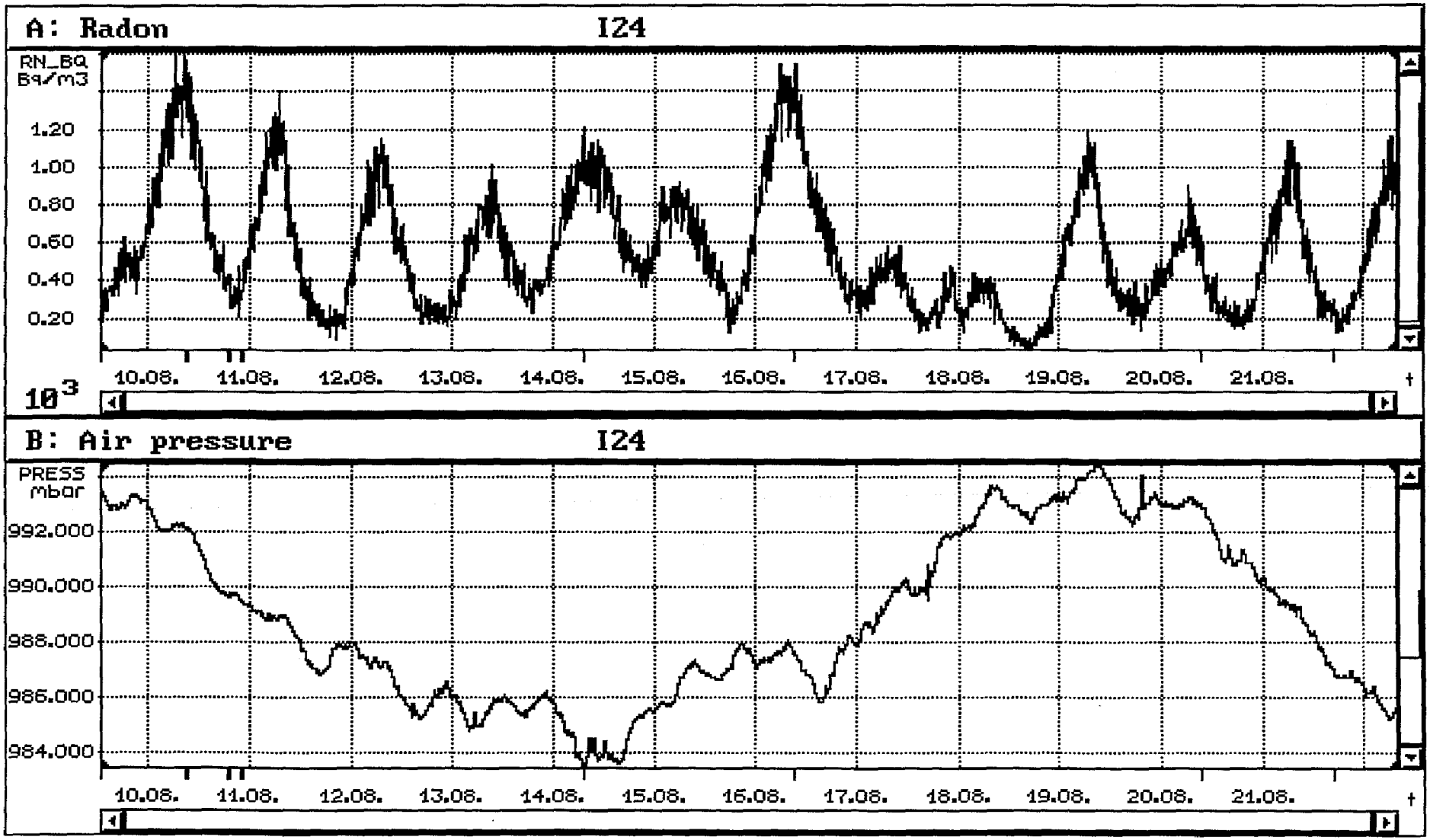

Fig.11. Continous measurment of Radon concentration in a house in Kraków 8-22.08.96

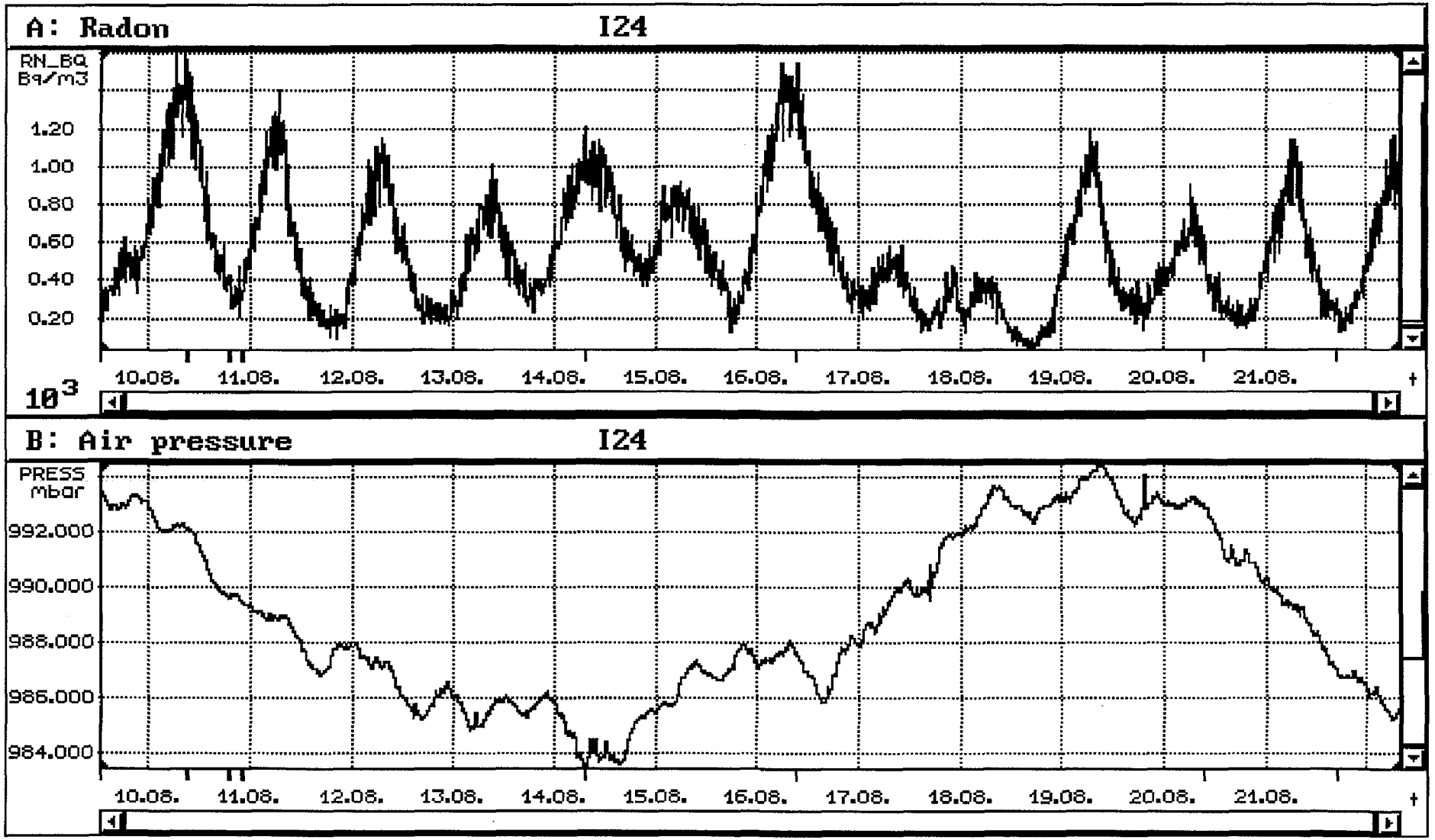

Fig.11. Continous measurment of Radon concentration in a house in Kraków 8-22.08.96