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# Radon induced lung cancer in residents of Rivne (Ukraine) calculated by various risk models

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The study estimates the risks of morbidity and mortality from lung cancer associated with residential Radon-222 exposure in Rivne (Ukraine). We used the Joint European miner cohort model, Joint European miner nested case-control model, and Joint European residential Radon model with some modifications, applied to the inhabitants of the urbanized territories of Europe. The estimated number of deaths in Rivne ranged from 6 to 39 males from 159 lung cancer deaths in 2014 and from 1 to 9 females out of 36 lung cancer deaths; this range was caused by Radon exposure in the city premises and uncertainty in the dose-effect dependence of these models. Our research should be extended to the other regions of Ukraine as the Radon problem is acute for the country and this is a matter of concern to the European Community and the World Health Organization.

Keywords: Radon; Cohort studies; Additional relative risk; Exposition; Lung cancer

# Introduction

Lung cancer causes more deaths than the other most common cancers. It is estimated that 19.7 % of all EU cancer deaths in 2006 were associated with lung cancer (Ferlay et al., 2007). The vast majority of these deaths are attributed to tobacco smoking, but studies of the concentration of Radon in residential buildings indicate that its effects may be responsible for a significant percentage of these deaths. Radon-222, commonly called Radon is a chemically inert radioactive gas that is part of the Uranium-238 radioactive family. Uranium ranks 38th in other natural elements by the distribution. The content of Uranium in the Earth's crust is  $4 \times 10^{-4}$  % (by weight). It can be found in rocks, soil  $(1.2 \times 10^{-5} - 9.3 \times 10^{-5} )$ , in rivers and seas  $(2 \times 10^{-8} - 5 \times 10^{-6} )$ , and in oceans  $(1 \times 10^{-7} )$  (Nesmeyanov, 1985). A daughter isotope of Uranium-238 in the breakup series is Radium-226. Radioactive gas, Radon-222 is formed naturally during a chain of radioactive decay from Uranium-238 and produced by most rocks and soils. There are several possible sources of Radon in the home and the most important is the infiltration of soil gas.

A series of short-lived daughter product of Radon decay (*DPD*) are formed in the household air, which can be adsorbed by aerosol particles in the air or deposit on the surfaces of buildings. Inhalation and deposition of short-lived airborne Radon derivative products of decay in the lungs lead to irradiation of alpha particles of sensitive cells in the lung tissue, such as basal cells of the bronchial epithelium (National Research Council, 1988). Considering the corresponding half-lives of Radon *DPD*, their physical and chemical properties, and the various suggested dosimetry models of the lungs, it can be argued that 5 a- and 4  $\beta$ -particles produced by the short-lived products of its decay (up to Pb-210) are formed in the decay of one Radon nucleus. The Polonium-218 (Ea = 6.00*MeV*) and Po-214 (Ea = 7.689*MeV*) are particularly harmful. Since these alpha particles have corresponding runs of only 48 and 71 µm in the lung tissue, they provide a high density of ionizing cell damage at such short distances. This pulmonary dose considered the cause of Radon-induced lung cancer, either alone or in conjunction with tobacco smoke carcinogens.

The US surgeon general named Radon the second cause of lung cancer after active smoking, and the IARC classified Radon as a group 1 carcinogen (United States, 2005; IARC, 1988). Previously, some researchers suggested that Radon exposure can cause other diseases such as leukaemia in children or adults (Laurier et al., 2001). Now it is clear that one of the effects of Radon on humans is the lung cancer.

An excess relative risk (R) of Radon-induced lung cancer is calculated by the formula (Zhukovsky, 2007):

$$R = \sum_{t=0}^{\infty} \lambda_0(t) p_0(t) K_{ERR}(t) \cdot \exp\left[-\sum_{t'=0}^{t} k_{let} \lambda_0(t') K_{ERR}(t')\right], \tag{1}$$

Where  $p_0(t)$  is the likelihood of person's survival to age t,  $\lambda_0(t)$  is the frequency of spontaneous lung cancer occurrence,  $K_{ERR}(t)$  is the excess relative risk factor calculated according to the selected risk model for Radon-induced lung cancer,  $k_{iet}$  is the mortality rate ( $k_{iet} = 0.95$ )).

# Methods

We monitored the volumetric activity (*VA*) of Radon in the household air of city buildings of Rivne (Klymenko & Lebed, 2017; Lebed et al., 2018a; 2018b), in the water (Lebed et al., 2018), in the soil gas (Lebed et al., 2018). We also checked the effect of Radon on lung cancer risk in Rivne residents (Lebed et al., 2018; 2019) in 2012-2019 by means of the radiometric equipment "AlfaRad Plus" (Instrument-making company "NTM-ZASHCHITA", Russia), intended for rapid measurements and continuous monitoring of *VA* of Rn-222 and Rn-220.

Total measurements were made in 185 basements, 215 semi-basements and 200 first-floor premises (600 rooms in total). The arithmetic mean of *VA* in the measured premises was 262.5  $Bq/m^3$  with a standard deviation of 194.4  $Bq/m^3$ , which indicates a significant variability of Radon levels in the air of residential buildings in the city of Rivne. The average geometric *VA* value for basements was 365  $Bq/m^3$ , which was much higher than the building norms of Ukraine (100  $Bq/m^3$  – for new buildings and 200  $Bq/m^3$  – for long-term occupied buildings). Separate basements of dwelling stock were recorded in which *VA* values reached 966±193  $Bq/m^3$ . The average geometric *VA* value for semi-basements was 161  $Bq/m^3$ . The average geometric *VA* on first floors or residential premises in Rivne was 127  $Bq/m^3$  (95% CI: 118-137); arithmetic mean – 145.6  $Bq/m^3$ , maximum value - 714  $Bq/m^3$ . For all the investigated premises, the average geometric value of *VA* was 200  $Bq/m^3$  and maximum value was 1,420  $Bq/m^3$  (Figure 1).



**Figure 1**. Density of probability distribution P(VA) of Radon VA in the air of investigated premises, Rivne.  $x = ln(VA/VA_{geom}), VA_{im} - most$  probable value of VA,  $VA_{geom} - geometric mean of <math>VA$ ,  $VA_{max} - predicted maximum value of <math>VA$ .

To determine the Radon induced risks of lung cancer in Rivne residents, the Radon exposure in city dwellings was calculated as the geometric mean of *VA*, according to the recommendations by National Research Council (1999). We assumed that the city residents spend on average 7,000 hours per year at homes, and the equilibrium coefficient between Radon and its *DPD* in the buildings is 0.4. The calculation of these data for Rivne gives the risk probability P = 0.784 WLM/year. We used this exposure value in the calculations K<sub>ERR</sub>(t) for selected risk models.

#### Models of risk calculation

**1. BEIR-VI model (I)**: The BEIR-VI Committee performed a pooled analysis of data from 11 cohorts of miners exposed to Radon in the mines of China, Canada, Europe, the United States and Australia (National Research Council, 1999). The analysis included more than 2,620 lung cancer deaths among 60,570 miners, which were traced for nearly 1.2 million people. The Committee has developed two models to evaluate the risk of Radon-induced lung cancer based on either the duration of exposure (exposure-age-duration, EAD model) or the average Radon concentration (exposure-age-concentration, EAC model). In both models, cohort, age group, other occupational characteristics, and ethnicity stratified the baseline risk. In addition, in both models, Radon radiation is considered to have a multiplicative effect on baseline lung cancer cases, and the additional relative risk (R, i.e. relative risk minus 1) decreases with increasing time from the moment of exposure and with age. In addition, both models consider different time intervals from the onset of exposure to death from lung cancer; cumulative exposure to Radon *P* was defined as the sum of three time windows of exposure:  $P = P_{5-14} + P_{15-24} + P_{25+}$ , where a person's exposure was obtained within 5–14, 15–24 and 25 years or more before attaining the age (i.e.  $P_{5-14}$ , means the average exposure accumulated over a period of 5 years and 14 years before attaining the age). These models do not take into account the smoking factor for the investigated persons (National Research Council, 1999). The EAC model is simpler than the EAD model because it compares the risks for miners with those for urban

dwellers who breathe Radon in their homes. The excess relative risk factor  $K_{ERR}(t)$  in BEIR-VI models is taken from (Health effects, 1999) and is expressed as:

$$K_{ERR}(t) = \beta \cdot (P_{5-14} + 0.78 \cdot P_{15-24} + 0.51 \cdot P_{25+}) \cdot \theta(t) \cdot \lambda_Z, \qquad (2)$$

where  $\beta$  is the risk-exposure ratio. It is different for smokers, non-smokers, and the general population.

We used values  $\beta$  for the general population ( $\beta$  = 0.055 for EAD- model and  $\beta$  = 0.0768 for EAC-model);  $P_{5-14}$ ,  $P_{15-24}$  and  $P_{25+}$  are aggregate Radon exposures (expressed in months of working level (WLM) obtained during 5–14 years, 15–24 years, and 25+ years or over up to the attained age,  $\theta$ (t) is a coefficient that determines the dependence of carcinogenic susceptibility of lung tissue on the attained age *t* (modification coefficient).

It is tabulated and defined as  $\theta(\le 55 \text{ years}) = 1$ ,  $\theta(55-654 \text{ years}) = 0.57$ ,  $\theta(65-74 \text{ years}) = 0.29$ ,  $\theta(\ge 75 \text{ years}) = 0.09$  and  $\lambda(z)$  is a function that depends on the duration of irradiation *z* (in years) or the volumetric activity level of Radon *DPD* expressed in units of the working level WL (1 WL corresponds to the equivalent equilibrium volumetric activity (*EEVA*) of Radon 3,700 *Bq/m*<sup>3</sup>). In this model, the exposure obtained over the previous 5 years suggests that it does not increase the risk of lung cancer. In addition, the temporal structure of the exposure was taken into account in the simulation, but not simply the time when it began and when it ended. In this case, as well as in the other models described below, *R* was considered the same for males and females.

Since the level of exposure in homes (less than 0.5 WL) is much lower than in mines, the coefficient  $\lambda(z)$  in the EAC model is equal to one.

**2. Cohort model (II):** This was established on the basis of a cohort study of the morbidity of uranium miners in the Czech Republic, France and Germany for lung cancer due to Radon in the framework of the Alpha Risk project, which is supported by the European Commission (Tirmarche et al., 2009). Within the framework of this project, 1,543 lung cancer deaths were investigated in three European countries among 5,149 uranium miners exposed to relatively low Radon exposure between 1946 and 1999, which were traced among nearly 1.3 million people. The peculiarity of this model is that it provides 5, not 4 as in BEIR-VI, values of the modifying factor  $\theta(t)$  depending on age and other windows of exposure *P* for 5-19, 20-34 and more than 35 years before attaining the age (Tirmarche et al., 2009):

$$K_{ERR}(t) = \beta \cdot (P_{5-19} + 0.42 \cdot P_{20-34} + 0.14 \cdot P_{35+}) \cdot \theta(t), \tag{3}$$

where  $\beta = 0.052$ ; *P* – time windows since exposure up to the attained age with a modifying factor  $\theta(t)$ :  $\theta(\leq 45 \text{ years}) = 1$ ,  $\theta(45-54 \text{ years}) = 0.66$ ,  $\theta(55-64 \text{ years}) = 0.39$ ,  $\theta(65-74 \text{ years}) = 0.33$ ,  $\theta(\geq 75 \text{ years}) = 0.49$ .

**3. Nested case-control model (III):** To investigate the multiplicative interaction of the effects of Radon respiration and cigarette smoking on the occurrence of lung cancer, a case-control study of three European cohorts of uranium miners was conducted. 1,476 cases of lung cancer were investigated among European miners and 3,389 control group miners, taking into account the intensity of their smoking (Leuraud et al., 2011; Hunter et al., 2013).

A modified version of the BEIR-VI (EAC) model was applied to this data. The information on smoking was reconstructed using selfadministered questionnaires and medical archives (Leuraud et al., 2011; Hunter et al., 2013). Smoking adjusted linear relative risk models were used to assess the risk of lung cancer associated with Radon exposure. It was concluded that *R* decreased with increasing time elapsed after irradiation and the attained age, but there was no statistically significant effect on exposure level. The model used here is similar to the model given by Hunter et al. (Hunter et al., 2013), but the model parameters are based on two rather than three exposure windows, namely 5-24 and 25+ years, since ERR/WLM estimates for the exposures 5-14 and 15-24years are very similar (Leuraud et al., 2011; Hunter et al., 2013). This model also takes into account the effect of the attained age, since Hunter et al. found that *R/WLM* decreased significantly with increasing age (Hunter et al., 2013). Two options for calculating the additional relative risk ratio are proposed for this model:

#### Model 1:

$$K_{ERR}(t) = \beta \cdot (P_{5-24} + 0.12 \cdot P_{25+}) \cdot \theta(t), \tag{4}$$

Where,  $\beta = 0.052$ ; *P* – time windows since exposure up to attaining the age with modifying factor  $\theta(t)$ :  $\theta(\le 55 \text{ years}) = 1$ ,  $\theta(55-64 \text{ years}) = 0.93$ ,  $\theta(65-74 \text{ years}) = 0.32$ ,  $\theta(\ge 75 \text{ years}) = 0.66$ .

An alternative model based on the obtained data was considered: namely, a constant linear *R*-model with modifying time effects from the first irradiation. For more information on creating this model, see (Hunter et al., 2013). Excess relative risk  $K_{ERR}$  for **Model 2** is calculated in the following way:

$$K_{ERR}(t) = \beta \cdot P \cdot \left[ \exp\left(\alpha \cdot \left(t_{sfx} - 30\right)\right) \right], \tag{5}$$

where *P* is cumulative exposure (P = P<sub>15-24</sub> + 0.12P<sub>25+</sub>), and *a* is a parameter associated with the time effect from the first exposure  $(t_{sfx})$ , in years. The coefficients were  $\beta = 0.013$  and a = 0.78.

This model was also obtained using the data not restricted to the exposure range. Equation (2) is a simplified version of the model obtained in (Hunter et al., 2013). It does not take into account the modifying factor, since the authors rightly believed that the level of Radon exposure in homes would typically be below 0.1 *WL*, which is equivalent to 800  $Bq/m^3$ . The results of an analysis of the multiplicative effect of Radon and smoking showed that *R* was higher among ex-smokers, who did not smoke for a long time, than among active smokers, although these differences were not statistically significant. (Leuraud et al., 2011; Hunter et al., 2013).

**4. Simple Linear Model (IV):** A joint analysis of 13 case-control studies of lung cancer cases in nine European countries included 7,148 people with lung cancer and 14,208 healthy individuals, with detailed information on smoking about all the participants (Darby et al., 2005; 2006). The analysis focused on the average concentration of Radon inside the buildings, in which the observed and the control subjects lived for 5–34 years before being diagnosed lung cancer among the former. The authors of this analysis found no evidence that *R* differed by age, and determined the relationship between Radon irradiation and lung cancer using a simple linear model for  $K_{ERR}(t)$ :

$$K_{ERR}\left(t\right) = \beta \cdot VA(t) \tag{6}$$

where  $\beta$  is the slope ratio of exposure-risk, and VA(t) is the total volumetric activity of household Radon (expressed in  $Bq/m^3$ ) over the previous 30 years (starting from 5 years and ending with 34 years before the time of risk estimation).

After correcting the random uncertainties in the measurement of Radon concentration, the estimated *R* of lung cancer per 100  $Bq/m^3$  was 0.16 (95% CI: 0.05–0.31). There was no evidence that *R* depend on smoking status, although the *R* score was higher in ex-smokers and non-smokers than in active smokers (Darby et al., 2006). Based on a constant *R*-value for different categories of smokers, the absolute risk of lung cancer due to Radon was significantly greater for active and ex-smokers than for non-smokers (Darby et al., 2005, 2006). When considering the conversion from units *Bq hour/m<sup>3</sup>* into *WLM/year*, then the formula (6) is transformed into

#### Model 1 (IV):

$$K_{ERR} = 0.012 \cdot P_{5-34} \tag{7}$$

Where  $P_{5-34}$  is the cumulative exposure of Radon to the premises for the previous 5–34 years, expressed in *WLM* (Hunter et al., 2013).

Hunter et al. (2013, 2015) presented that annual exposure within previous 5–34 years assumed to have the same effect on *R*. However, in order to take into account the possibility that Radon exposures in the last years of exposure to a person may have greater weight than the exposures, which are more distant in time, these authors also proposed an alternative approach, according to which the most important period in irradiation is considered to be the last 5-24 years. In this case, equation (7) is converted into

#### Model 2 (IV):

$$K_{ERR} = 0.018 \cdot P_{5-24}, \tag{8}$$

### Results

The calculations of excess relative risk  $K_{ERR}$  for risk probability P = 0.784 WLM/year in the residential premises of Rivne for the selected model are presented in Table 1 and Figure 2.

**Table 1.** Dependence of the excess relative risk  $K_{ERR}$  on the time of exposure and the selected model.

<i>t</i> , years				Model			
	BEIR-VI (I)		Model II	Model III		Model IV	
	EAD	EAC		1	2	1	2
10	0.060	0.301	0.204	0	0	0.047	0.071
15	0.190	0.602	0.408	0	0	0.094	0.141
20	0.259	0.837	0.612	0.204	0.023	0.141	0.212
25	0.491	1.071	0.697	0.408	0.069	0.188	0.282
30	0.553	1.225	0.783	0.432	0.114	0.235	0.282
35	0.950	1.378	0.868	0.454	0.169	0.282	0.282
40	1.047	1.532	0.897	0.481	0.263	0.282	0.282
45	1.143	1.685	0.611	0.506	0.407	0.282	0.282
50	1.240	1.839	0.630	0.530	0.631	0.282	0.282
55	0.695	1.136	0.383	0.516	0.974	0.282	0.282
60	0.745	1.223	0.394	0.538	1.502	0.282	0.282
65	0.428	0.667	0.343	0.193	2.313	0.282	0.282
70	0.454	0.712	0.352	0.201	3.555	0.282	0.282

It uses the scenario in which a person was born in Rivne and lived up to the age of 70 on the first floor, with the geometric mean of Radon volumetric activity  $VA = 200 Bq/m^3$ . This person spend in the apartment 7,000 hours per year on average, the equilibrium ratio between Radon and its *DPD* in the house is 0.4 (normally, it can be from 0.2 to 0.8).

An analysis of the obtained dependencies  $K_{ERR}$  on the irradiation time shows that up to the age of 60, the greatest value  $K_{ERR}$  is for the BEIR-VI (EAC) model. This was is confirmed by many calculations, for example Hunter et al. (2015). Further, the relative risk ratio for model 2 (II) sharply increases, since there are no reasonable grounds. We believe that in this model the coefficient *a* in equation (5) was not correctly selected.

In calculating the additional relative risk by the formula (1), we used the data for the probability of survival to a certain age for the city residents  $p_0(t)$  for 2014 (Lebed et al., 2018), based on the report of the Main Department of Statistics in Rivne region (Main Department..., 2015).



**Figure 2.** Dependence of the excess relative risk  $K_{ERR}$  on the time of exposure and the model selected. JEMC – Model II, JEMNCC – Model III, JERR – Model IV.

Unfortunately, it is impossible to calculate the age-specific frequency of lung cancer incidents  $\lambda_0(t)$ , which is specific to the investigated area. Moreover, the mortality in Ukraine subdivided into ranges: 0-1 year, 1-2 years, 2-18 years, 18-60, and more than 60 years old. However, in ICRP Publication 50 (1987), it was proposed to set the mortality lag of 5 years (0-5 years, 5-10 years, etc.). The working hypothesis in such cases is the assumption that the age-specific lung cancer rate for any particular region corresponds to the age-specific morbidity for the so-called "sample population" described in Publication 50 ICRP (ICRP Publication 50, 1987), differing from it only by a constant factor *k*:

$$\lambda_0(t) = k \lambda_0^{ref}(t), \tag{9}$$

where the value for males and females can be determined from the following expression (Zhukovsky, 2007):

$$k = \frac{\overline{\lambda}_{0m,f} \int_{0}^{\infty} p_{0m,f}(t) dt}{\int_{0}^{\infty} \overline{\lambda}_{0,m,f}^{ref}(t) p_{0m,f}(t) dt}.$$
(10)

In this case,  $p_{0m,f}(t)$  is the probability of survival for males and females in Rivne was up to 1 year t,  $\bar{\lambda}_{0m,f}$  is the average mortality rate per year for males and females in Rivne;  $\bar{\lambda}_{0,m,f}^{ref}$  is the average mortality per year for males and females of the "sample population". The calculated coefficient k for the residents of Rivne region in 2014 is 4.83. The calculations of the lifetime cancer risk, R for the risk probability P = 0.784 *WLM/year* in the residential premises of Rivne for the selected model are presented in Table 2 and Figure 3.

Table 2. Dependence of excess relative risk *R* on the time of irradiation and the selected model.

<i>t,</i> years							
	Model I		Model II	Model III		Model IV	
	EAD	EAC		1	2	1	2
10	0.000	0.000	0.000	0.000	0.000	0.000	0.000
15	0.000	0.000	0.000	0.000	0.000	0.000	0.000
20	0.000	0.000	0.000	0.000	0.000	0.000	0.000
25	0.000	0.000	0.000	0.000	0.000	0.000	0.000
30	0.000	0.000	0.000	0.000	0.000	0.000	0.000
35	0.000	0.001	0.000	0.000	0.000	0.000	0.000
40	0.002	0.003	0.002	0.001	0.000	0.000	0.001
45	0.006	0.008	0.004	0.003	0.002	0.001	0.001
50	0.015	0.023	0.009	0.007	0.007	0.004	0.004
55	0.026	0.040	0.014	0.014	0.021	0.008	0.008
60	0.045	0.072	0.025	0.029	0.061	0.015	0.015
65	0.062	0.098	0.038	0.036	0.152	0.027	0.027
70	0.085	0.134	0.056	0.046	0.325	0.041	0.041



**Figure 3.** Dependence of lifetime excess relative risk *R* on the time of irradiation and the selected model. JEMC – Model II, JEMNCC – Model III, JERR – Model IV.

There are a number of techniques available for estimating the number of deaths from lung cancer associated with indoor Radon exposure. We have applied the methodology proposed in (Catelinois et al., 2006), which offers the following formula for calculating death rates:

$$N_{r,a,d,s} = \frac{R_{r,a} \cdot N_{a,d,s}}{1 + R_{r,a}},$$
(11)

Where  $N_{r,a,d,s}$  is the number of deaths from lung cancer due to indoor irradiation with Radon at age *a*, in a house in the territory with number *d* and for sex *s*.  $R_{r,a}$  is an additional relative risk for people of age *a* who have been exposed to Radon *r*.  $N_{a,d,s}$  is the total number of deaths from lung cancer at age *a* in the territory with number *d* and sex *s*. The number of deaths in the city of Rivne due to the inhalation of Radon, when using the data for 2014, is presented for different models in Table 3. Throughout that year, 159 males and 36 females died from lung cancer in the city.

**Table 3.** Mortality from lung cancer in 2014 calculated by different risk models during 70 years of Radon exposure (Rivne, P = 0.784 WLM/year).

	Model I		Model II	Model III		Model IV	
	EAD	EAC		1	2	1	2
Males	12.46	18.79	8.43	6.99	39	6.26	6.26
Females	2.82	4.25	1.91	1.58	8.83	1.42	1.42

## Discussion

Lung cancer is the most common cause of cancer deaths for males and the third most common cause of death for females in Europe; it is responsible for almost 26% of all cancer deaths for males and 13% of all cancer deaths for females (Ferlay et al., 2013). A significant percentage of lung cancer deaths are accounted for the indoor Radon inhalation by a person (Ferlay et al., 2007). In the European Union, a comprehensive long-term measurement of indoor Radon concentration (Table 4) was conducted within the framework of the Anti-Radon Protection Programs (UNSCEAR, 2000; WHO, 2006). In many EU Member States, such long-term indoor Radon measurements are typically carried out over a period of at least three months and preferably during the heating season, when Radon levels are the highest. In these cases, the annual mean value can be obtained by applying seasonal correction coefficients. In some EU countries, one-year measurements are preferable to obtaining an average annual Radon concentration. The overall experimental approach was to place one Radon detector in the main living room and the other in the main bedroom. Due to differences in survey characteristics across countries, it was not possible to calculate the weighted average concentration of Radon in the EU premises, but it is probably close to 50  $Bq/m^3$  (Laughlin & Bochicchio, 2007).

Table 4. Indoor Rado	n concentration	in 25 European	countries* and Rivne.
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	Mean, <i>Bq/m³</i>	Geometric Mean, <i>Bq/m</i> ³		Mean, <i>Bq/m</i> <sup>3</sup>	Geometric Mean, <i>Bq/m</i> ³
Austria	102	n/a	Belgium	48	38
Cyprus	7	7	Czech Rep.	118	n/a
Denmark	53	29	Estonia	120	92
Finland	120	84	France	62	41
Germany	50	40	Greece	55	52
Hungary	107	82	Ireland	91	37
Italy	70	52	Latvia	n/a	n/a
Lithuania	32	22	Luxembourg	110	70
Malta	n/a	n/a	Netherlands	23	18
Poland	41	32	Portugal	62	45
Slovakia	87	n/a	Slovenia	87	60
Spain	45	42	UK	23	10
Sweden	108	56	Rivne City (Ukraine)	145.6	127

\* (UNSCEAR, 2000; WHO, 2006)

It is evident that the city has an extremely complex Radon situation. This is due to the geological structure of the territory on which the city is located, represented by Proterozoic, Paleozoic, Mesozoic and Cenozoic sediments within the Volyn-Podilsky Plate on the Rivne Forest Plateau. This Volyn-Podilskyi Plate is the western slope of the Ukrainian crystalline shield rich in Uranium and Radium deposits, broken by a complex system of cracks, which could cause Radon release from Radium decay into surface soils and later there into homes. The main soil-forming rocks of Rivne are loess, which have high concentration of Radon. The hilly terrain and considerable landscaping cause low breeziness, so the "wind tunnel", which causes the accumulation of Radon in the lowlands, does not work. Such geological conditions are responsible for the significant concentrations of Radon in household air and, consequently, for the increased risk of lung cancer in the city. There are various methods for assessing the risks of Radon induced lung cancer. For example, some EU Member States, such as the Czech Republic and Sweden, use risk classification based on the factors such as soil permeability (Radon diffusion coefficient), soil Radium-226 activity concentrations in rooms in future buildings and is reduced to determining the so-called Radon potential (Miksova & Barnet, 2002).

Although there are currently about 20 such techniques and models, we still consider the case-control models be the most reliable. In our study, we calculated the excess relative risk of lung cancer for Rivne up to 70-year residents, since the average life expectancy of residents in our region is 71 years. In most of the publications considered the residents of the EU, North America, Japan, and South Korea, such calculations were made for the residents up to 95 years old. An additional barrier to properly determine the Radon induced risks of lung cancer for the population age above 70 was official information restriction on death rates from lung cancer for this group. We should to mention that BEIR-VI risk calculations were also based on the age more than 75 years old (National Research Council, 1999). The performed calculations showed that in the scenario when person spent all his life on the first floor in Rivne for 70 years at the risk probability P = 0.784 WLM/year, the excess relative risk of lung cancer due to Radon increases from 4.1 (IV) to 32.5% (III model 2). The R = 2 for model III is in doubt, possibly due to incorrectly selected coefficients, as was discussed above. The greatest risk values for the EAS model are presented in some research, for example in Hunter et al. (2015). The R values we have calculated for this model have been adjusted for 70 years of exposure. According to EU estimates (Darby et al., 2006), the number of deaths from Radon induced lung cancer is about 9% (95% CI = 3-17%) compared to the total lung cancer deaths. According to our estimates, the mortality rates could be between 3.9 and 24.5% for males and females (based on 2014 mortality data).

# Conclusion

The study confirmed that high concentrations of Radon in household air increase the risk of lung cancer. We found that the average Radon exposure at houses was 0.784 *WLM/year*. This value determined the excess relative risk of lung cancer for the urban residents. We established that Radon exposure increased nonlinearly during 70 years of human life from 0 to 0.325, which lead to mortality rate in range of 3.9-24.5% for all Rivne residents died from the lung cancer.

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