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ORIGINAL ARTICLE

# Radon exposure and lung cancer: analysis of risk for residents of Rivne City (Ukraine)

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The analysis of risk from radon exposure on lung cancer among mining populations of world mines and residents of the city of Rivne was conducted. The calculation of additional relative risk for the residents of the city of Rivne by the method of epidemiological ecological studies performed on radiometric equipment "AlfaRad+" and calculated according to the BEIR VI risk model in two variants: "exposure-age-duration" (EAD) and "exposure-age-concentration" (EAC) was conducted. The values of the «risk coefficients» of the dependencies of the relative risk on radon exposure we obtained for residents of Rivne are lower than the ones obtained for miners (according to literature) were 0.31 (EAD) and 0.49 (EAC) per 100 Working Level Month (WLM), while the volumetric activity (VA) of radon in domestic conditions were 200 Bq/m<sup>3</sup> and 1420 Bq/m<sup>3</sup>. **Key words:** Radon, cohort studies, additional relative risk, exposure, lung cancer

## Introduction

The problem of the influence of Radon presence in the air on human health has been studied for about 60 years. Such research has begun on workers of uranium mines (mainly) and the ones of other metals. The first quantitative analyzes of epidemiological studies of the incidence of lung cancer in the uranium miners of the United States and Czechoslovakia were published only in the early 70's of the last century (Lundin, 1971). The most detailed pooled epidemiological analysis of the association of miners' lung cancer with the exposure dose of derivative products of Radon decay (*DPD*) in uranium and non-uranium mines was conducted in Lubin's works (Lubin et al., 1994), in Report IV of the Commission on Biological Effects of lonizing Radiation (BEIR IV) and in Report VI of the Commission on Biological Effects of lonizing Radiation (BEIR IV) and in Report VI of the Commission on Biological Effects of lonizing Radiation (BEIR IV) were based on studies on four miners' cohorts and the ones reported in BEIR VI were based on over 11 cohorts (see Table 1). Cohort studies on miners tracked the incidence of lung cancer in a population (cohort) exposed to the test factor (Radon exposure) and compared with the incidence in the control group. The part of the cohort that was exposed to the lowest levels of influence was used as the control group (for example, the experimental group consisted of the passers-by whereas the control one consisted of the repairmen).

able 1. Death from lung cancer in mining cohorts.												
Mine location	Mine type	Number of	Life years	Death from lung cance								
		workers										
Yunnan (China)	Tin	13,649	134,842	936								
Czechoslovakia	Uranus	4,320	102,650	701								
Colorado Plateau * (USA)	Uranus	3,347	79,536	334								
Ontario (USA)	Uranus	21,346	300,608	285								
Newfoundland (Canada)	Fluorite	1,751	33,795	112								
Malmberget (Sweden)	Iron	1,294	32,452	79								
New Mexico (USA)	Uranus	3,457	46,800	68								
Beaverridge (Canada)	Uranus	6,895	67,080	56								
Port Radium (Canada)	Uranus	1,420	31,454	39								
Radium Hill (Australia)	Uranus	1,457	24,138	31								
CEA-COGEMA (France)	Uranus	1,769	39,172	45								
Total**		60,606	88,890	2,674								

\* - the influence is limited by the level < 3,200 *WLM*; \*\* - cases of cure of the disease were taken into account in the results of Colorado and New Mexico.

The results of the cohort study allow calculating the relative risk *RR* as the ratio of the risk of appearance of the mortality effect in the experimental group (*A*) to the one in the control group (*B*):

$$R = \frac{A}{B}$$

where *A* and *B* was calculated through dividing the number of people who died from lung cancer by the number of people years in the experimental group and the control one respectively (see Table 2).

Sources	Cohort number	Miner quantity	Life years	( <i>RR</i> – 1) per 100 <i>WLM</i>	Confidential interval
ICRP, 1993 (Protection, 1995)	7	31,486	635,022	1.34	0.82-2.13
Lubin et al. 1994	11	60,570	908,903	0.49	0.20-1.00
Health, 1999	11	60,705	892,547	0.59	1.32 (standard
					error)
UNSCEAR – 2009 (Effects, 2009)	9	125,627	3115,975	0.59	0.35-1.00
Tomachek et al. 2008	2	10,100	248,782	1.60	1.00-2.30

Table 2. Generalized results of surveys conducted among miners, according to the publication ICRP 115 (The risk, 2013).

Related parameters in the cohort studies were the individual data of exposed miners: gender, age, operating time, smoking, and other characteristics. Thanks to them, the influence of various factors on the parameters of the dose-effect dependence was investigated. United cohort studies and some individual studies have shown that the following factors influence the relationship between radon exposure and lung cancer:

a) work experience in the field;

b) the age at which the irradiation began;

c) the elapsed time after irradiation;

d) the age reached.

One can estimate the relative risk *RR* using the expression that is the following:

 $RR = 1 + \beta \cdot P$ ,

(2)

(1)

where P-radon exposure,  $\beta$  - so called, «risk-coefficient».

Overall, the results of cohort studies of radon exposure in miners showed a statistically significant association between cumulative radon exposure and lung cancer mortality at exposure levels of 50 *WLM* and above (The risk of appearing ..., 2013). One of the most important results of cohort studies should be the conclusion that lung cancer is a significant stochastic effect of radon irradiation and its *DPD*.

#### Analysis of recent research

In the 1990s, due to intensive anti-radon activities in the mines, the average annual exposure of uranium miners to radon fell to a level commensurate with the effect of radon on humans in some homes of the regions of United States, Northern Europe, and Russia. The radon exposure of miners began to correspond to long exposure in the dwelling with an average annual volumetric activity of radon in it of about 200 Bq/m<sup>3</sup>.

The question arose about the possibility of transferring known information about the effect of radon radiation on miners, as a specific cohort, on the general population. But such a mechanical transfer of cohort survey results obtained for miners to the general population was contradicted by a number of significant differences between these populations (The risk, 2013):

1. Miners are almost all adult men (20 to 40 years old);

2. Equivalent equilibrium volume activity of Radon (EEVARn) in mines typically exceeds the levels that are typical for most homes;

3. The proportion of smokers among miners is well above the average population level;

4. The intensity of breathing when working in a mine is higher than when in a home;

5. Pollution of the atmosphere of the mines is much higher than in any dwelling, and the dispersed composition of Radon *DPD* differs significantly in them;

6. Other carcinogens may be present in the mine atmosphere: dust that may contain long-life natural radionuclides, diesel exhaust, and arsenic.

In this regard, there was a need for separate epidemiological studies of the relationship between the likelihood of developing lung cancer in humans and a radiation dose from radon in a residential setting. For the work in this direction, the most appropriate method was the case-control cohort study. The essence of this approach is based on determining the levels of radon exposure in a cohort of individuals with identified cases of lung cancer and comparing these levels with the levels of radon exposure for a control group (Auvinen et al., 1996; Kreienbrock et al., 2001; Letourneau et al., 1994; Schoenberg et al., 1990), which parameters match the ones of the main group. This type of research is considered to be the most valid for epidemiological assessments in the case of household radon exposure.

In the case-control study, the main group included individuals with the disease under study (e. g. diagnosed with lung cancer), and the control group included individuals who did not have the disease. For example, in (Field et al., 2000) in 1993-1997, such studies were conducted in lowa (USA) among females who have been living in their homes for at least 20 years (413 people with detected lung cancer are the main group and 614 healthy individuals – control group). Cases of the disease were selected from the lowa Cancer Association Register, and the age-matched control group was from drivers licensed by the lowa Department of Transportation. Of course, the sample of both the main and control groups was much more powerful, but it was

these numbers of people who agreed to install radon detectors in their homes for one year. The association of influence factor and disease (*OR*) is characterized by the ratio of odds (chance corresponds to the frequency of occurrence of the effect):

 $OR = \frac{A/B}{C/D}$ 

(3)

where *A* is the number of members of the core group affected; *B* is the number of members of the core group who were unaffected; *C* is the number of members of the control group affected; *D* is the number of members of the control group who were unaffected.

However, it should be understood that in most cases it is not possible to achieve the identity of the research conditions for the control and main groups. About 40 case-control studies related to radon in residential areas have been conducted in recent decades (Auvinen et al., 1996; Kreienbrock et al., 2001; Letourneau et al., 1994; Schoenberg et al., 1990). Part of the results is shown in Table 3. Based on this data, we can draw some conclusions, in particular that are the following:

experimental evidence suggesting an increase in the incidence of lung cancer from Radon exposure in the home is still lacking; low radon exposure in most homes around the world is a cause of great uncertainty in the risk assessment process; the high cost of case-control studies limits the number of regions studied (mainly the USA, Canada and EU countries (Health, 1999; Darby et al., 2005; Krewski et al., 2005).

Given the relatively low volume activity (*VA*) of radon in residential areas, it is to be expected that the additional risk of lung cancer is also low. Therefore, in order to identify the risk associated with radon against other factors and taking into account the stochastic nature of carcinogenesis, a very large, approximately 10,000 study groups is needed. In practice, it is impossible to form and investigate such the numerous groups of lung cancer patients within a framework of an epidemiological study of one territory. That is why none of the case-control studies had sufficient statistical power to draw conclusions about the presence and magnitude of the effect of radon influence. In addition to case-control studies, environmental (geographically correlated) studies are widely used (Neuberger, 1991; Stidley & Samet, 1993; Neuberger et al., 1994), based on a comparison of lung cancer mortality for different urban ecosystems with different mean values Radon *VA* indoors (mainly in residential buildings). Because of their relative simplicity and low cost, this type of research is quite common. However, the influence of a large number of additional factors, as a rule, does not allow evaluating their results correctly.

Table 3	Case-	control	studies fo	or rador	and	lung	cancer	in sn	nokers	and	non-smo	kers
Table J.	cuse-	Control	studies it	1 10001	anu	lung	cancer	111 211	IIUKCI 3	anu	1011-31110	KCI S.

Source	Country	Gender	Case	Control	Odds ratio (95 % Cl)	Mean/m indoor	nedian of radon	Duration of radon		
						level (	Bq/m³)	_ measurements		
						Case	Control	(months)		
Blot et al., 1990	China	Female	308	356	0.7 (0.4–1.3)	85		12		
Schoenberg et al. 1990	USA	Female	433	402	4.2 (0.99–17.5)	*NA		12		
Pershagen et al., 1992	Sweden	Female	210	209	1.7 (1–2.4)	128		3		
Létourneau et al., 1994	Canada	Both	738	738	0.77 (0.34–1.73)	120		12		
Pershagen et al., 1994	Sweden	Both	1360	2847	1.8 (1.1–2.9)	107		3		
Alavanja et al., 1994	USA	Female	247	299	0.71 (0.3–1.3)	57	60	12		
Auvinen et al., 1996	Finland	Both	517	517	1.15 (0.69–1.93)	103	96	12		
Ruosteenoja et al., 1996	Finland	Male	291	495	1.5 (0.8–2.9)	213		12		
Darby et al., 1998	GBR	Both	960	3126	1.79 (0.74–4.33)	58	56	6		
Field et al., 2000	USA	Female	413	614	1.79 (0.99–3.26)	100	89	12		
Pisa et al., 2001	Italy	Both	138	291	1.0 (0.3–3.1)	*NA		12		
Barros-Dios et al., 2002	Spain	Both	163	241	2.96 (1.29–6.79)	75	66	3		
Wang et al., 2002	China	Both	768	1659	1.58 (1.1–2.3)	230	222	`12		
Baysson et al., 2004	France	Both	486	984	1.11 (0.59–2.09)	83	80	6		
Bochicchio et al., 2005	Italy	Both	384	405	2.89 (0.45–18.6)	113	113	6		
Wichmann et al., 2005	Germany	Both	2963	4232	1.4 (1.03–1.89)	61	60	12		
Sandler et al., 2006	USA	Both	1474	1911	1.00 (0.93–1.07)	40	45	12		
Thompson et al., 2008	USA	Both	200	397	2.5 (0.47–13.46)	68	66	12		
Wilcox et al., 2008	USA	Both	561	740	0.76 (0.36–1.61)	46	46	12		
Barros-Dios et al., 2012	Spain	Both	349	513	2.21 (1.33–3.69)	*NA		3-6		
*Not applicable										

The purpose of our study was to assess the risk of lung cancer caused by radon exposure in the Rivne city by means of various methods and implement the results of cohort studies of miners to the population of Rivne region.

#### Materials and methods

To calculate the risk *R* (likelihood) of appearance of lung cancer that is radiation-induced by irradiation of *DPD* of Radon throughout human life in ecological radon studies the following quantities are used: probability of survival to a certain age  $p_0(t)$ , frequency of spontaneous occurrence of lung cancer  $\lambda_0(t)$ , and coefficient of additional relative risk  $K_{ERP}$  (t) from the selected model of risk of radon lung cancer. Such the calculation should take into account the additional reduction in the expected probability of survival to age *t* due to the occurrence of additional cases of lung cancer from other factors:

$$R = \int_{0}^{\infty} \lambda_0(t) \cdot p_0(t) \cdot K_{ERR}(t) \cdot e^{-\int_{0}^{t} k_{kr} \cdot \lambda_0(t') K_{ERR}(t') dt'} dt$$
 (4)

In equation (4), it is proposed to use the coefficient of lethality  $k_{let} = 0.95$  for the transition from radiation-induced morbidity to additional mortality (Radiation safety..., 1994). Lethality coefficients  $k_{let}$  are based on data from the US National Cancer Institute, which reported 5-year (1980-1985) survival rates at different locations (program SEEP). For lung and bronchial cancer, the mortality was 87% and, accordingly,  $k_{let} = 0.87$ . These values were too low to fully express mortality. But there are also mortality data for the period 1950-1970, which are too high according to the current standards (mortality – 96%,  $k_{let}$ , = 0.96 since in our time the degree of cure has improved compared to this earlier period.  $k_{let}$  is offered at the level  $k_{let}$ . = 0.95. If in this expression (4) we go from integration to summation, then it will take the following form:

$$RR - 1 = R = \sum_{t=0}^{\infty} \lambda_0(t) p_0(t) K_{ERR}(t) \cdot \exp\left[-\sum_{t'=0}^{t} k_{let} \lambda_0(t') K_{ERR}(t')\right].$$
 (5)

The survival function  $p_0(t)$  is defined as the probability of reaching the age t (from birth) on the part of a person. It takes into account the demographic characteristics of the region, as well as the fact that death is determined by various causes, not just by the influence of the radiation factor. In practice, the probability of living up to the age t on the part of a person is calculated according to such the expression:

$$p_0(t) = \prod_{i=1}^{t} p(i),$$
 (6)

where p(i) is the probability of not dying during the i-the year of his or her life. In the absence of radiation, the probability of surviving up to the age *t* (from birth) is given by a function  $p_0(t)$  and can be calculated according to the formula:

$$p_0(t) = \exp\left(-\int_0^t q_0(\tau)d\tau\right),\tag{7}$$

where  $q_0( au)$  – mortality at a certain age au from all causes, in a particular population that was not exposed to irradiation.

The additional relative risk coefficient K<sub>ERP</sub> (t) was calculated according to the BEIR VI model (Health Effects..., 1999). Depending on the input parameters, this model has two options:

- EAD the relative risk depends on the time elapsed since the exposure has been reached, the age and duration of the exposure («Exposure Age Duration» model).
- EAC the relative risk depends on the time elapsed since the exposure has been reached, the age, and the equivalent equilibrium VA of Radon (*EEVA<sub>Rn</sub>*) in the irradiation process («Exposure Age Concentration» model).

In this model the general form of the function  $K_{ERP}$  (t) f (x) is offered in the form:

$$K_{ERR}(t) = \beta (P_{5-14} + \theta_{15-24} \cdot P_{15-24} + \theta_{25+} \cdot P_{25+}) \cdot \varphi_{age} \gamma_Z,$$
(8)

where  $\beta$  – the main parameter of the dependence «exposure – response» («risk-factor»);

 $P_{5-14}$ ,  $P_{15-24}$ ,  $P_{25+}$  - exposure windows that determine the cumulative exposure of Radon and its *DPD* obtained at intervals of 5-14 years to time *t*, 15-24 years to time *t* and 25 years or more to the time for which risk is assessed;

 $9\Theta_{15-24}$ ,  $\theta_{25+}$ - coefficients that represent the relative contribution to the risk of lung cancer exposures obtained at these intervals of time to age t;

parameter  $\varphi_{age}$  determines the dependence of the carcinogenic susceptibility of the lung tissue on the reached age; the parameter Y<sup>z</sup> depends either on the duration of the exposure (in years) or on the exposure level *EEVA<sub>Rn</sub>* at which the received dose was generated. The BEIR VI model also incorporates a five-year latency period for the development of lung cancer, so exposures obtained over the last 5 years before age t are not taken into account in expressions (1-3). As a flaw of this model one can consider taking into account the fixed (average) proportion of smokers regardless of the country (58% of males and 42% of females). Radon exposure is determined by the formula that is the following:

$$P_{WLM} = \int_{0}^{T} EEVA_{Rn} dt = \left( EEVA_{Rn} \right)_{av} \cdot T = F_{Rn} \cdot \left( VA_{Rn} \right)_{av} \cdot T$$
(9)

where  $EEVA_{Rn}$  is time-varying Radon EEVA value; ( $EEVA_{Rn}$ )<sub>av</sub> is Radon EEVA averaged over the exposure time *T*; ( $VA_{Rn}$ )<sub>av</sub> is the average value of indoor Radon volume activity;  $F_{Rn}$  is the coefficient of equilibrium between Rn-222 and its *DPD*.

Indoor equilibrium coefficient varies from 0.14 to 0.6 and its average lies in the range 0.4–0.5 (Tsapalov et al., 2010). According to theoretical calculations for US value of F is recommended to be equal to 0.4. We used the value F = 0.5 because we believe that the climatic conditions of the Rivne region are close to the climatic conditions of the southern and central regions of Russia, for which this value is used. The conversion of the measured values of the average the exposure  $P^{(bq hour)}/m^3$  averaged over one year to the exposure value in *WLM* for the population  $P^{(population)}$  is given by the formula that is the following (Demin et al., 2014; Lipnitsky & Kostitskaya, 2004):

$$P^{(populatio)}\left(\frac{WLM}{year}\right) = \frac{EEVA_{av}\left(\frac{Bq}{m^3}\right) \cdot T(hour)}{170 \cdot 3700}.$$
 (10)

Then:

$$P^{(populatio)}\left(\frac{WLM}{year}\right) = \frac{EEVA_{av}\left(\frac{Bq}{m^3}\right) \cdot 24 \cdot 365}{170 \cdot 3700} = 0.0139 \cdot F_{Rn} \cdot VA_{av}\left(\frac{Bq}{m^3}\right) = 0.00696 \cdot VA_{av}\left(\frac{Bq}{m^3}\right).$$
(11)

Adjusting for the measurement season we measured volumetric activity (*VA*) of Radon using an «AlfaRad +» Radonometer in 600 premises of the housing and production facilities of 48 test sites of Rivne (between them there were the premises located on the first floor, as well as partially and fully underground) during 2013-2017 (Lebed et al., 2018; Lebed et al., 2019). The statistical distribution parameters for *VA* of Radon isotope derivative products that were determined are the following: the mathematical expectation (*VAmh*) for *VA*, the geometric standard deviation of *VA(\sigma)*, and the predicted maximum value of *VAmax*. As the last parameter, the value at the threshold of the probability density that corresponds to 3 $\sigma$  deviation from the maximum of the distribution was taken (the "three sigma rule").

The arithmetic mean of VA of all the premises under study was 262.5  $Bq/m^3$  with a standard deviation of 194.4  $Bq/m^3$ , a geometric mean of 200  $Bq/m^3$  and the geometric standard deviation  $\sigma = 0.7865$ . The maximum measured value of VA<sub>measured</sub> is 1000  $Bq/m^3$ , according to the results of our measurements the estimated maximum value of VA<sub>max</sub> is estimated to be no more than 1420  $Bq/m^3$ .

The measured  $VA_{mh}$  and  $VA_{max}$  values served as benchmarks for the calculation of the risk of suffering from the lung cancer on the part of the city population (Lebed et al., 2018; Lebed et al., 2019). For benchmarks  $VA = 200 Bq/m^3$  and  $VA = 1420 Bq/m^3 P^{(H)}$  is 1.392 WLM year<sup>-1</sup> and 9.88 WLM year<sup>-1</sup> respectively.

#### **Results and discussion**

The calculated values of  $K_{ERR}$  (t) and  $p_0$  (t) for Rivne residents for 2014 are shown in Table 4.

The statistical information from medical institutions about lung cancer mortality among residents of the city of Rivne is insufficient for calculating the age-specific incidence of lung cancer  $\lambda_0(t)$  specific to Rivne,. As a working hypothesis in this case,

we used the assumption that the age-specific lung cancer incidence for any particular region corresponds to the age-specific morbidity  $\lambda_0^{ref}(t)$  for the so-called «sample population» given in (ICRP Publication 50..., 1987) differing from it only by the constant factor *k*:

$$\lambda_0(t) = k \lambda_0^{ref}(t), \tag{12}$$

The coefficient k calculated by us for 2014 was 4.83, and the values of  $\lambda_0^{ref}(t)$  are shown in Table 5.

We will use the resulting formula to determine the additional relative risk of appearing on the part of lung cancer due to Radon. The results of calculations of the additional relative risk of appearing on the part of radiation-induced lung cancer due to Radon *DPD* radiation R during a human life in environmental Radon studies according to the formula (11) for the benchmark values *VAmh* and *VAmax* are presented in Tables 6 and 7.

t , years	E	AD	E	Po(t)	
	VA <sub>mh</sub> = 200	VA <sub>max</sub> = 1420	VA <sub>mh</sub> = 200	VA <sub>max</sub> = 1420	
5	0	0	0	0	0.98804
10	0.1064	0.7553	0.5345	3.794	0.98683
15	0.3384	2.4018	1.0691	7.5878	0.98549
20	0.4602	3.2665	1.486	10.5471	0.98210
25	0.8717	6.1874	1.903	13.5064	0.97593
30	0.9833	6.9788	2.1755	15.4412	0.96526
35	1.6868	11.972	2.4481	17.3762	0.94971
40	1.8586	13.1916	2.7208	19.311	0.93189
45	2.0304	14.411	2.9934	21.246	0.91030
50	2.2022	15.6304	3.2659	23.1809	0.88078
55	1.2345	8.7619	2.0277	14.3919	0.83630
60	1.3238	9.396	2.1724	15.4189	0.77674
65	0.7609	5.4008	1.1843	8.4058	0.71703
70	0.809	5.7422	1.2528	8.8918	0.66250

**Table 4**. The values of the additional relative risk ratio  $K_{ERP}$  (t) and the lifetime function  $p_0(t)$  for Rivne residents for 2014, depending on the time of Radon *DPD* exposure and benchmarks values of *VA* (*Bq/m*<sup>3</sup>) in residential buildings of the city of Rivne, calculated in the framework of the BEIR VI model for the whole population.

**Table 5.** Age-specific frequency of appearing spontaneously on the part of the lung cancer  $\lambda_0^{ref}(t)$  in "sample population" without the influence of Radon (ICRP Publication 50..., 1987)

Age, years	$\overline{\lambda}_0^{ref}(t) \cdot 10^{-5}$
30	< 0.1
35	0.85
40	2.75
45	7.5
50	18.5
55	36.5
60	70
65	115
70	160

If we expend the expression (5) in terms of MacLaren's series and leave only first two terms, we obtain the following:

$$R = \sum_{t=0}^{\infty} \lambda_0(t) p_0(t) K_{ERR}(t) \cdot \left( 1 - \sum_{t'=0}^{t} k_{let} \lambda_0(t') K_{ERR}(t') \right) =$$
  
= 
$$\sum_{t=0}^{\infty} \lambda_0(t) p_0(t) K_{ERR}(t) - \sum_{t=0}^{\infty} \lambda_0(t) p_0(t) K_{ERR}(t) \cdot \sum_{t'=0}^{t} k_{let} \lambda_0(t') K_{ERR}(t')$$
(13)

Table 6. The value of RR-1, determined in the framework of the ecological methodology for residents of Rivne with VA = 2	200
<i>Bq/m<sup>3</sup></i> and calculated according to two variants of the BEIR VI model.	

Model	Life years													
	5	10	15	20	25	30	35	40	45	50	55	60	65	70
EAD	0	0	0	0	0	0	0	0.01	0.02	0.06	0.09	0.17	0.23	0.31
EAC	0	0	0	0	0	0	0	0.01	0.03	0.08	0.15	0.26	0.36	0.49

**Table** 7. The value of RR-1, determined in the framework of the ecological methodology for residents of Rivne with VA = 1420  $Bq/m^3$  and calculated according to two variants of the BEIR VI.

Model	Life years													
	5	10	15	20	25	30	35	40	45	50	55	60	65	70
EAD	~0	~0	~0	~0	~0	~0	0.01	0.05	0.14	0.40	0.66	1.17	1.60	2.19
EAC	~0	~0	~0	~0	~0	~0	0.02	0.07	0.21	0.59	1.02	1.84	2.52	3.42



**Fig. 1** Calculation of the relative risk for miners by the cohort method (1-5) and for residents of Rivne city (at  $VA = 200 Bq/m^3$  in domestic) by the environmental method (6-7): 1 – ICRP..., 1993; 2 – Lubin et al., 1994; 3-4 – Health, 1999 and Effects, 2009; 5 – Tomachek et al., 2008; 6 – EAC; 7 – EAD.



**Fig. 2** The relative risk of lung cancer, calculated by the cohort method (1-5) for miners and environmental method (6-7) for residents of Rivne City (at VA = 1420  $Bq/m^3$  in housing), depending on the cumulative WLM exposure obtained / the number of life years with the respective VA: 1 – ICRP, 1993; 2 – Lubin et al., 1994; 3-4 – Health, 1999 and Effects, 2009; 5 – Tomachek et al., 2008; 6 – EAC; 7 – EAD.

### Conclusions

The studies and calculations performed in this paper show that the additional relative risk of appearing on the part of lung cancer triggered by Radon for residents of Rivne is slightly less than the one calculated according to multiple cohort studies for miners. The methodology of epidemiological ecological studies and radiometric equipment «AlfaRad +» were used, the calculations were performed in the framework of the BEIR VI risk model in two variants: «exposure-age-duration» – EAD and «exposure-age-concentration» – EAC. As a result, the values of the «risk coefficients» of the dependencies of the relative risk on Radon exposure we obtained for residents of Rivne are lower than the ones obtained for miners (according to literature) – 0.31 (EAD) and 0.49 (EAC) per 100 *WLM* for volumetric activity (*VA*) of Radon in housing 200 *Bq/m*<sup>3</sup> and 1420 *Bq/m*<sup>3</sup>. This indicates that the impact on the population Radon has is smaller than the one on the miners it has. The values of the data (Lubin et al., 1994) are the closest to the ones we obtained.

If we take the whole array of research data obtained in the frameworks of cohort, «case-control» and environmental studies over the past decades, we should admit the inability to automatically apply the Radon exposure dependencies of the additional relative risk of appearing on the part of lung cancer obtained for miners to the general population according to variants of risk models offered in BEIR VI. Thus, one can assert that accurate epidemiological surveys are required for each specific territory in order to accurately assess the impact of Radon on the health of the population, and that the data obtained for miners can only be used as a guideline.

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