Probability of Radon cancer occurrence in Rivne city residents (Ukraine), calculation by WISMUT and Radon-2014 models

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The aim of the study: Inhaling 222Rn present in the premises on the part of the population may provoke the development of cancer of the respiratory tract. This study assessed the risk of morbidity from lung cancer and the mortality of residents of Rivne, Ukraine, due to exposure to internal radioactive irradiation.

Methods: The biological model of the human respiratory tract was used and, based on it; the physical models of the additional relative risk of morbidity from lung cancer of the population of urban areas due to Radon were constructed. The constructed models include the model BEIR VI in two variants (TSE/AGE/DUR and TSE/AGE/WL), models WISMUT and Radon-2014.

Results: The geometrical mean of volume activity VA of Radon in the premises of the city of Rivne we measured equals 200 Bq/m³. It corresponds to the value of the average exposure to Radon equal to 1.392 WLM/year. In the framework of the model TSE/AGE/DUR, it is determined that residing in such the premises on the part of the population may provoke the development of the deaths of the residents of the city of Rivne with lung cancer due to exposure to Radon in the premises of the city depends on the uncertain dependence of the effect on the dose and ranges from 5 to 128 persons per year for the males and from 1 to 23 people per year for females. This is from 3 to 64% of all lung cancer deaths per year due to all causes during the study period for males and from 3 to 77% for females. Such the research should be extended to other regions of Ukraine as the Radon problem is a matter of serious concern to the European Community and the World Health Organization.

Conclusion: This research is the first estimation of the risk of morbidity and mortality from cancers of trachea, bronchi, and lungs due to exposure to Radon in the city of Rivne performed in the frameworks of the models WISMUT and Radon-2014. The quantity of the deaths of the residents of the city of Rivne with lung cancer due to exposure to Radon in the premises of the city of Rivne inclusive) regardless of the construction of the buildings, and its accumulation is connected with a complex of reasons that include the following:

- The presence of migratory routes of Radon flow from the sources to the premises;
- The action of the force of pressure which drives the Radon-containing air into the building;
- The presence of Radon intake channels inside the building shell materials;
- Low air exchange (ventilation) between the room and the outside atmosphere.

The average indoor Radon concentration values are (arithmetic mean) from 21 (UK) to 110 Bq/m³ (Sweden), which indicates that there are significant differences in this indicator between countries of the world. It has been established (The risk..., 2013) that...
Radon in the air of homes can be a significant and potentially dangerous factor determining the risk of population morbidity from cancer of respiratory system parts. Measuring such health risks is particularly important in shaping the health policies of the world's population and the allocation of available resources. The behaviour of Radon and its derivative products of decay (DPD) in confined spaces is determined by processes such as air exchange with the outside atmosphere, interaction with aerosols, and deposition of DPD on the surfaces of rooms. During α-decay of 222Ra, a positively charged 218Po ion with a kinetic energy of ~ 100 keV is formed. The length of the run of Polonium in the air is ~ 50 µm, in a few nanoseconds it loses energy when it collides with gas molecules and becomes neutral. However, during this time, it can adsorb water vapour molecules or combine with gas impurities, resulting in the formation of fine particles with sizes in the range from 0.5 nm to 5 nm. In addition, the decay products of Radon, migrating in the air, can collide with and adhere to atmospheric aerosol particles to form radioactive aerosols. Radon and Thoron enter the human body mainly via inhalation due to these aerosols. In this case, the dose due to DPD of Radon is approximately equal to 99% of the dose created by Radon. Thus, DPDs make a major contribution to exposure of internal organs to radiation. Short-lived DPDs decay mainly in the lungs prior to excretion. Two isotopes from 222Rn short-lived derivative products (218Po and 214Po) emit α-particles, which energy produces a significant dose of lung irradiation. Such the irradiation of the epithelium of the lungs leads to the oncological diseases, mainly adenocarcinoma and squamous cell carcinoma. To estimate the magnitude of the transition from the dose of exposure to Radon DPD to the effective dose, the methods of calculation with the usage of various models of the behaviour of radionuclides in the respiratory tract were used in the past. ICRP has developed a model of the human respiratory tract to calculate the radiation dose due to radionuclides present in the air (ICRP, 2015).

The variable that allows us to estimate the radiation impact of Radon DPD on humans is the so-called the exposure estimated according to Equivalent Equilibrium Volume Activity of Radon (EEVARN) exposure:

\[
P_{\text{WLM}} = \int_0^T \text{EEVA}_n \, dt = \left( \text{EEVA}_n \right)_{av} \cdot T
\]

where EEVARN is time-varying Radon EEVA value; (EEVARN)av is Radon EEVA averaged over the exposure time T; (VARn)av is the average value of indoor Radon volume activity;

Exposure can be determined in Bq × hour/m³, or in non-system units WLM. The number of deaths from lung cancer associated with Radon exposure in housing ranges from 150 (The Netherlands) to 40,477 people (South Korea) per year. According to estimates (Kim et al., 2016; Leenhouts and Brugmans, 2001) in the world from 3.3% (UK) to 20% (Sweden) of all lung cancer deaths are likely to be caused by exposure to Radon irradiation in the premises. The wide variation in estimates across countries may be driven by the use of the model of the dependence “exposure–effect” and the overall number of lung cancer deaths in every country. These data confirm that the exposure to Radon indoors poses a significant risk to public health. Indeed, the rate of deaths from lung cancer due to Radon may be higher than the rate of deaths from other cancers. For instance, the estimated number of deaths from lung cancer due to exposure to Radon in the United States is greater than the annual number of deaths from several cancers, including malignancies in the ovary, liver, brain, stomach, or melanoma (WHO, 2009). Three approaches to conducting complex epidemiological studies (Zhukovsky and Yarmoshenko, 1997), based on the dosimetric model of the human respiratory tract, have historically been used in the determination of Radon risks of human being morbidity from lung cancer:

a) Cohort studies for different occupational groups or the population in which it is assumed that individual radiation doses or average radiation doses for each cohort are known;

b) "case-control" based on the determination of levels of exposure to Radon in a cohort of persons (mainly for the population of urban areas) with identified cases of lung cancer based on medical data in the main group (case) and comparing them with levels of exposure to Radon for a control group which main parameters (residence time in the same area and in similar architectural plans, attitude to cigarette smoking, educational attainment, gender, age, etc.) match the ones of the main group. This type of research has been found to be the most valid for epidemiological assessments when irradiation due to Radon in the premises occurs but requires significant funding with the involvement of a large number of technical staff to collect information;

c) Ecological studies of identified territories based on a comparison of mortality from lung cancer for different territories with varying averages of indoor Radon volume activity (mainly in residential buildings) and the calculation of additional risks of morbidity from lung cancer due to Radon with the usage of formulas, proposed by the ICRP or by independent researchers. The influence of a large number of additional factors, as a rule, does not allow to correctly estimating the results of such the calculations, but they are permissible for assessing the ecological state of the territory with regard to this indicator.

The first quantitative analyses of epidemiological cohort studies of morbidity from lung cancer were conducted for uranium mines in the work of (Lundin et al., 1971). Cohort studies in the 1970s concluded that the risk of morbidity from lung cancer increases monotonically with the increase in exposure to DPD of Radon. The most detailed pooled epidemiological analysis of the association between the morbidity from lung cancer and exposure to DPD of Radon published only in the early 70's of the last century (Lundin et al., 1971). Cohort studies in the 1970s concluded that the risk of morbidity from lung cancer increases monotonically with the increase in exposure to DPD of Radon. The most detailed pooled epidemiological analysis of the association between the morbidity from lung cancer and exposure to DPD of Radon in uranium and non-uranium mines was made in the work of (Lundin et al., 1994), in the IV Report of the Commission on Biological Effects of Ionizing Radiation (BEIR IV) and in the VI Report of the Commission on Biological Effects of Ionizing Radiation (BEIR VI) (National Research Council, 1999). Speaking of epidemiological studies of cohort of miners, it should be noted that one of the valuable results of these studies was the conclusion that lung cancer is the only significant stochastic effect of irradiation due to Radon and its DPD. Based on cohort studies of miners conducted in 1986, the World Health Organization recognized Radon as a first-class carcinogen and the second most important cause of morbidity from cancer of human lungs after smoking cigarettes (World Health Organization, 1988). Cohort studies of the irradiation of miners due to Radon have for a long time not given firm confidence in the possibility of applying the results of such studies to urban populations when Radon volumetric activity is the characteristic one for residential buildings with the purpose of assessing the risk of morbidity from lung cancer induced by Radon. (The risk..., 2013). To perform work with the aim of determining the respective risks when Radon concentration in housing, is relatively small the most appropriate method was the epidemiological study "case-control" (Lebed, Voloshkina et al., 2019). In the time frame from the 90s of the twentieth century to date, there have been approximately 40 case-control epidemiic studies of the impact of residential Radon on morbidity from lung cancer (Handbook on Indoor..., 2009) in the world. Between them there have been more than twenty with large volume of the main group (at least 200 people). The most

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comprehensive review of epidemiological studies of the risk of developing lung cancer during exposure to Radon at home is presented in the UNSCEAR Report (Effects of..., 2009) and 115 ICRP Publications (The risk..., 2013). With Radon volume activity in homes, as a rule, much lower than in mines, it should be expected that the additional risk of morbidity from lung cancer in urban populations is also much lower than in miners. Therefore, to identify the risk associated with Radon against the background of influence of other factors and to take into account the stochastic nature of carcinogenesis, very large groups (tens of thousands of individuals) are needed for the study. In practice, it is impossible to form and investigate such large groups of lung cancer patients within the framework of an epidemiological study of one territory. Therefore, none of the "case-control" studies performed had sufficient statistical power to draw conclusions about the presence and magnitude of the effect due to the influence of Radon on the population. The results of individual epidemiological studies, each of which does not have sufficient statistical power, can be used for generalized analysis. There are two types of analysis: meta-analysis and combined primary data analysis. Over the past 20 years, about 10 statistically significant meta-analyses have been conducted.

For example, in the meta-analysis (Yarmoshenko et al., 2005) the total volume of the main group was 12,044 whereas the volume of the control group was 20,932 people. In the meta-analysis, it is possible to summarize the results of several epidemiological studies quite quickly, but this approach has several shortcomings due to the lack of coincidence of different factors being taken into account by different researchers, which affects the accuracy of the relative risk determination. In addition, the meta-analysis uses heterogeneous results from studies conducted with the use of different methodological approaches. Such shortcomings can be avoided through relying on the combined analysis of primary epidemiological data, which integrates the original personal data of individuals and establishes a single standard methodology for statistical analysis. In the mid-2000s, three analyses were conducted that examined the combined data of 13 European (Darby et al., 2006), 7 North American (Krewski et al., 2006), and 2 Chinese (Lubin et al., 2004) case-control studies. With the use of the results obtained, physically reliable models of the risk of morbidity from lung cancer due to Radon were developed. Environmental studies of Radon concentration in the premises of Rivne (Lebed, Klymenko et al., 2018; Lebed, Pryschepa et al., 2018), in water (Lebed, Myslinchuk et al., 2018), in the ground gas (Lebed, Lysytsya et al., 2018) and the Radon influence on Rivne residents' morbidity from lung cancer (Lebed, Pryschepa et al., 2018; Lebed, Trustheva et al., 2019) were conducted by us in 2012-2019. The average annual Radon exposure in the air of 600 studied buildings was 1.392 WLM/year. Based on the results obtained, we calculated the additional relative risk of Rivne residents' morbidity from lung cancer using proportional constant risk models, Jacobi (GSF) and BEIR-VI in two variants: TSE/AGE/WL (exposure-age-concentration model, EAC model) and TSE/AGE/DUR (exposure-age-duration, EAD model). Some of these models are already outdated (proportional constant risk and Jacobi models), while others are actively used. It should be noted that for the calculation of radiation risks in the case of irradiating the population on the part of Radon, currently the most reliable with respect to coincidence of morbidity forecasts with the results of epidemiological studies are models based on data obtained for uranium miners:

1. BEIR-VI model based on epidemiological data from 11 cohorts of miners (National Research Council, 1999);
2. WISMUT model based on epidemiological data of the cohort of miners of German Uranium Mines (Grosche et al., 2006; Walsh et al., 2010) (modification of the BEIR VI model using original epidemiological data);
3. A model that is based on the analysis of cohorts of miners of France and the Czech Republic and takes into account the modifying effects of age at the time of irradiation and the time elapsed since the moment of irradiation (Tomasek et al., 2008);
4. WISMUT model based on epidemiological data of the cohort of miners of German Uranium Mines (Grosche et al., 2006; Walsh et al., 2011) (modification of the model (Tomasek et al., 2008) conducted with the use of original epidemiological data);
5. Tomasek model that analyzes research data from 11 miners' cohorts (Tomasek, 2014) (modification of the model (Tomasek et al., 2008) conducted with the use of another set of epidemiological data);
6. Models of coming into being on the part of cancer during a two-stage mutation that are based on the epidemiology of the French-Czech (Brugmans et al., 2004) and German (Van Dillen, 2011) cohorts;
7. Model that is similar to the BEIR VI model and based on data on morbidity and mortality from lung cancer in a cohort of miners who work in uranium mines of Eldorado (Lane et al., 2010);
9. Radon-2014 model developed in the Russian Federation (Kiselev et al., 2016) (modification of BEIR VI model conducted with the use of coefficients in the form of continuous functions).

The purpose of our work consists in calculating the values of the additional relative risk of Rivne residents' morbidity from lung cancer in the frameworks of WISMUT and Radon-2014 models, not yet used for Rivne residents, and comparing them with previously obtained BEIR-VI results.

**Materials and Methods**

**Model BEIR-VI**

In 1999, the National Academy of Sciences of the United States proposed the BEIR-VI risk model upon investigating 11 cohorts of miners with a total volume equal to 60,606 individuals. The model (National Research Council, 1999) takes into account a large number of factors that influence the processes of coming into being on the part of radiation-induced lung cancer. The following are the main differences between this model and the proportional risk and Jacobi models. Firstly, the BEIR-VI model predicts a decrease in the relative risk per unit of exposure with an increase in the power of the equivalent dose the lung tissue receives, that is, an increase in the EEEVA of Radon in the irradiation process (inverse dependence on the dose power). Secondly, this model uses different values of additional relative risk for smokers and non-smokers that correspond to the sub-multiplicative interaction between smoking and irradiating on the part of Radon DPD.

This model has two versions:

1. - relative risk depends on the elapsed time since exposure, the age at the moment of exposure, and the exposure duration (TSE/AGE/DUR model, exposure-age-duration model, EAD model);
2. - the relative risk depends on the time elapsed since the exposure, the age at the moment of exposure, and the level of Radon EEEVA in the irradiation process (TSE/AGE/WL model, exposure-age-concentration model, EAC model).

The additional relative risk in the model BEIR-VI is calculated according to the equation (1)

$$ R = \sum_{t=0}^{\infty} p_0(t) \int K_{ERR}(t) \exp \left[ - \sum_{t'=0}^{t} \int k_{let} \lambda_{let}(t') K_{ERR}(t') \right] $$

(2)

where $p_0(t)$ – the likelihood of surviving to a certain age $t$, $\lambda_{let}(t)$ – frequency of spontaneous coming into being on the part of
lung cancer, and $K_{ERR}(t) - additional relative risk factor calculated in the framework of the selected model of risk of Radon lung cancer, $k_{let} - mortality coefficient ($k_{let} = 0.95$).

The general form of a function $K_{ERR}(t)$ in these variants of models is offered in the form:

$$K_{ERR}(t) = \beta \cdot (P_{5-14} + \theta_{5-24} \cdot P_{15-24} + \theta_{25+} \cdot P_{25+}) \cdot \varphi(t) \cdot \gamma(z)$$

(3)

where $\beta$ – the main dose-effect parameter (in three variants - for smokers, non-smokers, and general population), $P_{5-14}, P_{15-24}, P_{25+}$ – exposure windows that determine the cumulative exposure of Radon and its DPD obtained in the time frames from (5 - 14) years to time t, from (15 - 24) years to time t, and from 25 years or more to time t for which risk assessment is conducted; $\theta_{5-24}, \theta_{25+}$ – coefficients that determine the relative contribution to the risk of coming into being on the part of lung cancer due to exposures obtained at the specified time intervals to age t; parameter $\varphi(t)$ – determines the dependence of carcinogenic susceptibility of lung tissue on the achieved age t; parameter $\gamma(z)$ – the function that depends on the duration of irradiation z (in years) or the Radon DPD volumetric activity level expressed in units of the working level WL (1 WL corresponds to an Radon EE equal to 3,700 Bq/m³).

The proposed models also incorporate the presence of a five-year latency period in the development of lung cancer. Therefore, the exposure obtained over the last 5 years prior to the age t is not taken into account in the expression (3). As some sort of a flaw of this model, one can view taking into account the fixed proportion of smokers, regardless of country (58% - males and 42% - females).

Model WISMUT. The model was developed with the use of results of epidemiological studies conducted in the time frame from 1946 to 1990 in a cohort of 59,001 males, who were uranium miners of the Wismut Company in the German Democratic Republic (Grosche et al., 2006). Among the miners, 2,388 deaths due to lung cancer were identified. The peculiarity of these epidemiological studies consists in the fact that the total number of people in the cohort is approximately equal to the one in the 11 cohorts of epidemiological studies that were used and formed the basis of the BEIR-VI model. The WISMUT model uses the equation for $K_{ERR}$, similar to equation (3) of the BEIR-VI model, but with different values of parameters $\beta, \theta, \varphi, \gamma$. In addition, all the miners of the German cohort were from the same geographical region and with the same social conditions of life. The studies, including the Radon DPD exposure assessment, were performed with the use of the identical procedures. These circumstances add great confidence to the reliability of the developed WISMUT model. Contrary to the model BEIR-VI, the WISMUT model presents only one variant of the coefficient $\beta$ and the modifying factor $\gamma(z)$ as a function of dose power. In addition, the parameter $\theta_0$ in the WISMUT model has the highest value in the time interval $\Delta t = 15 - 25$ years, contrary to the BEIR-VI model, in which this parameter is maximal in the initial interval $\Delta t = 5 - 15$ years. In this case, there is a “trimming” of the parameter value from the maximum value to zero at $\Delta t \leq 5$ years. It is difficult to perceive from the standpoint of medical and biological ideas about the development of malignant formations. It should also be taken into account that the WISMUT model was developed a few years later than the BEIR-VI model. This made it possible to make use of past experience and later publications.

Combined model Radon-2014. The use of data of epidemiological studies performed among miners to estimate the radiation risk of exposure to Radon and its DPD in buildings has some limitations. Basically, these restrictions are caused by gender differences between the miners' cohorts and the population of the urban ecosystem and the differences in the chemical composition of the air in the mines and in the homes (aerosol concentration, air exchange rate, presence of other impurities, etc.). In connection with the data obtained from a joint analysis of epidemiological studies "case-control" of the association of lung cancer with irradiation by Radon, an interest in developing a combined model that would take into account both the temporal and age characteristics of the radiation risk obtained as a result of investigations among miners and the population of urban ecosystems emerged. The development of such a model, called "Radon-2011" and later "Radon-2013" and "Radon-2014" was proposed in (Demin et al., 2011; Zhukovsky et al., 2014; Demin et al., 2015). The model Radon-2014 is based on a formula for the dependence "dose-effect" that determines the relationship between the effect of Radon influence and the one-time (short-term) exposure $P$:

$$\lambda_r(s, e, t, P) = \lambda_0(s, t) \cdot K_{ERR}(e, t, P),$$

(4)

where $\lambda_r(s, e, t, P)$ – the incidence of coming into being on the part of radiation-induced lung cancer, $\lambda_0(s, t)$ – the coefficient of base age-specific morbidity, $K_{ERR}(e, t, P)$ – the coefficient of additional relative risk, $s$ – the parameter that determines gender, $e$ – time of exposure, $t$ – the time of detecting the effect of irradiation with the dose $P$.

Here

$$K_{ERR}(e, t, P) = \alpha(e, t) \cdot P,$$

(5)

The value of the function $\alpha(e, t)$ is determined by three parameters, as in the models BEIR-VI and WISMUT:

$$\alpha(e, t) = \beta \cdot \theta(t-e) \cdot \varphi(t).$$

(6)

The authors propose to assume that the risk ratios $K_{ERR}$ are identical for males and females in the framework of the model Radon-2014. For long exposure duration, the dependence of $K_{ERR}$ on $P$ becomes nonlinear, and the model proposes to determine $K_{ERR}$ according to the formula:
\[ K_{ERR}(e, t, P) = e^{-\beta \varphi(t) \int e^{\theta(t-e')} P(e') de'} - 1. \] (7)

For relatively small levels of influence, when the value of the function \( K_{ERR} \) becomes much smaller than unity, one can decompose the exponential function in formula (7) into the zero and first terms and obtain an approximate expression for \( K_{ERR} \):

\[ K_{ERR}(e, t, P) \approx \beta \cdot \varphi(t) \cdot e^{\theta(t-e')} \cdot P(e') de'. \] (8)

The model BEIR-VI actually uses this approximation. However, with relatively high exposures and (or) accumulated doses, this approximation can result in a significant error. Contrary to the model BEIR-VI, where the parameters \( \varphi(x) \) and \( \theta(x) \) are tabulated, the model Radon-2014 proposes to use the smoothing function for them

\[ F(x) = \frac{A}{1 + B \cdot e^{C x}} + D \] (9)

where the parameters \( A, B, C, \) and \( D \) are determined from (Kiselev et al., 2016, table 3.7). The smoothed dependences \( \varphi(x) \) and \( \theta(x) \) are depicted in Figure 1.

From figure 1b, it follows that parameter \( \theta \) does not have a minimum latency period (MLP) of 5 years on which the parameter equals 0, as in the model BEIR-VI. In the neighbourhood of point \( t = 5 \), the parameter \( \theta \) smoothly and rapidly decreases to zero.

To ensure that the proposed model is fully consistent with the results of the epidemiological studies, a normalization procedure was performed so that the additional relative risk of coming into being on the part of lung cancer, calculated in the framework of the model Radon-2014, was consistent with the data reported in (Darby et al., 2006): 16% per 100 Bq/m3 of volume activity of 222Rn in a residential premises. To achieve being in agreement with other models on the part of this model in it, it is proposed to use the coefficient \( \beta = 0.01/WLM \) with a reliability equal to 0.95 and a confidence interval (0.005–0.015). We calculated the risks based on the experimental data obtained using models BEIR-VI in two versions, WISMUT, and Radon-2014. Table 1 presents the parameters of the models we selected. We used the value of the parameter \( \beta \) for the general population.

### Table 1. Parameter values of selected models.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>TSE/AGE/DUR</th>
<th>TSE/AGE/WL</th>
<th>WISMUT</th>
<th>Radon-2014</th>
</tr>
</thead>
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<tr>
<td>( \beta = 0.01/WLM )</td>
<td>0.0055</td>
<td>0.0768</td>
<td>0.0135</td>
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<td>( \theta_{5-14} )</td>
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<td>1.0</td>
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<td>( \theta_{15-24} )</td>
<td>0.72</td>
<td>0.78</td>
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<td>( \theta_{25+} )</td>
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<td>0.51</td>
<td>0.56</td>
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<tr>
<td>( \varphi_{5-14} )</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.080</td>
</tr>
<tr>
<td>( \varphi_{15-19} )</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.072</td>
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Results and Discussion

The results of the calculations of the additional relative risk coefficient $K_{ERR}$ and the additional relative risk $R$ that depend on the irradiation time and the model selected are presented in Table 2 and Figures 2-3. The results obtained and analyzed by us show that the total number of lung cancer deaths associated with exposure to Radon in the premises of the city of Rivne in 2011-2015, averaged over Radon exposure by age and geographical location of the building depends on the choice of the risk model and ranges from 5 to 128 for males and from 1 to 23 for females. It accounts for 3 to 64% of all deaths from lung cancer that occur during one year time frame during the study period for males and 3 to 77% for females. The smallest number of deaths due to Radon is obtained in the framework of the model Radon-2014 whereas the highest one is obtained in the framework of the model TSE/AGE/WL. It should be noted that the assumption that the exposure level equal to 1.392 WLM/year is not too high is widespread. Similar calculations in the US showed that 21,800 and 15,400 lung cancer deaths were probably caused by Radon indoors each year if the models TSE/AGE/WL and TSE/AGE/DUR were respectively used to calculate the risks. For France, this number equals 3,337 in the case of the model TSE/AGE/WL and 2,361 in the case of the model TSE/AGE/DUR. The comparison shows that, taking into account the ratio of the mortality rate to the population of the study region one can conclude that the "Radon problem" in Rivne (Ukraine) is much more acute than in France, as evidenced by our previous comparisons with similar studies in Corsica (Lebed, Klymenko et al., 2018).

Table 2. The dependences of the coefficient of the additional relative risk $K_{ERR}$, and $R$ on irradiation time and the choice of the model.

<table>
<thead>
<tr>
<th>t, years</th>
<th>TSE/AGE/DUR</th>
<th>TSE/AGE/WL</th>
<th>Model</th>
<th>WISMUT</th>
<th>Radon-2014</th>
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<tr>
<td></td>
<td>$K_{ERR}$</td>
<td>$R$</td>
<td>$K_{ERR}$</td>
<td>$R$</td>
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<tr>
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<td>0.631</td>
</tr>
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<td>0.048</td>
<td>2.017</td>
<td>0.075</td>
<td>0.631</td>
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Table 1. Probability of Radon cancer occurrence in Rivne city

<table>
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<tr>
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Figure 2. Dependence of the additional relative risk coefficient $K_{ERR}$ on time with constant Radon exposure in dwelling equal to 1,392 WLM/year for models: 1. TSE/AGE/DUR; 2. TSE/AGE/WL; 3. WISMUT; 4. Radon-2014.

Figure 3. Dependence of the additional relative risk R on time with constant radon exposure in dwelling equal to 1,392 WLM/year for models: 1. TSE/AGE/DUR; 2. TSE/AGE/WL; 3. WISMUT; 4. Radon-2014.

Conclusion

This research is the first estimate of the risk of morbidity and mortality from tracheal, bronchial, and lung cancer associated with Radon exposure in Rivne obtained in the frameworks of the models WISMUT and Radon 2014. Such research should be extended to other regions of Ukraine as the Radon problem is a matter of serious concern to the European Community and the World Health Organization. The estimated number of deaths of Rivne residents from lung cancer associated with Radon exposure in the city is uncertain due to uncertainty in the dependence "dose-effect" and ranges from 5 to 128 people per year for males and from 1 to 23 persons per year for females. It accounts for 3 to 64% of all deaths from lung cancer that occur during one year time frame during the study period for males and 3 to 77% for females. Such the number of deaths greatly exceeds the figures obtained from similar calculations of mortality in France (Catelinois et al., 2006). This may be due to the difficult Radon situation in the Rivne region, a much larger measurement campaign in France, which was funded by the EU (our research was carried out at our own expense with enthusiasm) and different instrument base with a larger measurement error.
References


of Health and Human Services.


Citation:

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