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Ecological Risk Assessment on the Territory of Belarus After Chernobyl Accident

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1. Introduction

Radiation contamination of several regions of Belarus was analyzed. As a part of analysis, statistical data on cancer cases was compared to the predicted data. Ecological situation due to chemical impact of industrial facilities' atmospheric releases was studied. For a biological model, an experimental study was carried out to investigate combined impact of radiation and chemical factors.

Complex assessment of radiation and chemical state was performed for 6 districts of Gomel region and 4 districts of Mohilev region. As the result of the assessment, model values for radiation and chemical risks were obtained.

In the relocation zone of Chernobyl Nuclear Power Plant (NPP), biological consequences of ecological factors (including radiation) combined impact were studied in a series of experiments. Laboratory mice of Af line (line of mice with high spontaneous frequency of lung tumors) were exposed in the relocation zone. The influence of exposure and natural background radiation level on the spontaneous and chemically induced process of lungs tumor development process was studied.

Linear risk assessment methodology proposed by the International Commission on Radiological Protection (ICRP) was used to analyze radiation risk. The US Environmental Protection Agency (EPA) methodology (with adjustments to local conditions) was used to analyze chemical risks.

2. Radiation risk assessment for Belarus regions which were most affected by Chernobyl accident

To assess health detriment of population of these regions, value of radiation induced injuries (probabilities of stochastic effects, which newly formed malignancies being the most dangerous) was used. Data on radiation risk assessment, as calculated according to linear non–threshold "Dose–effect" model, is presented in Table 1 for the Belarus territories most contaminated with radionuclide. In assessment, a lifelong risk coefficient for radiation

induced cancers was used (5%/Sv, as suggested by *ICRP Recommendations*, 1990). The values of doses used for risk assessment were taken from *Compendium of Exposition Doses of Belarus Population* (1992). Therefore, the assessments presented determine possible cancers due to exposure at that period of time, and do not consider thyroid cancers induced by radiation and leukemia cases among the liquidators of the Chernobyl accident.

Location		**AEED*, mSv**	R, ×10^{-5}	N	n_p	Σn_p
Brahin	City	2.5	12.5	5635	0.704	2.1
	District	1.5	7.5	18083	1.356	
Vetka	City	3.1	15.5	9714	1.506	3.3
	District	2.1	10.5	16904	1.775	
Korma	City	2.0	10.0	6424	0.642	1.9
	District	1.7	8.5	15140	1.287	
Narovlya	City	2.3	11.5	11166	1.284	2.7
	District	3.4	17.0	8294	1.410	
Hoyniki	City	0.9	4.5	15813	0.712	2.5
	District	2.1	10.5	17015	1.787	
Chechersk	City	2.9	14.5	8915	1.293	2.8
	District	2.1	10.5	13986	1.469	

* – here, AEED is Annual Effective Equivalent Dose, R – risk, N – population, n_p – calculated values of found newly formed malignancies for a particular location, Σn_p – sum of n_p

Table 1. Radiattion risk assessment for Gomel region.

3. Carcinogenic risk analysis of environmental chemical contamination impact for Belarus regions which were most affected by Chernobyl accident

Quantitative connection between probability of sickness and dose of an individual is exposed is determined, when assessing risk from carcinogens. The value of risk may be presented in one of the following ways: as normalized risk per unit dose, as concentration risk corresponding to the given risk level, as individual risk, and as risk for a group of people. Knowing the values of individual risk allows predicting risk of cancer development at particular exposure values. Values of carcinogenic risks for the substances proven to be carcinogens at the locations in study are presented in Table 2. Calculated values of carcinogenic risk due to chemical contamination of the environment are presented in Tables 3 – 4 for two districts of Belarus.

Chemical compound	**Upper limit for risk**
Benzopyrene	7.3×10^{-12}
Bensol	7×10^{-6}
Formaldehyde	5×10^{-5}
Manganese	4×10^{-4}
Chromium	1.2×10^{-2}

Table 2. Additional lifelong cancer risk for a person weighting 70kg due to inhaling 1μg/m^3 of a chemical compound during 70 years of life.

Region	Substance	C^*	R, $\times 10^{-5}$	n per 10^5 people	N	n_p
Brahin	Manganese	0.32	1.82	1.8	23718	0.4
Vetka	Manganese	0.24	1.37	1.4	26618	0.4
Korma	Manganese	0.5	2.85	2.9	21564	0.6
Narovlya	Manganese Formaldehyde	0.05 0.36	0.29 0.08	0.3 0.1	19460	0.1
Hoyniki	Manganese Chromium	0.05 0.27	0.29 6.87	0.3 6.9	32828	2.4
Chechersk	Manganese	0.09	0.51	0.5	22901	0.1

* – concentration as fraction of Maximum Permissible Concentration (MPC)

Table 3. Calculated values of carcinogenic risk due to chemical contamination of the environment (Gomel district).

Predicted carcinogenic risk (both radiation and chemical) values were compares to eh statistical data on cancer sickness. In our work it is shown, that combined radiation and chemical contamination cannot be the source of detectable (above the background) carcinogenic risk. Impartial assessment of ecological risk can be developed only with the help of combined multifactor theoretical and experimental modeling.

Region	Substance	C^*	R	n per 10^5 people	N	n_p
Bykhov	Benzopyrene	0.0015	1.56×10^{-13}	≈0	53383	4.4
	Bensol	0.003	4.5×10^{-6}	0.45		
	Chromium	0.3	7.71×10^{-5}	7.71		
	Formaldehyde	0.2	4.29×10^{-7}	0.043		
Krasnopol	Manganese	0.22	1.26×10^{-5}	1.26	14720	0.4
	Chromium	0.05	1.29×10^{-5}	1.29		
Slavgorod	Benzopyrene	0.07	7.3×10^{-12}	≈0	19511	0.7
	Manganese	0.22	1.26×10^{-5}	1.26		
	Chromium	0.1	2.57×10^{-5}	2.57		
Chaussy	Benzopyrene	0.083	8.66×10^{-12}	≈0	22716	0.2
	Manganese	0.15	8.57×10^{-6}	0.86		

* – concentration as fraction of Maximum Permissible Concentration (MPC)

Table 4. Calculated values of carcinogenic risk due to chemical contamination of the environment (Mogilev district).

It is obvious that the obtained estimates of additional sicknesses number (Tables 3 – 4) are maximal, as required by the general "ideology" of risk assessment.

4. Study of tumor formation in animals and their progeny when exposed to combined influence of ecological factors of different nature

Radiation effects may result not only from action of artificial radio nuclides, but from naturally occurring radioactive isotopes as well. Radioactive uranium isotopes, that are

widely distributed, are accumulated in elevated amounts in organs of people, which live in goiter endemic regions of Belarus. Significant amount of enriched uranium from the wrecked unit of Chernobyl NPP got into the environment. So far, the issue of possible contribution of uranium and other naturally occurring radio nuclides to progeny inheritance of genetic system damages induced by radiation particularly or in the process of evolution have not been considered. However, this aspect of the issue has both a fundamental importance in analysis of natural radioactivity role and especial relevance for population of regions suffered from radiation damage, because total increased background radiation level has become a global ecological factor.

Discussed further, is a series of studies aimed on investigation of biological effects of joint influences of ecological factors.

Belarus population, which suffered from Chernobyl NPP accident and is now living on radiation–contaminated territory, form a group exposed to an increased risk of developing stochastic and genetic effects related to exposure (Spiridonov et al., 2007, Lorimore et al., 2005). One of the fundamental aspects of this problem is still to estimate stability of cells' genetic apparatus functioning, because damages to genome may become basis to immune system impairment, carcinogenesis, and decrease in life expectancy (Suskov et al., 2006).

Biological effects of joint influences of ecological factors, including radiation, can be most profoundly studied in the Chernobyl NPP relocation zone. Chernobyl NPP relocation zone is a unique place, where radiation is the dominating ecological factor, which is caused by the environment contamination level. The Polessky Radiation–Ecology Reserve (PRER) was created on this territory in 1988. Various radiobiological studies are carried out in PRER (Konoplya et al., 2011).

Series of studies were carried out in PRER to evaluate influence of:

1. Exposure on laboratory animals; and
2. Natural radioactivity on spontaneous and chemically induced tumor process in lungs (adenomas) of mice of Af line and their progeny (first generation – F1).

Usage of a biological model "lungs adenomas" as an accelerated test for Af-line mice to determine carcinogenicity of chemical substances is based on responsiveness in adenomas development under the action of various carcinogenic agents (Turusov and Parfenov, 1986). Mice used in the experiment are of "highly carcinogenic" Af-line, characterized by high sensitivity of lung tissue to blastomogenic effect of urethane and genetic predisposition to formation of tumors in lungs. Previously, urethane was widely used in pediatrics as a sedative measure. However, it was experimentally established that urethane causes a wide spectrum of embryonic disorders and tumors of different localization, independent of the way it was injected into body (Porubova, 2000).

Influence of exposure duration (1 and 4 months) in the Chernobyl NPP relocation zone on spontaneous and chemically induced mutagenesis and tumor formation in lungs of mice (Af line) and their progeny (first one – F1) was studied. The procedure of experiments was the following: male and female laboratory mice (3 month old) were delivered to the relocation zone in May 2007. Dose rate of 3.29±0.10 μGy/hr was detected at the ground level where the mice were placed. After one month mice were split into the following groups: one part was relocated from the contaminated area to read background indicators, while the other part

was allowed to copulate. Later pregnant females were relocated to vivarium in Minsk to get progeny F_1. In 24 hours after being relocated from the contaminated area, the animals were injected with urethane (1 mg/g, in the form of 10% sterilized solution with crystalline urethane – ethyl-carbonate, ($C_3H_7NO_2$) – Sigma - Aldrich). Animals were removed from the experiment after 72 hours after relocation from the contaminated area. 3–month old progeny F1 from parents, which were in the Chernobyl NPP relocation zone for 1 month, were injected with urethane (1 mg/g). Control was performed with the help of progeny F1 of the same age from Minsk vivarium. The same procedure was followed for laboratory animals after 4–month exposure in Chernobyl NPP relocation zone (PRER).

For parents and progeny, tumor process was assessed according to the number adenomas/mouse after 20 weeks of urethane injection. Influence of mice's uranium intoxication on sensitivity of the progeny to combined impact of exposure and urethane was studied by testing lungs adenomas. In this study, 3 weeks old immature male and female mice of Af line who were drinking solution of nitro–acid uranium ($UO_2(NO_3)_{2*}6\ H_2O$) in concentrations 180 mg/liter during the next 3.5 months, were used. (Novikov, 1974). After 3.5 months from the start of drinking, a part of animals, as well as control, were relocated for 7 – 10 days for copulation. Urethane (1 mg/g of body mass) was injected peritoneally to a part of immature animals 3.5 months after uranium intoxication as well as to intacted animals of the same age; adenomas were recorder after 20 weeks. Progeny of the intacted animals and 8– to 10–weeks old mice after uranium intoxication were exposed to 0.35 Gy gamma–rays (1 Gy/hr dose rate, ^{60}Co source), injected with urethane (1 mg/g), and exposed to combined radiation of the same dose and urethane concentration. Control was injected with the same amount of physiological solution. Tumor process was estimated by tumor rate (% of mice with adenomas) and adenomas/mouse number after 20 weeks according to the process described by Turusov and Parfenov (1986).

Experimental data were processed statistically using Student's criteria. An interaction coefficient K_w was used to describe quantitatively results of joint influence of factors of different origins. Interaction coefficient K_w was determined as a ratio of a system response to the combined radiation and toxic effect to the total effect of system responses to independent effects of individual factors (Kuzin, 1983). Effects were defined as and excess of level, induced by stressors, over spontaneous value. The result of interaction was considered to be additive if $K_w = 1$, and as antagonistic (synergetic) if K_w was reliably less (greater) than 1 (Geraskin et al., 1996).

The results of study of induced tumor process in lungs of mice (adenomas) placed in the relocation zone and in mice injected with urethane are presented in Table 5.

Analysis of results of influence of mice exposure in PRER zone on lung tumor process showed, that radio-ecological factors of Chernobyl NPP relocation zone increase tumors occurrence insignificantly (1.2 times) due to 1 months exposure, and increase tumors occurrence statistically significantly (more than 2.9 times) due to 4 months containment of animals in the zone. Also, intoxication of mice with urethane increases the number of adenomas in lungs statistically significantly for all the time period considered in the research.

Also, urethane injection to mice kept in Chernobyl NPP relocation zone for 1 and 4 months increases average adenomas/mouse number statistically significantly as compared to

control. Interaction coefficient K_w was found to be 2.4 and 1.1 for mice exposed in the relocation zone during 1 and 4 months respectively. This signifies that joint effect of relocation zone factors and carcinogen (urethane) is synergetic, which decreases with exposure period in zone along with the increase in radiation contribution from 4.8 to 9.6%.

Exposure period	**1 month**			**4 months**		
Impact	**Mice number (male+ female)**	**Group–average number of adenomas / mouse**	K_w	**Mice number (male+ female)**	**Group–average number of adenomas / mouse**	K_w
Control (background level)	39	0.31 ± 0.09	–	27	0.26 ± 0.06	–
Urethane (Minsk vivarium)	26	7.03 ± 1.51*	–	27	3.36 ± 0.43*	–
Chernobyl NPP relocation zone	24	0.37 ± 0.02	–	22	0.77 ± 0.17*	–
Chernobyl NPP relocation zone + urethane	24	7.67 ± 2.08*8	2.4	21	8.05 ± 1.00*	1.1

* – Statistically significant difference from control-group, $p < 0.05$; ** – statistically significant difference from urethane control-group, $p < 0.05$.

Table 5. Number of induced adenomas/mouse in animals exposed in Chernobyl NPP relocation zone during 1 and 4 months.

Genome damages caused by the environment of Chernobyl NPP relocation zone are inherited by progeny. Originally, research of consequences of ionizing radiation influence for mammals was developing as studies of radiation effects for progeny of one exposed and one un-exposed parents. Thus, progeny was formed and developed from one exposed and one intact sex cell. However, since ionizing radiation became a global ecological factor, studies of radiation effects for progeny of parents who had been both exposed have become relevant. Studies of this problem have both fundamental and practical importance, because it has been neither unambiguously accepted, nor scientifically proven as to what dose to use to calculate genetic risk for progeny of both exposed parents (Nefedov et al., 2003).

Results of spontaneously and chemically induced carcinogenesis in lungs of F1 progeny of Af-line mice, both intact and exposed in PRER zone during 1 – 4 months, are presented in Table 6.

Evaluation of late effects showed that spontaneous occurrence of lungs adenomas had risen significantly (more than 5 times) for F1-mice progeny, that had been contained in Chernobyl NPP relocation zone during 1 month, as compared to the number of adenomas in lungs of progeny of intact mice.

Urethane injection to progeny of mice exposed in Chernobyl NPP relocation zone statistically significantly increases number of induced adenomas/mouse as compared to control-group (intact progeny of mice intoxicated with urethane).

Groups	Exposure period of mice–parents					
	1 months			4 months		
	Number of mice. *n*	Group average number of adenomas/mouse	K_w	Number of mice. *n*	Group average number of adenomas/mouse	K_w
F1 progeny (vivarium)	23	0.33 ± 0.07	–	34	0.23 ± 0.11	–
F1 progeny (vivarium + urethane)	25	6.10 ± 0.81*	–	26	6.38 ± 0.68*	–
F1 progeny of parents from Chernobyl NPP relocation zone	20	1.75 ± 0.61*	–	14	1.14 ± 0.36*	–
F1 progeny of parents from Chernobyl NPP relocation zone + urethane	14	10.28 ± 1.40*	1.38	14	8.85 ± 1.08*	1.22

* - Statistically significant difference from control-group, $p \leq 0.05$.

Table 6. Number of induced adenomas/mouse in F1 mice progeny that were exposed in Chernobyl NPP relocation zone during 1 and 4 months and/or were injected with urethane.

For F1-mice progeny which were exposed during 4 months in Chernobyl NPP relocation zone, the number of spontaneous tumors exceeded that for intact F1-mice progeny more than 4.9 times. Injection of urethane to F1-mice progeny, that had been previously exposed in Chernobyl NPP during 4 months, increased number of induced tumors by 7.76 times as compared to that of progeny of mice from Chernobyl NPP relocation zone (K_w = 1.22). It is important to notice an increased sensitivity of progeny of mice that had been exposed in Chernobyl NPP relocation zone, compared to their parents. This was pronounced in higher values of interaction coefficient K_w, namely, 1.38 – 1.22 with simultaneous decrease of radiation contribution from 17 to 12%.

A more pronounced difference in the level of induced tumor process occurred in the group of originally immature mice 6 months after stopping drinking uranium solution (adenomas/mouse number exceeded that of control-group more than 1.6 times). This signifies importance of age as a factor of animals' responsiveness to radiological and toxicological action.

Differences in responsiveness between intact animals and mice, which had been intoxicated with uranium, was also revealed while studying occurrences of tumors among animals after separate and joint uranium and urethane action, see Fig. 1.

Adenomas/mouse number increased statistically significantly after urethane action as compared to control-group ($p < 0.05$). Maximal value of tumor effect was observed among animals, which had been subjected to joint uranium and urethane action. This value exceeded level of urethane carcinogenesis more than by 10%, and reflects effect of combining influencing factors.

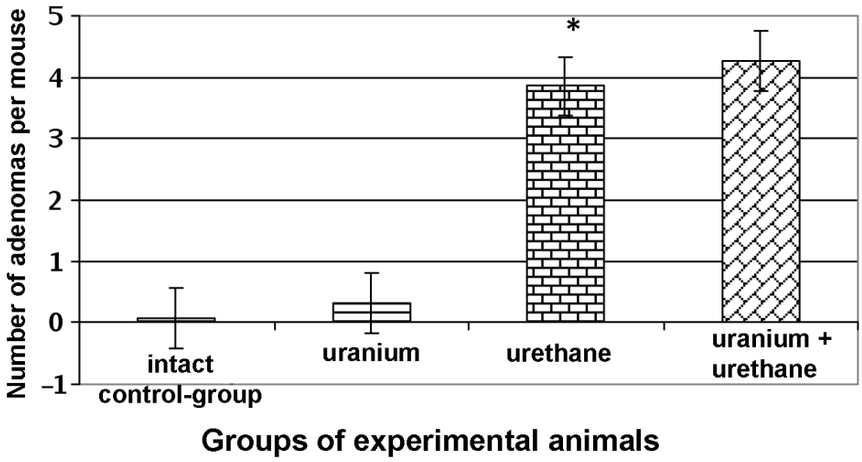
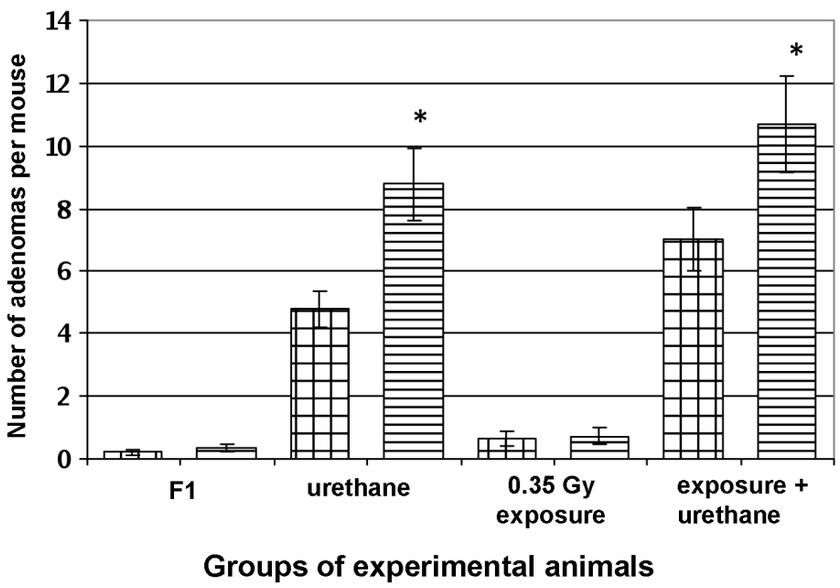


Fig. 1. Tumor formation among mice intoxicated with uranium (3.5 months) and being injected with urethane.

Apart from this, uranium intoxication as well as influence of low levels of various toxicants changes the responsiveness (sensitivity) of cells to the further action of different genotoxic factors. These changes manifest themselves in the form of a modifying effect, the exhibited level of the latter being determined by the level an organism is "familiar" with it.

Transgenetic transfer of sex cells; genome damages induced by uranium was also studied on Af-line mice. The results of studies are shown in Fig. 2.



- F1 progeny of intact parents

- F1 progeny of parents intoxicates with uranium

Fig. 2. Tumor formation and sensitivity of progeny (F1) of intact and intoxicated with uranium mice to exposure and chemical carcinogen.

Comparison of level in spontaneous tumor formations within progeny F1 of intact and intoxicated with uranium mice indicates a significant increase in adenomas/mouse number (more than by 63%) for progeny (F1) of mice that received nitric acid uranium (0.22 ± 0.09 and 0.36 ± 0.12 adenomas/mouse, respectively). Of a non-lesser importance is the difference in progeny sensitivity to the influence of additional agents. Urethane injection into progeny (F1) of uranium mice lead to statistically significant almost doubled (8.78 ± 1.17) increase in adenomas/mouse number that was observed within progeny of intact mice (4.77 ± 0.58). However, progeny responsiveness to exposure was somewhat different. In fact, the was almost no difference in number of induced adenomas/mouse between exposed progeny of uranium mice and that of exposed progeny of intact mice (0.72 and 0.66 adenomas/mouse) respectively. Subjecting to a joint effect of gamma-radiation and urethane group of progeny F1 of uranium mice, lead to the maximal (10.7 ± 1.52 adenomas/mouse) and statistically significantly exceeded the same indicator for a subjected to the joint effect of gamma-radiation and urethane group of progeny F1 of intact mice (7.05 ± 1.00 adenomas/mouse).

It follows from the obtained data, that:

1. Damages are induced in the genetic system of sex cells as a result of uranium intoxication;
2. These damages are transferred to progeny and manifest themselves as an increase in sensitivity of progeny to transformation under additional influence of genotoxic factors (i.e. genetic instability is induced).

Genetic instability may be one of the contributors to the formation mechanism of genetic burden.

4.1 Mathematical modeling of the consequences of the joint effect of the factors of radiation and non–radiation nature on the organism

Principles of the risk evaluation, used today for the population, residing on the territories, contaminated after the disaster at the Chernobyl NPP, are based mainly on the radiation influence analysis. However, results of the radiation risk assessment come into collision with the actually established changes in the quantity of the stochastic effects (officially published annual statistics), requiring an elaboration of new criteria of analysis that would be based on a multifactor influence.

In the framework of one of the problems of the present work, an attempt has been made to introduce a mathematical model of the synergic interaction of the factors of the radiation and non–radiation (on the example of a chemical carcinogen) nature and the damage manifestation as a result of such an interaction. A biological experiment has been carried out in the Radiation–Toxicological Ecology Laboratory of the Institute for Radio–Ecological Problems, National Academy of Sciences, Belarus (Malenchenko et al., 2001).

A hypothesis is proposed to create a mathematical model able to describe effects under a combined interaction, based on the assumption that a formation of additional effective damages takes place due to the interaction of subdamages, induced by each agent and not effective under a separate influence of each factors, both under combined and joint effect of any adverse environmental factors (Petin and Zhuravkovskaya, 1999).

Mathematical modeling of the consequences of the joint effect, based upon the above described method, turned out to be especially successful for a dose of 0.35 Gy owing to a monotonic character of the dependence "Number of the revealed damages" – "Time of the urethane introduction after the exposure to radiation". In accordance with the foregoing:

$$N_{\Sigma} = N_1 + PN_1, \tag{1}$$

where

N_{Σ} – total value of damages;
N_1 – amount of damages from the direct affect of the hazard factor (in our case – this is radiation);
P – share of "sub–damages", that are not revealed under a single radiation influence but reveal with addition of the second hazard factor (here – urethane).

Model (1) is stationary. If the sub–damages are not revealed under isolated influence of a single factor (radiation), then they will relax with time in the direction of the undamaged condition. We will consider this process as a purely probabilistic one with a constant relative rate (as in the case of the radioactive decay); i.e. the constant ratio of recoveries from the sub–damages rehabilitates per unit time. Then:

N_0 – amount of the damages from radiation;
PN_0 – amount of the sub–damages from radiation;
$PN_0 \cdot \exp(-t/\tau)$ – decreasing in the amount of sub–damages with time t and τ being "sub–damages lifetime".

Thus, we can find the amount of damages at any instant after irradiation under the urethane incorporation to be:

$$N(t) = N_0 + PN_0 \cdot \exp(-t/\tau), \tag{2}$$

where the second term of the sum is the amount of the sub–damages at the moment t, revealed as damages due to incorporation of the second hazardous factor (urethane).

On the basis of the experimental data, we are able to find average values of the parameters P and τ, corresponding to the experimental data with minimum error ε at the whole range of values:

$$P = 5.8 \pm 0.6\ (\varepsilon \approx 10\%); \qquad \tau = 16 \pm 3 \text{ days } (\varepsilon \approx 20\%)$$

Hence, as for the dose of 0.35 Gy we get the following dependence of damages on time of the urethane incorporation after irradiation:

$$N(t) = 1.3 + 7.4 \cdot \exp(-t/16) \tag{3}$$

The modeled values shown in Fig. 3 were obtained in accordance with Eq. (3) and quite well correspond to the experimental values. This indicates a good fit of the model (2) to the experimental data in case of a monotonic character of dependence of the amount of damages on time.

Difficulty in getting an estimate for the tumor genesis process appraisal under combined action of radiation and chemical carcinogen is not only limited by the time period between

interactions of carcinogens. Genesis of the radiation–induced tumor depends both on the dose and on its time distribution – the dose rate.

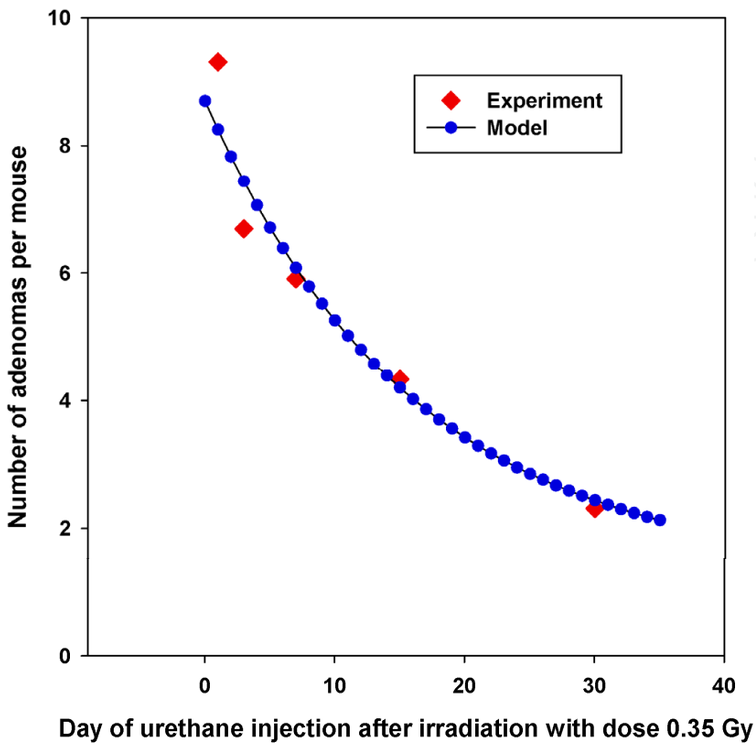


Fig. 3. Calculations according to model and experimental data.

Technical possibilities and latest experimental data allow revealing specific contribution of the radiation factor in the real ecological conditions and distinguishing single–factor risk due to radiation only. However, the attempt to extrapolate the findings for the current ecological conditions is connected with considerable difficulties that can mislead or show failure of the performed calculations. This was clearly pronounced in discrepancies between the predictions and the actual data on thyroid gland cancer occurrences among children in Belarus after the Chernobyl accident. In the view of this, we state that there is an obvious necessity to develop new methodological principles of the sanitary and ecological evaluation of the environmental quality under the conditions of joint effect of the ionizing radiation and factors of the non–radiation origin on human the organism.

5. Discussion

Currently, a special attention is paid to the issue of genome instability, which is caused by ionizing radiation. The main reason for this is that this issue is immediately related to understanding mechanisms of such important radiobiological phenomena as radiation mutagenesis, carcinogenesis, ageing, which are all remote consequences of ionizing radiation effects (Pelevina et al., 2003). Within concept of genome mutability, the phenomenon of genome instability induced by radiation is interpreted as a transition of progeny of exposed cells to the state of readiness to adaption changes. Such a state can have

two outcomes: 1) adaptation to new conditions with a gradual normalization of cell systems and their functions by repairing and eliminating defective cells; or 2) transition to a modified state by means of preserved instability of genome; this can be accompanied by an occurrence of a malignant phenotype (Mazurik and Mikhaylov, 2001).

Obtained experimental data within out studies indicate that a combination of ecological factors present in Chernobyl NPP relocation zone induced damages in the sex cells genome of parents and promotes transgeneration transfer of these damages to the progeny. Obviously, similar process are occurring in organisms of animals and people that leave in contaminated areas that are close to Chernobyl NPP relocation zone. That's why, an increase of child sicknesses, that has been detected in recent years, may be not only a consequence of the current ecological situation, but also be a result of their parents being subjected to harmful to health effects after Chernobyl accident and accompanying adverse socio-economical and ecological factors (including naturally occurring radiation). These data pronounce an increase in the rate of genetic burden formation in organisms, subjected to the elevated radiation background level and influence of toxic factors. Such conditions are characteristic of territories suffered as the result of Chernobyl accident. Therefore, an impartial assessment of an ecological risk can be elaborates only be methods of combined multifactor theoretical and experimental modeling.

6. Conclusions

1. The radiation contamination due to Chernobyl accident cannot be the source of detectable carcinogenic risk on the territory of Belarus.
2. Current ideas about carcinogenic danger due to chemical contamination of the environment cannot be used for satisfactory explanation of the observed stochastic effects. Impartial assessment of ecological risk can be developed only with the help of combined multifactor theoretical and experimental modeling.
3. Combined-impact coefficient of interaction shows the mutually enhanced pattern of carcinogenic factors impact. The studied ecological factors induce damages to genome of parents' reproductive cells and its transgeneration transfer to the progeny.
4. Strategic aim of ecological risk studies is to solve the general problem: *to establish specific features and outputs of negative consequences (e.g. cancers) for human exposed to the specific combinations of technogenic and natural contamination*.

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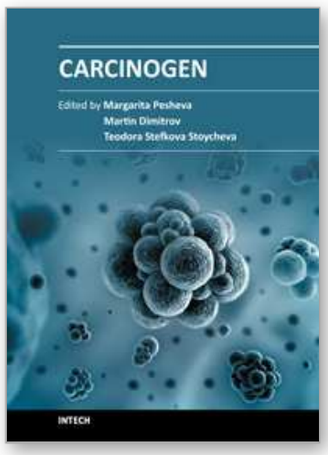

Carcinogen

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During the last decades, cancer diseases have increased all over the world. The low quality of food and strong pollution of environment are the main prerequisites for carcinogenesis. The main problem for scientists is to find strategy for prevention of cancer diseases. Therefore, the information about the models for studying carcinogenesis and mutagens which appear during cooking, environmental pollutants, and tests for specific detection of carcinogens is particularly important. The book "Carcinogen" is intended for biologists, researchers, students in medical sciences and professionals interested in associated areas.

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