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POST–ACCIDENT BIOTA OF SERIOUS AND MAJOR RADIATION ACCIDENTS AND RADIOADAPTATION

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Abstract. *The hypothesis of potentially lethal effects on biota during serious and major radiation accidents (SMA) is presented as a key event determining the characteristics of post-accident biota. At the same time, its actual observed state is the result of an evolutionarily fixed adequate reactivity to the risk of extinction. The programmed radioprotective–adaptive response includes epigenetic modifications and all levels of bioorganization, from molecular, intrapopulation (maternal effect, biosocial processes, etc.), to the formation of radiation ecosystems. The universality and effectiveness of this reactivity are the main indicator of overcoming biota extinction. Multiple anomalies imitating the effects of reduced radioresistance are also markers of damage. However, increased sensitivity to radiation in the natural environment is incompatible with the basic principles of biodiversity, which increases resistance to stress. Therefore, suppression of radioresistance cannot serve as an alternative to potentially lethal irradiation by SMA. Real SMA radiation loads are an order of magnitude lower than the doses characteristic of the observed damage to biota, ranked in laboratory and controlled conditions. It has been shown that the abnormally high radiotoxic load in SMA is provided by the synergy of damaging factors, both physical (acute/subacute + chronic irradiation) and biological (two– and multicomponent), to one degree or another associated with mutagenesis. In the early stages, for example, in rodents – hematological syndrome, then mainly reproductive damage, genetic drift, synergistically enhanced by a small population size. A constant level of increased mutagenesis against the background of a decrease in the absorbed dose may indicate a molecular-adaptive mechanism for blocking the synthesis products of damaged DNA (proteins, mRNA). Radioadaptation depends on isolation: animals living in families in an underground colony (mole vole Ellobius talpinus Pallas, 1770) are perfectly adapted, i.e. without anomalies, while the adaptation of mobile terrestrial rodents is imperfect. Radioadaptation is widely represented in the post–accident biota of the Chernobyl disaster under conditions of epigenetic transgenerational heredity. However, its implementation in the experiment has not been noted.*

Keywords: *biphasic irradiation, epigenetics, genetic drift, SMA, maternal effect, mutagenesis, radioadaptation, radioresistance, risk of extinction, synergy of factors*

1. INTRODUCTION

Serious and Major accidents (SMA) on the International Scale of Nuclear and Radiological Events, INES (level 6 and 7 on the INES scale: Kyshtym (East Ural Radioactive Trace, EURT, 1957; Chernobyl, 1986; Fukushima, 2011) are actually the main source of information about biological processes in radioactively contaminated areas. Exposure during these most "significant nuclear and radiological events" (INES) is characterized by the scale of the damage and the characteristics of the post-accident biota. These features are manifested, in particular, in discrepancies between estimates of an increasing number of dose-dependent parameters of biota and the verified accuracy indicators of radiobiological experiments. Statistically significant signs of damage to all studied representatives of various taxa of the impact biota of Chernobyl (that is, hundreds of species of plants and animals, invertebrates, vertebrates, aquatic organisms) are described. The authors believe that morphophysiological anomalies in irradiated organisms from the natural environment at a lower dose of chronic irradiation than in the experiment are a sign of increased radiosensitivity [1, 2]. Based on this, a hypothesis has been put forward about a reduced level of radioresistance in the natural environment compared to the control.

This conclusion, however, seems insufficiently correct:

- No direct evidence has been provided.

- The statement that the wild nature of the Chernobyl zone is more radiosensitive than the biota in control areas contradicts the general biological principle of biodiversity.

- The pattern that is well substantiated in radiation oncology is also violated: low radioresistance is fraught with cancer, high radioresistance minimizes radiogenic cancer [3]. Reliable cases of oncogenesis in the natural environment in SMA are rare.

- Apparently, there are no analogies in nature or they have not been given.

- Most of the other real problems of post-accident biota are not consistent with and are not explained by the conclusions of the hypothesis.

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There is a clear incompatibility between reducing radioresistance and at the same time increasing survival rate with additional sublethal irradiation of seeds and rodents from the epicenter of the SMA [4, 5].

This conclusion is also contradicted by publications that appeared in the scientific literature [6] about permanent packs of dogs and wolves in Chernobyl, that do not die out from cancer in its exclusive zone, one of the most radioactive natural focis. This is the hope of human cancer protection.

However, the hypothesis of a decrease in radioresistance in the natural environment is currently the only discussed general theory of bioprocesses in the territory of the SMA. Therefore, its refutation (or confirmation) is necessary. The reason for its apparent indisputability is the observations of anomalies. This phenomenon has been observed repeatedly, including by us [7, 8].

It would be logical to refuse to recognize the presence of morphophysiological anomalies in organisms of irradiated biota as a criterion for radioresistance. It seems more likely that the increase in the frequency of anomalies is attributable to signs of an overcoming catastrophe caused by the SMA. But for this it is necessary to show that the potential biological danger of SMA is many times underestimated. However, from the well-documented data on the dose-dependence of bioeffects [9], it is obvious that the dose load characteristic of SMA is not capable of itself leading to the lesions observed in impacted biota. Obviously, the point is a synergy of factors that dramatically enhance the effect. The changes in effects observed with complex exposure compared to their arithmetic sum are well known. A radiation dose effect an order of magnitude higher than that observed under real SMA conditions can be achieved with a corresponding increase in the mutagenicity effect.

Goal of the work: To substantiate the hypothesis of the potential threat of extinction of biota due to the synergy of acute–subacute and chronic irradiation of SMA and its consequences.

2. Materials and Methods

The results of field studies were obtained on 9 species of rodents of the East Ural Radiation Trace (EURT), living in areas with a contamination density of ^{90}Sr of 18.5 and 37 MBq/m² (500, 1000 Ci/km² – the epicenter of EURT) and 74–3700 kBq/m² (2–100 Ci/km² – the periphery of the EURT), as well as in control territories with a contamination density of 7.4–74 kBq/m² (0.2–2 Ci/km²), located at a distance of 0.5–4 km from the EURT border, as well as in areas outside the zone VURSA. The main dose-forming radionuclides at EURT are β-emitters ^{90}Sr and the daughter ^{90}Y and β-γ emitter ^{137}Cs (its specific activity in soil is two orders of magnitude lower than ^{90}Sr + ^{90}Y).

Laboratory studies were carried out on strains of inbred mice (CBA, BALB/c and BC). We assessed the hereditary component of variability (intrafamilial correlation, *R*) in the metabolism of bone-seeking toxic substances – ^{90}Sr (single administration) and stable fluorine (chronic intake) in comparison with the correlation of morphological characteristics, the hereditary condition of the development of which is known. The assessment was carried out while controlling the effects of sex, age and strains affiliation of animals, as well as the conditions of their development and litter size [10, 11].

Determination of specific activity of ^{90}Sr and fluorine concentration (F^-) in bone tissue – according to laboratory methods [12, 13].

Statistical data processing was performed using a package of licensed programs Microsoft Excel 2002 and Statistica 6.0 (StatSoft Inc.).

3. Results and Discussion

Analysis of the processes of damage and protective-adaptive reactivity in irradiated biota allows us to characterize the features of the interaction of factors of various processes and the role of their synergy.

3.1. Factors of defeat

Two-phase irradiation of SMA: 1st phase – acute/subacute emergency sublethal irradiation, 2nd phase – long-term (centuries) chronic as a result of radioactive fallout on the soil and vegetation cover.

The specific sequence of SMA irradiation is an example of a synergy that is not usually taken into account. A clear demonstration of its role is a comparison of the damage to the natural environment after the nuclear bombing of Japan (1945) and in a single nuclear test (Totsk, 1954), as well as the effects of other irradiation options. Thus, in Hiroshima and Nagasaki, the phase of acute exposure was reproduced (high-altitude explosion, minimal radioactive contamination). The damage to the natural environment is not discussed in the scientific literature. In Totsk, both phases were reproduced (an explosion at an altitude of 300 m above ground level, which resulted in the formation of radioactivity induced by nuclear radiation in the soil and dust cloud). The level of soil contamination is comparable to the periphery of the EURT. Damage to the natural environment closely models SMA [14]. The picture of the increasing radiation impact on biota during nuclear tests over time does not differ from the predictable one.

Factors of physical dual (acute/subacute and chronic) radiation ionizing injury are primary.

The radiation effect at the molecular level is carried out by secondary physical and chemical factors of direct and indirect influence:

- direct damage – transfer of radiation energy directly to the affected molecular structure;

- indirect damage by hydrolysis products (ROS and RNS, reactive oxygen and nitrogen species). They produce single and double DNA breaks and other damage.

Four biogenic factors arising during irradiation are secondary:

- DNA damage (mutagenesis and activation of selection);

- morphophysiological and reproductive radiation injuries;

- a sharp reduction in the habitat area and increase number of fragments of irradiated populations, from which new impact populations are formed;

- a significant increase in the frequency of genetic drift occurring in small populations in each of the taxa of the impact biota.

The most important features of damage and reactive defense in SMA have been described repeatedly. But the probabilities of extinction of the SMA impact biota, apparently, have not been discussed in the scientific literature.

We believe that the complex of damage and protection of SMA biota is due to the long-term threat of mutagenesis. However, its complexity seems excessive to protect the ecosystem from the relatively low risk of individual mutations. But genetic drift that occurs in small populations is harmful [15, 16]. Extinction is accelerated by increased genetic drift and weakened selection. Random events can affect the survival of a small population. Mutations introduce new alleles, some of which lead to reproductive disorders. Populations are gradually becoming more vulnerable to environmental pressures. The risk of harmful mutations increases. Against this background, the multiplication of attempts at multidirectional selection aggravates depression.

Thus, populations – the units of evolution – are the main target of the SMA. Probably, such populations died out more than once until natural selection consolidated a specialized radio-adaptive program, which included epigenetic mechanisms of protection against products of increased mutagenesis.

3.2. Radioadaptation factors in SMA

Protective-adaptive reactivity is adequate to the potential lethal risk of radiation damage. It clearly meets the main criteria: preservation of structural and functional integrity and pre-accident territorial location. However, its signs are ambiguous, distributed and expressed extremely unevenly and often not obvious.

Recent review works [4, 5] do not doubt radio adaptation. In the work [17], radioadaptation in SMA is considered as a consequence of the high radioactive background in the history of evolution. The authors are interested in evolutionary genetic adaptations that arise when the frequency of existing "protective" alleles increases or are formed de novo as a result of mutations. However, they are extremely rare against the background of mass material for natural selection. At the same time, the authors do not reject the current role of physiological adaptation, which includes epigenetics. The works [18] provide examples of radio adaptation of a wide range of animal and plant species.

Using the example of describing a butterfly from Fukushima [19] and a pine tree from Chernobyl [5] as indicators, in our work we rely on data on EURT rodents. We have described perfect and imperfect forms of radio adaptation [7, 8, 20, 21]. The beginning of studies of radio adaptation in rodents at EURT dates back to the 1970s, but we called it "imperfect" when in the 2000s we became acquainted with "perfect" in the northern mole vole (*Ellobius talpinus* Pall., 1770) from an underground colony at the epicenter of EURT. With an absorbed dose of 2 Gy/year and 12 Gy over a 6-year life, no pathological changes were found in her. For example, the cytogenetics of peripheral blood and bone marrow is without any peculiarities, while the absorbed dose, which is almost an order of magnitude lower, in rodents living on the surface of less contaminated areas manifests itself as stable shifts. A physiological sign of perfect adaptation is the background level of oxygen absorption compared to hypooxygenia characteristic of the imperfect form [22]. The most significant deviations (adaptive, but not pathological) are phenetic signs of epigenetic modification and complete isolation of the colony, established by the absence of immigrants significantly different in the level of ^{90}Sr [23]. This is an indication of a transition to a change in characteristic behavioral attributes, first of all, the cessation of a very high, up to 70% per year and higher, migration exchange of individuals of individual age strata with neighboring colonies [24] and living in complete isolation.

With imperfect radio adaptation, moderate isolation is necessary and sufficient for its development – less than 10% [25]. In work with our participation [26], the migration rate of the small wood mouse (*Sylvaemus uralensis* Pall., 1811) was estimated at 7 and 15% per year (for underyearlings and overwintered individuals, respectively). The average period for half-replacing the exposed population with migrants from uncontaminated areas is 8 years.

Adaptive traits are constantly accompanied by various anomalies. In many works, the detected anomalies are identified with pathology. This is true, for example, in cases of deformities, cataracts or aspermia. On the contrary, a moderate decrease in immunity in rodents cannot be considered an exclusively negative phenomenon, if we focus on the survival of the population and not the individual. Weakening of the immune control of the antigenic status of the gestating offspring leads to a decrease in embryonic death, an increase in litters and maintenance of the number at the control level or higher due to non–eliminated inferior individuals. This reproduction strategy during radiation damage is similar to that described by us for pike from a radiation reservoir near EURT [27]. The number of hatched eggs and prelarvae with pronounced anomalies increases. But the numbers of observed populations of rodents and fish have remained at the same level for decades.

3.3. On the role of epigenetics and some molecular mechanisms in SMA

It is not possible to reproduce radio adaptation in experiments [28]. However, this is a common occurrence in the natural environment of the SMA. There are sufficient grounds to assert that this is associated with epigenetic modification of probably the entire irradiated biota. Indeed, epigenetics has been described in EURT rodents [29], in Chernobyl pine [30], and later on other objects.

Mechanisms of epigenetics include DNA methylation, histone modification, and noncoding RNA action. Biochemical epigenetic processes in rodents are associated with the formation of alternative threshold microstructures (signs) in bone tissue, the variability of which indicates the epigenetic status of the animal and the population [31, 32]. The "recording" of information in bone structures appears in the developing bone tissue and obviously reflects a stage of more intense tension in epigenetics. Thus, at EURT and in Totsk, epigenetics is expressed, while after a natural disaster (windstorm and forest fire) the indicators are at the control level.

It is expected that with a decrease in the dose of chronic radiation, all symptoms of damage, including the mutagenic effect, will decrease in direct proportion. Then a special approach requires the interpretation of an increase in the frequency of mutations per unit dose

with a decrease in dose and radiation dose rate [4]. Perhaps this is how the restriction of the functional role of DNA manifests itself according to the gene silencing type, i.e. manifestation of the increased role of epigenetic mechanisms. This may be a condition that ensures radioadaptation in the natural environment.

One of the keys to the mechanisms of radioadaptation seems to be a well-known paradox: acute irradiation reveals adaptation to chronic irradiation, but additional chronic irradiation does not. The test detects radioresistance by the reaction to additional acute radiation.

The dynamics of increasing radioresistance of populations over a number of generations was traced in the 70s-80s of the 20th century at EURT and in the 80s-90s in Chernobyl [33, 34]. In addition to the general increase in life expectancy after a single irradiation, the number of survivors in the impact group increased, equaling or even exceeding the lifespan of intact vivarium animals. The results we obtained at EURT are consistent with those described [35]. In contrast, with chronic exposure to repeated injections of ^{90}Sr, extinction occurs almost simultaneously (Fig. 1) [27].

Figure 1. Comparative mortality with control during chronic irradiation of pygmy wood mouse from the territory of the head part of EURT [27].

We explain the phenomenon by the fact that the main adaptive mechanism of the cell is the presence of a reserve of bioantioxidants and their rapid production if necessary. When an acute high dose of gamma radiation is applied, the adapted cell meets it with a large amount of rapidly renewable antioxidants; unadapted – has no reserves and does not have time to accumulate them. Under the influence of chronic irradiation of increased power, at each moment (or in each period of time of its total duration), the irradiation power is not so high that even the antioxidants of non-adapted cells cannot cope with it - therefore they die out at the same time. But at a lethal dose, the adapted organism will still die – but later, from the inability to repair DNA.

It has been shown that radioresistance to acute and chronic ionizing radiation are different processes. During chronic irradiation, two DNA protection factors are active: neutralization of oxidizing agents and accelerated restoration of its damaged sites. Improvement in both processes may be associated with adaptation to radio radiation. But acute radiation obviously disrupts, rather than accelerates, DNA repair. Accelerated synthesis and increased supply of bioantioxidants lead to success. And, therefore, the mechanism of radioadaptation is predominantly antioxidant. This is in good agreement with the therapeutic role of antioxidants in chronic radiation injury. Bioantioxidants, for example, glutathione, provide radioadaptation in birds; increased levels of antioxidants in Chernobyl bank vole skin fibroblasts increase their radioresistance, resistance to oxidative stress, and lower sensitivity to apoptosis [18].

3.4. Maternal effect and radio adaptation

One of the leading mechanisms of radioadaptation in the post-accident biota of the SMA is the maternal effect, which affects the fitness of the offspring to the environment [*36]*. The maternal effect manifests itself at all stages of gametogenesis, conception, gestation, childbirth itself, lactation, growth and maturation. It is associated with both maternal mitochondrial inheritance (paternal mitochondria are destroyed) and epigenetics and radioadaptation.

In the EURT territory, as shown [*37*], males predominantly migrate, replacing natives. In another work [*34*], after three generations of isolation of wood mouse (*Apodemus sylvaticus*) from a radioactive environment with complete cleansing of the body from ^{90}Sr, hybrids were obtained from mothers with EURT and male control mice. These hybrids turned out to be significantly more resistant to the survival test after additional lethal irradiation than hybrids from an experimental male and a control female. This is a typical positive maternal effect. Consequently, it serves as one of the prominent mechanisms for maintaining radioadaptation. To a certain extent, thanks to radio adaptation, the offspring retain the mother's predominantly sedentary lifestyle.

Individual differences in mineral metabolism and maternal diets and the associated accumulation of ^{90}Sr in the skeleton, that is, the dose load of internal radiation, require special attention. The maternal effect plays a significant role in their regulation. Studies in the natural environment and in experiment have shown the inheritance of the metabolism of bone-seeking radionuclides (in particular, ^{90}Sr), which accumulates in bone tissue and acts as a long-term source of internal radiation. Thus, in the northern mole vole (*Ellobius talpinus* Pall., 1770), living in the EURT territory, significant interfamily differences in ^{90}Sr deposition were found (Fig. 2) [23].

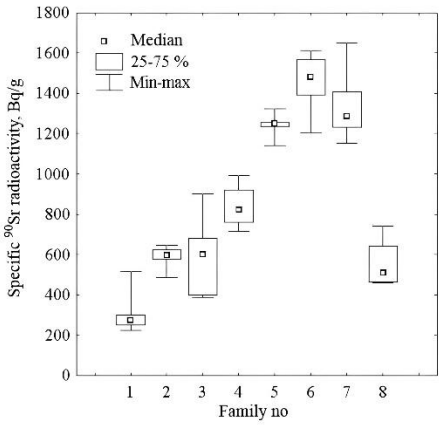

Figure 2. Specific activity of ^{90}Sr in the bone tissue of northern mole voles (n = 60) from the EURT epicenter (density of ^{90}Sr contamination – 37 MBq/m^2 or 1000 Ci/km^2) [23].

The hereditary dependence of ^{90}Sr accumulation in the litters of different female strains mice CBA (intrafamilial correlation coefficient R = 0.4–0.5; $p < 0.001$) was also revealed in a number of laboratory experiments [10, 11, 38]. Similar results were obtained for bone-seeking F^- in an experiment on mice of the CBA, BALB/c and BC strains with a significant absence of interstrain differences. The correlation of ^{90}Sr and F^- accumulation coincides with the reference correlation of morphological characters (Table). In the context of the topic of this publication, inclusion in the consideration of the metabolic features of the non-radioactive (stable), "weighty" (versus "weightless" radionuclides) non-metal anion is justified. Close analogies of the metabolism of F^- and ^{90}Sr indicate that all bone-seeking radionuclides, including radioisotopes of lanthanides and actinides that have not been studied in this regard, including Ra, Pu, U and others, must obey the identified pattern.

Table 1. Coefficients of intrafamilial correlation (R) between different test parameters recorded in experiments on the kinetics of ^{90}Sr and F^- in inbred mouse strains

R				Source
^{90}Sr	F^-	Body weight	Femur weight	
0.513	–	0.391	0.443	[10]
0.542	–	0.546	0.532	[11]
–	0.417	0.455	0.478	[10]

Genetically homogeneous strains animals differ from each other only in maternal nutrition during gestation and subsequent milk feeding and care. These same factors distinguish mice of the same litter from litters of other females of the same strain kept under the same conditions, which lead to different accumulations of ^{90}Sr and F^-. The idea of the epigenetic nature of inheritance of ^{90}Sr and F^- deposition is supported by literature data [*39*]. In particular, they indicate that females transmit biochemical signals to their offspring from the moment of implantation until the end of milk feeding, thus modulating its development. The same is confirmed by a body of work on the influence of maternal nutrition on epigenetic marks [40].

From the presented materials it follows that the maternal effect takes part in maintaining a stable level of internal exposure to ^{90}Sr and other bone-seeking radionuclides in the body of mammals in contaminated areas. At the same time, the flow of unadapted genetic material carried by "pure" immigrants does not weaken, but even strengthens radio adaptation, that is, the maternal effect is one of the noticeable mechanisms of its maintenance.

4. Conclusion

The study confirms the hypothesis about the potential risk of mortality in biota from the synergistic effect of acute/subacute and chronic radiation exposure in Major and Serious accidents. The threat of mortality was overcome by the synergistic action of protective and adaptive factors at all levels of biota organization. During the long (century) period of restoration of the affected biota to the state preceding the accident, multiple minor anomalies are detected. It is necessary to recognize their inevitability and temporary nature as a consequence of imperfect radioadaptation in a dynamically changing radiation environment. It has been shown that epigenetics plays a crucial role in ensuring the effectiveness and reversibility of recovery processes. Blocking the risks of long-term mutagenesis, which is carcinogenic and dangerous to humans, as well as other successful environmental recovery mechanisms should be considered as valuable lessons from radiation accidents.

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References

1. J. Garnier-Laplace et al., "Are radiosensitivity data derived from natural field conditions consistent with data from controlled exposures? A case study of Chernobyl wildlife chronically exposed to low dose rates," *J. Environ. Radioact.*, vol. 121, pp. 12 – 21, Jul. 2013. DOI: 10.1016/j.jenvrad.2012.01.013 PMid: 22336569
2. K. Beaugelin-Seiller, C. Della-Vedova, J. Garnier-Laplace, "Is non-human species radiosensitivity in the lab a good indicator of that in the field? Making the comparison more robust," *J. Environ. Radioact.,* vol. 211, 105870, Jan. 2020. DOI: 10.1016/j.jenvrad.2018.12.012 PMid: 30578084
3. F. Cortese et al., "Vive la radiorésistance!: converging research in radiobiology and biogerontology to enhance human radioresistance for deep space exploration and colonization," *Oncotarget*, vol. 9, no. 18, pp. 14692 – 14722, Feb. 2018. DOI: 10.18632/oncotarget.24461 PMCid: PMC5865701 PMid: 29581875
4. S. Fesenko, "Review of radiation effects in non-human species in areas affected by the Kyshtym accident," *J. Radiol. Prot.*, vol. 39, no. 1, pp. R1 – R17, Mar. 2019. DOI: 10.1088/1361-6498/aafa92 PMid: 30577037
5. S. A. Geraskin, S. V. Fesenko, P. Yu. Volkova, N. N. Isamov, "What Have We Learned about the Biological Effects of Radiation from the 35 Years of Analysis of the Consequences of the Chernobyl NPP Accident?," *Biol. Bull. Russ. Acad. Sci.*, vol. 48, pp. 2105 – 2126, Dec. 2021. DOI: 10.1134/S1062359021120050
6. G. J. Spatola et al., "The dogs of Chernobyl: Demographic insights into populations inhabiting the nuclear exclusion zone," *Sci. Adv.*, vol. 9, no. 9, pp. 1 – 16, Mar. 2023. DOI: 10.1126/sciadv.ade2537 PMid: 36867701 PMCid: PMC9984172
7. Н. М. Любашевский и др., "Жизнеспособность популяций мелких млекопитающих в радиационной и экотоксичной среде,"

Проблемы отдаленных эколого-генетических последствий радиационных инцидентов: Тоцкий взрыв: Сборник тезисов конф., Россия, Екатеринбург, 2000, стр. 54 – 66.
(N. M. Lyubashevsky et al., "Viability of small mammal populations in a radiation and ecotoxic environment," in *Book of Abstr. Problems of long-term ecological and genetic consequences of radiation incidents: Totsky explosion*, Russia, Ekaterinburg, 2000, pp. 54 – 66.)

8. Н. М. Любашевский, И. А. Пашнина, О. В. Тарасов, "Оценка здоровья среды в окрестностях города Озёрска (данные биоиндикации)," *Сборник тезисов Региональной научно-практической конференции "ВУРС-45,* Россия, Озерск, 2002, стр. 167 – 187.
(N. M. Lyubashevsky, I. A. Pashnina, O. V. Tarasov, "Assessment of environmental health in the vicinity of the city of Ozersk (bioindication data)," in *Proc. EURT-45: Regional Scientific and Practical Conference*, Russia, Ozersk, 2002, pp. 167 – 187.)
9. *Environmental Protection – the Concept and Use of Reference Animals and Plants*, vol 38, ICRP Publication 108, ICRP, Ottawa, Canada, 2008.
Retrieved from: https://www.icrp.org/publication.asp?id=ICRP%20Publication%20108
Retrieved on: Dec. 20, 2023
10. В. И. Стариченко, "Метаболизм остеотропных токсических веществ: наследственная детерминация," *Экологическая генетика*, т. 8, но. 3, стр. 27 – 37, Июнь, 2010.
(V. I. Starichenko, "Metabolism of osteotropic toxical substances: hereditary determination," *Ecol. Genet.*, vol. 8, no. 3, pp. 27 – 37, Jun. 2010.)
Retrieved from: https://journals.eco-vector.com/ecolgenet/article/view/5485/4268
Retrieved on: Dec. 20, 2023
11. V. I. Starichenko, "Hereditary component of variation in ^{90}Sr deposition in inbred mice under exogenous conditions that affect bone formation," *Appl. Radiat. Isot.,* vol. 140, pp. 126 – 132, Oct. 2018.
DOI: 10.1016/j.apradiso.2018.07.006
PMid: 30015041
12. В. И. Стариченко, Н. М. Любашевский, "Индивидуальные особенности аккумуляции Sr-90 в организме двух видов серых полевок, обитающих на территории Восточно-Уральского радиоактивного следа," *Радиац. биология. Радиоэкол.*, т. 38, но. 3, стр. 375 – 383, 1998.
(V. I. Starichenko, N. M. Lyubashevsky, "Individual characteristics of ^{90}Sr accumulation in the body of two species of gray voles living in the territory of the East Ural radioactive trace," *Radiat. biology. Radioecol.*, vol. 38, no. 3, pp. 375 – 383, 1998.)
13. М. В. Чибиряк, "Популяция домовой мыши в условиях техногенного загрязнения среды фтором," *Автореф. дисс. ... канд. биол. наук*, Екатеринбург, 1996.
(M. V. Chibiryak, "House mouse population under technogenic pollution of fluoride," *Extended Abstract of ... Cand (Biol.) Dissertation*, Ekaterinburg, 1996.)
14. А. Г. Васильев, *Отдаленные эколого-генетические последствия радиационных инцидентов: Тоцкий ядерный взрыв (Оренбургская область, 1954 г.)*, 2-е изд., Екатеринбург, Россия: Российская академия наук, 2000.
(A. G. Vasiliev, *Long-term ecological and genetic consequences of radiation incidents: Totsky nuclear explosion (Orenburg region, 1954)*, 2nd ed., Ekaterinburg, Russia: Russian Academy of Sciences, 2000.)
15. M. Lynch et al., "Genetic drift, selection and the evolution of the mutation rate," *Nat. Rev. Genet.*, vol. 17, no. 11 pp. 704 – 714, Oct. 2016.
DOI: 10.1038/nrg.2016.104
PMid: 27739533
16. M. C. Chiu, K. Nukazawa, V. H. Resh, K. Watanabe, "Environmental effects, gene flow and genetic drift: Unequal influences on genetic structure across landscapes," *J. Biogeogr.*, vol. 50, no. 2, pp. 352 – 364, Feb. 2023.
DOI: 10.1111/jbi.14537
17. A. P. Møller, T. A. Mousseau, "Are Organisms adapting to ionizing radiation at Chernobyl?," *Trends in Ecol. Evol.*, vol. 31, no. 4, pp. 281 – 289, Apr. 2016.
DOI: 10.1016/j.tree.2016.01.005
PMid: 26868287
18. T. A. Mousseau, "The Biology of Chernobyl," *Annu. Rev. Ecol. Evol. Syst.*, vol. 52, pp. 87 – 109, Nov. 2021.
DOI: 10.1146/annurev-ecolsys-110218-024827
19. A. Hiyama et al., "The biological impacts of the Fukushima nuclear accident on the pale grass blue butterfly," *Sci. Rep.*, vol. 2, 570, Aug. 2012.
DOI: 10.1038/srep00570
PMid: 22880161
PMCid: PMC3414864
20. Н. М. Любашевский и др., "Новые материалы по популяционно-генетической радиоадаптации мелких млекопитающих на ВУРСе," *Материалы междунар. науч. конф. Экологические проблемы горных территорий*, Екатеринбург, Россия, 2002, стр. 244 – 249.
(N. M. Lyubashevsky et al., "New data on population-genetic radioadaptation of small mammals on EURT," in *Proc. Int. Sci. Conf. Ecological problems of mountain territories*, Yekaterinburg, Russia, 2002, pp. 244 – 249.)
21. Н. М. Любашевский, В. И. Стариченко, "Адаптивная стратегия популяций грызунов при радиоактивном и химическом загрязнении среды," *Радиац. биол. Радиоэкол.,* т. 50, но. 4, стр. 405 – 413, Июль-Авг. 2010.
(N. M. Lyubashevsky, V. I. Starichenko, "The adaptive strategy of rodent populations living in conditions of radioactive and chemical environmental pollution," *Radiat. Biol. Radioecol.*, vol. 50, no. 4, pp. 405 – 413, Jul.-Aug. 2010.)
PMid: 20968052
22. М. В. Чибиряк, Н. Г. Евдокимов, Н. В. Синева, "Особенности энергетического обмена обыкновенной слепушонки из головной части ВУРСа," *Сб. материалов докл. VIII Всерос. популяц. семинара Популяции в пространстве и времени*, Нижьи Новгород, Россия, 2005, стр. 463 – 465.
(M. V. Chibiryak, N. G. Evdokimov, N. V. Sineva, "Features of energy metabolism of the northern mole vole from the head part of the EURT," in *Book of Abstr. VIII All-Russian. popul. seminar Populations in space and time*, Nizhny Novgorod, Russia, 2005, pp. 463 – 465.)
23. V. I. Starichenko, "Accumulation of ^{90}Sr in the bone tissue of northern mole voles in the head portion of the East Ural Radioactive Trace," *Russ. J. Ecol.*, vol. 42, no. 1, pp. 64 – 70, Jan. 2011.
DOI: 10.1134/S1067413611010115
24. Н. Г. Евдокимов, "Структура поселений обыкновенной слепушонки (*Ellobius talpinus (RODENTIA, CRICETIDAE)*," *Зоол. жур.*, т. 92, но. 3, стр. 325 – 336, 2013.
(N. G. Evdokimov, "Structure of the mole lemming (Ellobius talpinus, Rodentia, Cricetidae) colonies," *Zool. zhur.*, vol. 92, no. 3, pp. 325 – 336, 2013.)
DOI: 10.7868/S0044513413030082

25. V. I. Starichenko, N. M. Lyubashevskiy, M. V. Modorov, M. V. Chibiryak, "Skeletal ^{90}Sr as a marker of migration activity of murine rodents in the zone of the Eastern Ural radioactive trace," *Russ. J. Ecol.*, vol. 45, no. 3, pp. 231 – 241, May 2014.
DOI: 10.1134/S1067413614030126
26. E. A. Shishkina, V. I. Starichenko, E. R. Valeeva, N. M. Lyubashevsky, M. V. Modorov, "Assessment of herb field mouse (*Sylvaemus uralensis*) migration in the area of the east urals radioactive trace using measurements of bone-seeking ^{90}Sr," *J. Environ. Radioact.*, vol. 237, 106663, Oct. 2021.
DOI: 10.1016/j.jenvrad.2021.106663
PMid: 34120785
27. Н. М. Любашевский, О. В. Тарасов, А. И. Смагин, "Толерантность к добавочной лучевой нагрузке как критерий специфической адаптации к радиационной среде," *Мат. конф. Адаптация биологических систем к естественным и экстремальным факторам среды*, Челябинск, Россия, 2001, стр. 19 – 25.
(N. M. Lyubashevsky, O. V. Tarasov, A. I. Smagin, "Tolerance to additional radiation exposure as a criterion for specific adaptation to the radiation environment," in *Book of Abstr. Conf. Adaptation of biological systems to natural and extreme environmental factors*, Chelyabinsk, Russia, 2001, pp. 19 – 25.)
28. В. Л. Шведов, А. В. Аклеев, *Радиобиология стронция-90*, Челябинск, Россия: УНПЦ РМ, 2001.
(V. L. Shvedov, A. V. Akleev, *Radiobiology of Strontium-90*, Chelyabinsk, Russia: UNPTs RM, 2001.)
29. I. A. Vasil'eva et al., "Phenogenetic analysis of pygmy wood mouse (*Apodemus uralensis* Pall.) populations in the zone of the Eastern Ural radioactive trace (EURT)," *Russ. J. Ecol.*, vol. 34, no. 6, pp. 405 – 412, Nov. 2003.
DOI: 10.1023/A:1027364517929
30. O. Kovalchuk et al., "Genome hypermethylation in *Pinus silvestris* of Chernobyl – a mechanism for radiation adaptation?," *Mutat. Res.*, vol. 529, no. 1 – 2, pp. 13 – 20, Aug. 2003.
DOI: 10.1016/S0027-5107(03)00103-9
PMid: 12943916
31. А. Г. Васильев, *Эпигенетические основы фенетики: на пути к популяционной мерономии*, Екатеринбург, Россия: Академкнига, 2005.
(A. G. Vasil'ev, *Epigenetic bases of phenetics: towards population meronomy*, Ekaterinburg, Russia: Akademkniga, 2005.)
32. А. Г. Васильев, И. А. Васильева, *Гомологическая изменчивость морфологических структур и эпигенетическая дивергенция таксонов: Основы популяционной мерономии*, Москва, Россия: Товарищество научных изданий КМК, 2009.
(A. G. Vasil'ev, I. V. Vasil'eva, *Homological variability of morphological structures and epigenetic divergence among taxa: Principles of population meronomy,* Moscow, Russia: Sci. Press LTD, 2009.)
Retrieved from:
https://zmmu.msu.ru/files/%D0%91%D0%B8%D0%B1%D0%BB%D0%B8%D0%BE%D1%82%D0%B5%D0%BA%D0%B0%20%D0%9F%D0%B0%D0%B2%D0%BB%D0%B8%D0%BD%D0%BE%D0%B2%D0%B0/vasilieva-2009_homolog_inzmench.pdf
Retrieved on: Jan. 10, 2024
33. A. I. Il'enko, T. P. Krapivko, Radioresistance of populations of bank voles *Clethrionomys glareolus* radionuclide-contaminated areas," *Dokl. Akad. Nauk.,* vol. 336, no. 1, pp. 714 – 718, 1994.
Retrieved from:
https://inis.iaea.org/search/translate.aspx?RN=26029153&recordsFor=GoogleTranslate
Retrieved on: Jan. 10, 2024
34. Т. П. Крапивко, "Экология природных популяций грызунов в зоне Восточно-Уральского радиоактивного следа," *Сборник тезисов Региональной научно-практической конференции "ВУРС-45*, Озерск, Россия, 2002, стр. 137 – 166.
(T. P. Krapivko, "Ecology of natural populations of rodents in the zone of the East Ural radioactive trace," in *Proc. EURT-45: Regional Scientific and Practical Conf.*, Ozersk, Russia, 2002, pp. 137 – 166.)
Retrieved from:
https://elib.biblioatom.ru/text/regionalnaya-konferentsiya-vurs-45_2002/p137/
Retrieved on: Jan. 10, 2024
35. E. B. Grigorkina, N. M. Lyubashevsky, "On the issue of radioresistance of rodents living in contaminated areas of the Urals," in *Book of Abstr. IV International. Symp. Atomic Urals, Industrial Urals*, Ekaterinburg, Russia, 1996.
36. J. Van Cann, E. Koskela, T. Mappes, A. Sims, P. C. Watts, "Intergenerational fitness effects of the early life environment in a wild rodent." *J. Anim. Ecol., vol. 88, no. 9, pp. 1355 – 1365, Sep. 2019.*
DOI: 10.1111/1365-2656.13039
PMid: 31162628
37. N. A. Orekhova, M. V. Modorov, "Effects of environmental low-dose irradiation on functional-metabolic organ responses in a natural mouse population (Apodemus agrarius Pallas, 1771) within the East Urals Radioactive Trace (EURT) area, Russia," *Int. J. Radiat. Biol.*, vol. 98, no. 9, pp. 1414 – 1423, 2022.
DOI: 10.1080/09553002.2022.2033340
PMid: 35073242
38. Н. М. Любашевский, В. И. Стариченко, "Лактация и феномен биоразнообразия (эпигенетика минерального обмена у грызунов)," в *мат-лы Междунар. науч. конф. Генетика популяций: прогресс и перспективы*, Звенигород, Россия, 2017, стр. 152 – 154.
(N. M. Lyubashevsky, V. I. Starichenko, "Lactation and the phenomenon of biodiversity (epigenetics of the mineral metabolism of rodents)," in *Proc. Int. Conf. Genetics of Populations: Progress and Perspectives*, Zvenigorod, Russia, 2017, pp. 152 – 154.)
Retrieved from:
https://pure.spbu.ru/ws/portalfiles/portal/9329149/Conf2017_PopGen_pro_eedings.pdf
Retrieved on: Jan. 10, 2024
39. Z. J. Ge, Q. Y. Sun, "Maternal epigenetic inheritance," in *Transgenerational Epigenetics*, vol. 13, 2nd ed., Cambridge (MA), USA: Academic Press, 2019, ch. 5, pp. 75 – 105.
DOI: 10.1016/B978-0-12-816363-4.00005-5
40. B. M. Murdoch, G. K. Murdoch, S. Greenwood, S. McKay, "Nutritional Influence on Epigenetic Marks and Effect on Livestock Production," *Front. Genet.*, vol. **7**, 182, Oct. 2016.
DOI: 10.3389/fgene.2016.00182
PMid: 27822224
PMCid: PMC5075561