A NEW APPROACH TO THE CALCULATION OF THE CARCINOGENIC RADIOGENIC RISK

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ABSTRACT: *A new method of calculating the carcinogenic risk factor (CRF) in accordance with the influence of ionizing radiation is proposed based on data concerning the level of spontaneous DNA degradation. It is shown that the CRF value can be at least 2 orders of magnitude less than the generally accepted values of this parameter. Accordingly, the costs of radiation protection can be reduced to the same extent. The specified estimates were obtained using assumptions about the non-threshold action of ionizing radiation and the linear dependence of 'dose-effect' on the parameter of carcinogenic action, and may be overestimated for this reason.*

 Key words: radiation carcinogenesis, dietary risk

 Currently, the expenses for radiation protection and radiation regulation on a global scale have amounted to about 10 trillion US dollars. The approach proposed in the article for assessing radiogenic carcinogenic risk will reduce the level of current expenditures for these purposes by at least two orders of magnitude.

Our interest in this problem has arisen in connection with a paradoxical situation – an extreme discrepancy between the significance of the problem of radiation safety and regulation, on the one hand, and the number of relevant studies conducted in the countries of the former Soviet Union. The problem of assessing the risk of stochastic consequences (primarily carcinogenic) of the action of ionizing radiation has two aspects. Firstly, it is a dosimetric sub-problem (the adequate assessment of the dose of ionizing radiation, its distribution over critical tissues and organs, etc.). Secondly, it is a sub-problem of assessing the kind and type of relationship among absorbed doses and the probability of stochastic effects. As far as the analysis of the Russian literature allows, the efforts of radiobiologists in the post-Soviet space are concentrated mainly on the first aspect. As for the second aspect, in risk calculations, almost everyone is guided by the recommendations of the ICRP [1, 2].

According to WHO experts, annually in the world, out of the total number of 50 million people who die, more than 5 million people have cancer as the cause of death. In the USSR, back in the years of stagnation, malignant tumors accounted for 14% of all causes in the overall structure of mortality.

From all non-accident sources of human exposure (natural radiation background (NRF), nuclear testing, nuclear power engineering, occupational exposure (NPP workers, radiologists, X-ray specialists, pilots, cosmonauts), medical exposure for diagnostic and therapeutic purposes, the use of fertilizers containing radionuclides (40K), household radiation from goods whose work is accompanied by ionizing radiation (computer monitors and televisions), the average annual individual effective equivalent dose of radiation in 1981-1985 was 5.05 mSv. 2.25 mSv of this dose was due to exposure to NRF.

According to L. A. Ilyin [3], the average per capita dose expected during the coming life due to the Chernobyl accident for the population of the former USSR will be 1.17 mSv (0.46%), including for Belarus - 10.6 mSv (4.04%), for residents of the Zhytomyr, Kyiv, and Chernihiv regions - 3.68 mSv (1.4%), for residents of the central regions of Ukraine - 1.43 mSv (0.56%), for 272.8 thousand people living in areas of strict radiation control - 197.6 mSv (43.9%).

With the accumulation of doses in human organs and tissues from units to several tens of Sv, the relationship between the dose and the incidence of tumors is linear. The difficulties in determining the dose dependence of the frequency of malignant neoplasms (MNs) begin in the dose range that exceeds the doses from NRF but is close to the background values. The radiobiology of quasibackground doses (the doses close to the background values) does not have a sufficiently powerful arsenal of methods and means for solving this problem yet, but even now we can assume that the non-threshold concept underlying the calculation of radiation risks is taken into account only for humane reasons, the basis for which is the material possibilities of the state to protect the population from the blastomogenic effects of radiation.

For the calculations in the field of quasi-background ('low') doses, the ICRP, IAEA, UNSCEAR, USSR NCRP, Russian NCRP, and Ukrainian NCRP have adopted and continue to accept as a basis a non-threshold concept, the essence of which is the assumption of a linear dependence of the frequency of malignant neoplasms (MN) on the dose throughout the dose range. It is believed that for 1 million people per cSv in addition to the background values of absorbed doses, from 100 to 1000 or more cases of fatal cancer may occur (see Table 1) [4].

Table 1. The risk of developing radiation-induced malignancies during a person's life in the population of 1 million, each member of which received a radiation dose of 1 cSv

It is easy to see that the assessment of radiation risk is very vague, indicating once again the absence of a sufficiently rigorous level of scientific validity in the proposed assessments. Consequently, we have attempted to evaluate (reassess) it based on data concerning the spontaneous instability of DNA (thermodynamic, chemical).

However, firstly, let's see how the carcinogenic risk coefficients recommended for usage by the ICRP [1] work in relation to the assessment of the consequences of the Chernobyl accident. Taking into account the value of the collective dose received by the inhabitants of the "affected" regions of Ukraine, Belarus, and Russia (approximately 200,000 person-Sv according to the estimates of L. A. Ilyin and his colleagues [5]) and the carcinogenic risk coefficient proposed by the ICRP, the number of lethal cancers should be estimated at approximately 10,000 (we note, by the way, that the Chernobyl Forum estimates this value at 4,000), which is less than one percent of the spontaneous level of diseases of this type. Identifying such a relatively small "addition" to the spontaneous level is practically impossible, given the rather high level and significant annual fluctuations in the spontaneous level of carcinogenesis.

The comparison of the qualitative and quantitative ranges of spontaneous and radiation-induced DNA damage, as well as the frequency ranges of the distribution of cancer in individual human organs and tissues, has shown a high level of similarity. If we arrange the organs in ascending order of the probability of inducing cancer deaths per unit of absorbed dose, we get the following order: bone surface, thyroid gland, ovaries, liver, mammary gland, bladder, esophagus, red bone marrow, lungs, stomach.

When compared with several organs and tissues arranged according to the increase in spontaneous frequencies of induced cancer deaths (thyroid gland - ovaries - liver - esophagus mammary gland - skin - lungs - stomach), it can be concluded that the pattern of spontaneous cancer coincides with the pattern of radiation-induced cancer. The exception is the skin, for which the spontaneous level occupies one of the first places (resulting from UV radiation).

There is also a qualitative similarity in the spectrum of DNA damage (single breaks because of depurination and/or depyrimidinization, direct breaks, and deletions of DNA, etc.) induced by endogenous factors (thermodynamic lability of the DNA molecule, DNA-modifying enzymes and agents, ROS, etc.).

 Thus, it can be stated that there is no clear specificity in the carcinogenic effect of ionizing radiation compared to the spontaneous spectrum of carcinogenesis.

 According to existing estimates [6, 7, 8], the rate constant of DNA degradation as a result of spontaneous single-strand breaks practically coincides in various biological objects belonging to different radiotaxa (phages, bacteria, mammals, etc.) and varies in the range (1- 9×10^{-11} s⁻¹, which is six orders of magnitude higher than the corresponding constant of DNA degradation under the action of radiation from the average natural radiation background (NRF) $(2\times10^{-17} \text{ s}^{-1}).$

Based on the foregoing, we assume that the frequency of spontaneous cancers (approximately 3,000 cases per 1 million people per year [9]) is directly dependent on the level of spontaneous DNA degradation and that the frequency of cancer induced by NRF is in the same direct correlation with the level of DNA degradation induced by radiation from NRF. At the same

time, the proportion of cancer induced by NRF should be 1×10^{-6} th of the level of spontaneous cancers, i.e., approximately 0.003 additional cases of diseases per year per 1 million people.

During the year, 1 million people receive from NRF radiation a collective effective dose of the order of 0.24×10^6 cSv, which, according to our assumption, is likely to induce these 0.003 additional cases of cancer. There will be 0.012 additional cases of cancer per 1 million cSv people per year. Even over a 100-year life, this risk will be 1.2. Thus, the coefficient of the carcinogenic risk due to ionizing radiation can be at least two orders of magnitude less than the generally accepted values of this parameter. It should also be considered that our estimates were obtained using assumptions about the non-threshold and linearity of the dose-effect relationship and may even be overestimated for this reason.

It would probably be appropriate to mention the phenomenon of radiation hormesis, which, although not sufficiently studied yet to base the regulation of irradiation on it, is nevertheless essential evidence of the threshold effect of ionizing radiation. At the same time, there are sufficient grounds to consider the possible anticarcinogenic effect of irradiation hormesis, which was observed in Japanese children who suffered from the atomic bombings of 1945 – the number of spontaneous (!) leukemia cases in children irradiated at doses of 5–100 mSv decreased by two-thirds [10].

Thus, taking into account the value of the collective dose received by the inhabitants of the "affected" regions of Ukraine, Belarus, and Russia (approximately 200,000 person-Sv according to the estimates of L. A. Ilyin and his colleagues [5]), the carcinogenic risk coefficient calculated by us, the number of lethal cancer cases should not exceed 20-30 cases, which is more than two orders of magnitude lower than the estimates of the Chernobyl Forum.

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