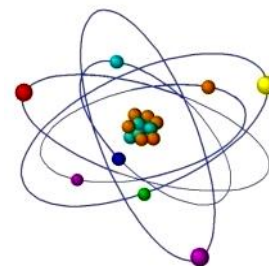


THE ROLE OF THE LEAD ^{210}Pb IN THE MANIFESTATIONS OF THE EFFECTS OF RADON EXPOSURE ON LIVING ORGANISMS: A CONCEPTUAL ANALYSIS



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ABSTRACT

The article discusses the auto-synergistic effects arising from the decay of radon in a living organism and the role of radonogenic radioactive lead ^{210}Pb in these effects. Assessments of the influence of this isotope on the long-term consequences of above effects and their relationship with various modes of radon therapy are investigated.

Key Words: radon, lead ^{210}Pb , combined effects, dose-effect model, long-term effects

It is generally accepted that the obvious pathological effect of radon exposure on the body is lung cancer resulting from a radiation burn of alveolar tissues by alpha radiation generated by the decay of radon and its daughter elements in the chest cavity [1-3]. This effect is created directly during the respiration process, although its realization takes some time due to the accumulation of radiation damages. That's why it can be considered as a long-term effect of radiation exposure.

At the same time, the existence of this primary effect is in no way an obstacle to the development of other pathological processes that have the character of more long-term consequences of radon exposure with much more complex mechanisms and require much longer time intervals for their implementation. These mechanisms include the combined impact of radon decay products. Let's consider these processes in more detail.

As you know, radon is a radioactive gas, formed during the natural decay of radium, an element of the uranium-238 series, which accumulates in rocks, soil and water. Radon is easily released from the soil into the air, where its concentration drops rapidly and is generally not dangerous. The average level of background radiation induced by radon in the atmospheric air fluctuates in the range of 5–15 Bq / m³, however, in enclosed spaces, the concentration of radon is higher, with the highest values of its concentration being observed in mines, caves and water treatment facilities. In buildings and structures, the level of radioactivity associated with radon can range from 10 Bq/m³ to more than 10 kBq/m³ [4].

It is also known that, historically, miners in underground works had an increased incidence of lung disease, which ended lethally. This phenomenon, or rather, its consequences, were known long before the discovery of radon itself, and was called the "disease of miners", being exactly the same scythe that Rock reaped the lives of underground workers, but its causes and mechanisms were revealed much later [5].

Studies of the physiological effects of "rays and emanation", begun in 1901 by A. Becquerel and P. Curie and then was continued by E. Rutherford and F. Soddy, were associated with the discovered in 1900 by F.E.Dorn and A.Debierne radioactive gas radon, and in 1904 P. Curie together with A. Laborde carried out the first studies of the radioactivity of waters and gases of mineral springs.

As a result of further research, it was found that radon is the second cause of lung cancer after smoking [1]. The mechanism that causes lung cancer, as indicated above, is considered to be a radiation burn of the tissues of the alveoli by alpha particles generated during the decay of both radon itself and its daughter

elements and the damage to chromosomes caused by them. This phenomenon occurs for the most part just in the conditions of underground workings with a pronounced saturation of air with radon and its progenies [1]. According to various estimates, radon causes from 3 to 14% of all lung cancer cases, depending on the average level of radon concentration in the soil and the prevalence of smoking in the country [1].

In addition, studies in Europe, North America and China have confirmed that radon, even in low concentrations, for example in residential areas, is also dangerous for health and is a significant factor in the incidence of lung cancer worldwide [6].

It was later found that an increase in the average value of long-term volumetric activity of radon for every 100 Bq / m³ increases the risk of lung cancer by 16% [7].

It was assumed that the dose-effect relationship is linear, and the risk of lung cancer increases in proportion to the increase of the radiation dose caused by inhalation of radon [8]. It has also been found that smokers are 25 times more likely to develop lung cancer from exposure to radon than non-smokers [8]. The risk of developing other types of cancer was not found.

The healing properties of radon were known long before the discovery of radioactivity. It was noticed that after bathing in some springs and staying in caves, people gained strength and diseases receded. The waters of Baden-Baden were known to the ancient Romans. They have built large bathing structures for their soldiers on these springs, and in 214 AD. - Imperial baths - Baths of Caracalla. Centuries later, in 1861, R. Bunsen made the first chemical analysis of the waters of the famous resort, and in 1904 the physicist H.F.Geitel and a resident of Baden-Baden, the pharmacist O.Ressler, discovered the radioactivity of Baden's waters. In the same year, the radioactivity of numerous thermal healing springs of another German resort - Bad Kreuznach, built in 1817, was revealed, and in 1912, after the discovery of radon adits, the resort opened the world's first inhalation radon center.

In 1904, the first reports appeared in the press about the radioactivity of the waters of Bad Gastein in Austria, known for their healing properties since ancient times. The world's first radon resort was built in 1911 in the Czech town of Jachymov, where Pierre and Maria Curie conducted their experiments with uranium ore, and it is rightfully called the cradle of the atomic age.

Balneological procedures with radon water are a traditional method of spa treatment and are successfully used in case of number of diseases. The high efficiency of using radon waters for health purposes has been proven by numerous experimental and clinical studies and is widely presented in the literature [9].

The specificity of radon water treatment is ionizing radiation accompanying the decay of radon and its daughter products. During radon balneotherapy, radon and its short-lived decay products, including alpha-radioactive - ²¹⁸Po and ²¹⁴Po, are deposited on the skin of patients, forming an active plaque, which is a source of α -radiation, which makes up 90% of all radiation energy emitted during the decay of radon [10-11].

Taking a bath is accompanied by the entry of radon into the body, primarily into the skin, where a kind of radon depot is formed, from where, through the microcirculation system, it enters the blood or diffuses into the internal organs, and then leaves the body at different speed, being released through the lungs (60%) or skin (40%) [11].

According to modern concepts, radon baths are α -irradiation of the skin. At the same time, it is believed that inhalation of radon during medical procedures, unless it occurs in conditions of a high concentration of radon in the air, as, for example, in mines, does not have a significant effect on the body due to its low concentration and thanks to protective devices (shields above the bathroom, aspirators and forced ventilation).

The alpha radiation of radon and daughter products interacting with a substance leads to its ionization,

which causes complex physicochemical and biological changes in cellular metabolism, stimulates oxidative processes. Radiolysis products (radio toxins) cause disruption of glycolysis and oxidative phosphorylation Processes, which underlies changes in cell function.

Under the influence of the active plaque of radon and its daughter products formed on the skin, afferent impulses in the skin increase, reaching the central parts of the nervous system, which in turn causes a stream of efferent impulses that contribute to a change in the level of functioning of organs and systems. A change in the skin analyzer leads to a restructuring of the activity of the cerebral cortex and the peripheral nervous system. Radon baths affect the nervous system at all levels.

Radon has analgesic and anti-inflammatory effects on the body, helping to eliminate chronic inflammatory processes in various organs. In low concentrations, radon increases the function of the thyroid gland and ovaries, and in high concentrations inhibits it. The complex positive effect of radon baths on the human body becomes clear within the framework of the general biological hypothesis of radiation hormesis [12]. This hypothesis is based on the assumption that low doses of ionizing radiation (equal to or slightly higher than natural background levels) are beneficial in stimulating the activation of repair mechanisms.

In addition, due to the impossibility of penetration of α -particles emitted by radon through the outer layer of human skin (the stratum corneum of the epidermis, which does not contain living cells), the only possible effective way of radon exposure to the human body is its decay inside the body or in the immediate vicinity of mucous covers.

That is why, as a result of further research, lung cancer was recognized as the most studied manifestation of the pathological effect of radon on a living organism, which is in good agreement with the historical data presented above. However, it is unlikely that a radiation burn of the lungs and the pathology caused by it are the only way of exposure to radon on the body, especially since nowadays the ability of radon to penetrate from the chest cavity through the lungs into the bloodstream and spread throughout the body is well known [13]. It is also known that part of the radon entering the body can be excreted from the body through the same systems [13].

These processes imply that a certain part of radon will decay inside the body and exert one way or another effect on biological processes in it. At the same time, today only model calculations are attempted to determine how much radon enters the body and how much is ejected back into the external environment [14-15], but it is difficult to assess how reliable are the models underlying these calculations.

For this reason, if it is necessary to estimate the radiation dose absorbed by specific organs under the influence of radon, the concentrations of radon and its daughter elements in the environment must be recalculated using certain model concepts, the study of the adequacy of which is a separate and, at best, non-trivial problem to be solved. In such cases, further effects are influenced by the ratio of the lifetimes (half-lives) of progenes and the times of their migration in the body until they are converted into long-lived isotopes, in particular, lead ^{210}Pb , which is the longest-lived radioisotope in this decay chain.

Many works today are devoted to assessments of the penetration of daughter products of radon decay into the body [16-17]. Among other things, this is due to the fact that the products of its decay, which are metals, form microscopic nodules that can penetrate both the pulmonary barrier and through the skin, and this ability is largely related to the distribution of these nodules in their geometric dimensions. Investigations of the size distributions of such nodules in specific underground workings and the construction of lung cancer risk models on this basis are also devoted to many works [18-19]. On the other hand, it should be taken into account that radon progenies (isotopes of lead and polonium, see Table 1) are very active chemical substances that easily enter into connection with biological structures.

It should be expected that the efficiency of their interaction with living matter will be the higher, the longer the time of their contact within the body. Since, as mentioned above, lead ^{210}Pb belongs to the

lead in terms of the duration of existence in the body of all the daughter radioactive elements of radon, it can be assumed that this isotope of lead will bring the maximum chemical effect into the biological consequences of the decay of radon in the body.

It also directly follows from the above table and the decay scheme that any effects of radon exposure on a living organism will necessarily be complex, associated with the indispensable participation of lead ^{210}Pb in them, including one in which this effect can be synergistic, i.e. enhancing the pathogenic properties of radiation.

In addition, today the toxicological properties of lead in the body, including the processes of its transport, accumulation and excretion, are well studied. According to these studies, lead is a toxicant that accumulates on a range of body systems and is especially harmful for young children. In the body, lead is absorbed into the brain, liver, kidneys and bones. Over time, lead builds up in teeth and bones [20-21]. Exposure to humans is generally measured by blood lead measurements [22]. Lead in the blood binds to hemoglobin, replacing iron in it and is transferred between organs, including in the form of a secondary stream. In particular, the lead accumulated in the bones enters the bloodstream during pregnancy and becomes a source of exposure to the developing fetus. It has also been established that there is no threshold concentration of lead below which it would not pose a health hazard [23].

Radon decay schema

For this reason, the long-term deposition of radioactive lead in the body, which occurs mainly in the bone tissue, in the future can lead to damage the bone marrow by the decay products of radon and lead itself, together with its chronic low-energy gamma radiation (44 keV) and cause pathological changes in the blood, including being a possible cause of leukemia.

Thus, this behavior of lead puts under the question mark the expediency and safety of any radon treatment procedures, if they create conditions for the decay of radon inside the body and, along with a large half-life of its decay (more than 22 years), respectively, to the long-term deposition of this isotope of lead in it. Let us now consider some informational properties of the indicated lead isotope in the process of radon decay. In contrast to penetrating radiation, the dose of which absorbed by a biological object can be measured instrumentally, that sequentially decays into a number of daughter elements, practically does not allow such measurements in the body.

Table 1. Decay of radon and its progenies.

Element	Decay type	Energy, MEV	Half life	Efficiency
^{222}Rn	α	5.49	3.8235 days	
^{218}Po	α	6.00	3.11 minutes	99.98%
^{214}Pb	β γ	0.67, 0.73 0.35, 0.30, 0.24	26.8 minutes	
^{214}Bi	β γ	1.54, 1.51, 3.27 0.61, 1.76, 1.12	19.9 minutes	99.98%
^{214}Po	α	7.69	164.3 microseconds	
^{210}Pb	β γ	0.06, 0.02 0.044	22.3 years	99%
^{210}Bi	β γ	1.16 0.27, 0.30	5.012 days	99.9999%
^{210}Po	α	5.30	138.376 days	
^{206}Pb	--		stable	

However, from the well-known in nuclear physics [24] and given in Table. 1 of the scheme of radon decay directly implies that the number of ^{210}Pb lead atoms formed in the body under the influence of radon is exactly equal to the number of radon atoms decayed in the same place (of course, taking into account the corrections for the latter biological withdrawal from the body).

On the other hand, the decay energy of each radon atom to lead ^{210}Pb , which is a physical constant, makes it possible to determine the radiation dose absorbed in this process in the form of the product [25]

$$E = N_{210\text{Pb}} \times P_{222\text{Ra}/210\text{Pb}} \quad (1),$$

where E is the absorbed dose, $N_{210\text{Pb}}$ is the number of ^{210}Pb lead atoms deposited as a result of radon decay, and $P_{222\text{Ra}/210\text{Pb}}$ is the decay energy of one radon atom to lead ^{210}Pb , i.e. lead accumulated in specific organs is an indicator and measure of the radiation dose absorbed by these organs. Expression (1), along with the experimentally determined amount of radiation damage, makes it possible to construct a dose-effect model that is characteristic and accepted in radiation biology. The construction of such a model requires at least two specific estimates - an estimate of the absorbed dose, which can be the amount of lead deposited as a result of exposure to radon, and an estimate of the changes caused by this dose. In this case, quantitative indicators of radiation damage, which usually means chromosomal aberrations, can participate as the effect of radon exposure [26-27].

The construction of a dose-effect model makes it possible, in principle, to establish a one-to-one relationship between the absorbed dose and the number of radiation defects caused by this dose, which makes it possible to perform forward and reverse linear prediction procedures.

On the other hand, it is known that the generation of chromosomal aberrations can occur not only under the influence of radiation, but also under the chemical influence of the same lead ^{210}Pb [28-29], which together can create both a purely additive and nonlinearly additive, in other words, a synergistic response, and there are certain indications of the existence of such an effect [1].

In this case, the degree of synergism will be determined precisely by the lifetime and biological activity of the chemical decay products, and the process itself should be qualified as **auto-synergistic**, or generated by the simultaneous effect of radiation and chemical factors of radon decay.

Therefore, the magnitude of the complex effect of radon and its decay products will be depended from two factors: radiation factor (1) and chemical factor

$$E_{chem} = N_{210\text{Pb}} \quad (2)$$

i.e. that (2) indicates the multiplex, dual role of lead in these estimates. It should be noted that such kind of nature of the processes associated with the effect of radon on a organism can be indirectly points to the well-known fact of the nonlinear relationship between the effect of radon and smoking [1] (due to the ingestion of heavy metals, including lead) on the development of pathological processes. Thus, it must be recognized that in the processes of radon exposure on the body, the radioactive lead ^{210}Pb , which is formed during the decay of radon, is also an obligatory actor, and all the effects of radon exposure are directly related to the participation of this metal in them. In addition, it becomes clear that in the described process it will be difficult to separate the effects of a radiation and chemical nature, which should be taken into account when constructing a dose-effect model. Besides, one should also take into account the different efficiency of the individual parts of the specified process. Therefore, the complete process should be written as

$$H = (N_{210\text{Pb}} \times P_{222\text{Ra}/210\text{Pb}})^\alpha + (N_{210\text{Pb}})^\beta + K_{Sinerg,210\text{Pb}/222\text{Ra}}(3),$$

where $(N_{210Pb} \times P_{222Ra/210Pb})^\alpha = N_{rad}$ is the radiation component of the total number of defects, $(N_{210Pb})^\beta = N_{chem}$ is the chemical component of the total number of defects, and $K_{210Pb/222Ra} = N_{sinerg.}$ is the synergistic component of the total number of defects. Thus, the problem of constructing a dose-effect model for radon, taking into account the processes associated with the generation and effect of lead 210Pb on living objects, is comes down to establishing a numerical relationship between expressions (2) and (3) at different levels of radon exposure (absorbed dose).

To clarify the unknown indicators of efficiency α and β , as well as the indicator of the degree of synergy $K_{Sinerg,210Pb/222Ra}$, it is proposed to conduct experimental studies on small laboratory animals, exposing them to radon and lead, including artificially introduced, at various ratios of these components and studying the biological effects of such an impact, as well as the mechanisms of accumulation, transport and removal of radioactive lead from the body in order to construct correct estimates of both the absorbed dose of radiation and the degree of synergy of the above-described phenomenon, as well as the ability to display predicted results on other biological objects, including humans.

Thus, it must be recognized that in the processes of radon exposure in the body, the radioactive lead ^{210}Pb , which is formed during the decay of radon, is also an obligatory actor, and all the effects of radon exposure are directly related to the participation of this metal in it.

In addition, it becomes clear that in the described process it will be difficult to separate the effects of a radiation and chemical nature, which should be taken into account when constructing a dose-effect model.

Considering everything above mentioned, it becomes clear that without taking into account the multifaceted role of lead ^{210}Pb generated by the decay of radon, it is not possible to construct correct estimates of the effect of radon on the body, including its medium-term and long-term consequences. Our subsequent studies are aimed at solving this problem.

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