Radon Exposure

Subjects: Others

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Radon, an imperceptible natural occurring radioactive noble gas, contributes as the largest single fraction to radiation exposure from natural sources. For that reason, radon represents a major issue for radiation protection. Nevertheless, radon is also applied for the therapy of inflammatory and degenerative diseases in galleries and spas to many thousand patients a year. In either case, chronic environmental exposure or therapy, the effect of radon on the organism exposed is still under investigation at all levels of interaction. This includes the physical stage of diffusion and energy deposition by radioactive decay of radon and its progeny and the biological stage of initiating and propagating a physiologic response or inducing cancer after chronic exposure.

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1. Introduction

Radon is a naturally occurring, radioactive noble gas that contributes as the largest single fraction to radiation exposure from natural sources [1]. It is produced by various decay chains of uranium and thorium and has no stable isotopes [2]. However, there are three naturally occurring isotopes: ^{222}Rn with a half-life of 3.825 days, originating from the uranium series, ^{220}Rn (thoron, T1/2 = 55.6 s) derived from the thorium series and ^{219}Rn (actinon, T1/2 = 3.96 s) from the actinium series [3]. As these isotopes are noble gases, there are no known chemical interactions at physiological temperatures [4].

In 1899, Rutherford and Owens discovered radiation emanating from thorium oxide and uranium [5]. In further studies, Rutherford identified a radioactive substance, permanently emitted from thorium compounds, which turned out to be ^{220}Rn [6]. In parallel, Marie and Pierre Curie discovered the ^{222}Rn isotope by studying the emanation from radium, which stayed radioactive for several days due to the comparatively long half-life of this isotope [7]. Based on the work of Rutherford and Curie, Dorn confirmed their results with both uranium and thorium [8], while Debierne discovered the isotope ^{219}Rn by measuring radioactive emanation from actinium [9].

Due to their half-lives of 3.8 days and 55.6 s, respectively, ^{222}Rn and ^{220}Rn isotopes are the only radon-nuclides that exist long enough to emanate from natural rocks and soil where they are formed. Due to its short half-life, ^{220}Rn has a shorter diffusion length than ^{222}Rn. Nevertheless, if ^{220}Rn is present, it can contribute significantly to the total inhalation dose and should not be neglected [10]. Thus, both isotopes, ^{222}Rn and ^{220}Rn, are the only significant contributors to human radon exposure from natural sources [1]. After emanation in ambient air, radon isotopes accumulate indoors and represent the most important contributor to annual radiation dose of the population [11][12]. However, the radon activity concentrations in homes highly depend on geological conditions such

as the uranium versus thorium content and the gas permeability of the soil. In addition, anthropogenic factors such as building materials, ventilation systems, or living habits play a significant role. Interestingly, some building materials are not only sources for indoor ^{222}Rn but also ^{220}Rn exposure [1], and its concentration varies considerably with the distance from the wall and the airflow [13]. All these facts together lead to large regional differences [12][14][15] and, in average, to higher radon concentration indoors than outdoors [16]. Regions like Kerala in India and cities like Yangjiang (China) or Ramsar (Iran) have particularly high radon concentrations in soil and indoors [17]. However, not only indoor accumulation, but also showering with radon-containing water releases radon to moist air, which represents a substantial source of radon exposure [18]. This fact is supported by measurements of the radon activity concentration in spa treatment rooms during filling of the bathing tubes, enhancing radon activity concentrations [19]. Nevertheless, the level of radon daughter nuclides usually remains low during filling, since they attach to vapor and are removed by ventilation and air circulation [20]. Intake of radon via drinking radon-containing water represents a minor source of exposure compared to inhalation [21].

Both radon isotopes disintegrate into several unstable daughter nuclides, emitting different radiation types (see Table 1).

Table 1. Decay scheme of ^{222}Rn and ^{220}Rn [22].

^{222}Rn			^{220}Rn		
Nuclide	**Half-Life**	**Decay-Mode**	**Nuclide**	**Half-Life**	**Decay-Mode**
^{222}Rn	3.825 d	α	^{220}Rn	55 s	α, γ
^{218}Po	3.05 min	α	^{216}Po	0.15 s	α
^{214}Pb	26.8 min	β, γ	^{212}Pb	10.64 h	β, γ
^{214}Bi	19.9 min	β, γ	^{212}Bi	60.6 min	α, β, γ
^{214}Po	164 µs	α	^{212}Po	304 ns	α
^{210}Pb	22.3 a	β, γ	^{208}Tl	3.05 min	β, γ
^{210}Bi	5.0 d	β, γ	^{208}Pb	stable	
^{210}Po	138.4 d	α			
^{206}Pb	stable				

After decay in air, the nuclides react in less than one second with trace gases and air vapor, forming clusters of 0.5–5 nm size, also called the “unattached progeny”, which are positively charged and highly mobile [23][24]. Within 100 s, those clusters may attach to aerosol particles by diffusion, described by gas kinetic laws. The parameter that mostly influences the fraction of attached daughter nuclides is the number of aerosols [25] with the influence of electrostatic forces considered to be negligible [23][26]. The formed particles build the “attached progeny” for which diffusion coefficient measurements showed three distinct size ranges. These are called nucleation mode covering

sizes from 10–100 nm, accumulation mode with particle sizes ranging from 100–450 nm and the coarse mode for particles larger than 1 µm [1]. The size distribution is strongly influenced by the aerosol mixture in the air. Accordingly, all studies show slightly different results but were consistent in the fact that the highest activity originates from radon decay products bound to aerosols associated with the accumulation mode [1][25][27]. Moreover, measurements showed that over 90% of the activity is associated with the “attached progeny” while the “unattached progeny” accounts for only 10% [21][23], being, in turn, three to five times more effective in dose commitment due to its smaller size [28].

Once built, solid daughter nuclei deposit on surfaces such as walls and furniture by different mechanisms (sedimentation, impaction, interception and diffusion), resulting in a lower activity concentration of the decay products in indoor-air than expected from equilibrium with radon [23][27]. This and other removal processes reduce the concentration of radon decay products, depending on a number of interlinked parameters such as the loss by radioactive decay, ventilation or the aforementioned deposition on room surfaces [29].

2. Radon as a Therapeutic Agent

In spite of the aforementioned risk associated with radon exposure, it is used as a therapeutic agent. In ancient history, applications of “hot bathes” as well as inhalation were basic medical principles applied for treatment of inflammatory diseases. At the beginning of the 20th century radon was found to be a therapeutic agent in several thermal springs [30][31]. Therefore, the rise of so-called radon spas started and the application of radon for relief of pain caused by chronic degenerative diseases became popular. Although there was only clinical experience, the results of several recent trials suggest a positive effect of radon treatment related to pain reduction [30][31][32][33].

At present, the main application of radon for therapy is inhalation at former mines or bathing in radon-containing water. As the application procedures and indications for treatments expanded, the EURADON (European Association Radon Spas e.V.) was founded and started to define the indications for radon application, i.e., musculoskeletal and chronic pain diseases as well as pulmonary and gynaecological diseases (see Table 2).

Table 2. List of recommended indications for radon treatment [34].

Musculoskeletal disorders and chronic pain diseases	Ankylosing spondylitis and other spondylarthropathies (AS)
	Chronic polyarthritis (rheumatoid arthritis, RA)
	Chronic arthritis urica
	Psoriasis arthropathy
	Polymyalgia rheumatic
	Arthrosis and osteoarthritis (OA)

	Degenerative diseases of the spinal column
	Auxiliary treatment consecutive to intervertebral disc operations
	Osteoporosis
	Non-inflammatory soft tissue rheumatism (e.g., fibromyalgia)
	Chronic consequences of casualty or sporting injuries
	Auxiliary treatment consecutive to orthopedic operations
	Neuralgia, neuritis, polyneuropathy
	Multiple Sclerosis (MS)
	Insufficiently healing wounds (e.g., ulcus cruris)
	Atopic dermatitis (neurodermatitis)
Cutaneous disorders and diseases	Psoriasis
	Scleroderma
	Low grade circulatory problems of the skin
	Asthma bronchiale
	Chronic-obstructive pulmonary diseases (COPD)
Pulmonary diseases	Rhinitis allergica
	Chronic sinusitis
	Praeclimacteric and climacteric disorders
Gynaecological diseases	Pelvipethia spastica

3. Conclusions

In summary, experimental research on the effects of radon exposure is needed on multiple levels. For risk assessment related to different exposure scenarios including therapeutic application, the estimations of organ doses and mechanisms of intake and elimination of radon and its progeny have to be underpinned with more solid experimental measurements. The clinical applications have to be further analyzed in high quality and placebo-controlled trials, accompanied by biomedical investigations, to increase the level of evidence of the therapy as well

as for assessment of potential side effects. This will help not only the patients directly in enhancing their mobility, but also might have a positive socioeconomic effect for an aging population.

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