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Radiation burden of healthy and asthmatic subjects due to inhaled radon progeny in indoor environment

Introduction: Radon progeny inhalation is the main risk factor of lung cancer worldwide. Radon is a noble gas, what is mainly inhaled and exhaled without decaying but its progeny can deposit and spend hours in the airways. Due to their short half-life 218Po and 214Po isotopes are likely to emit alpha-particles after deposition. These alpha-particles can reach the radiation sensitive basal and secretory cells of the airway epithelium. In indoor environment the average radon concentration is around 50 Bq/m3, in contrast to the outdoor value, where the estimated annual average value is approximately10 Bq/m3. Since people spend more than 80% of their life in indoor environments (Trassierra et al., 2016), it is an important task to understand the health risks of this element for healthy and asthmatic subjects (around 300 million people are involved worldwide) as well.
Objective: To compare the bronchial deposition of the inhaled radon progeny and cell nucleus doses originating their alpha-decay for a healthy adult and an adult with asthma.
Methods: The airway deposition distribution of the inhaled attached and unattached radon progeny was calculated with the Stochastic Lung Model (Koblinger and Hoffmann 1990, Hofman and Koblinger 1990,1992). This model has uniquely fine, airway generation (the level of airway bifurcations starting from the trachea) specific resolution. In addition, this model is very flexible, so it is able to simulate the pathway and deposition of the inhaled particles in healthy adults or children's airways. With this model it is possible to simulate the altered airway geometry and breathing pattern for asthma and Chronic Obstructive Pulmonary Disease (COPD).
The location of alpha-decays during mucociliary clearance was calculated by a self-developed Monte Carlo clearance model. As the next step, the pathway of the emitted alpha particles and the absorbed energy in the nuclei of the basal and secretory cells of the airway epithelium was calculated with a radon dosimetry model.
Results: In asthma, the bronchial airways are contracted, and the mucus is thicker and slower than for healthy subjects. The inhaled air volume for the asthmatic person was higher, than for the healthy subject and the breathing frequency was also higher, what resulted in higher inhaled progeny number, than for the healthy subject. For the healthy person, the breathing was symmetric (the inhalation time was equal to exhalation time) but for the asthmatic subject, the exhalation was longer than the inhalation. These differences have a considerable effect on the airway deposition rate (deposited progeny number/min) of the inhaled radon progeny. For the asthmatic subject, much more radon progeny deposit in the bronchial airways than for the healthy adult. For the asthmatic subject, the airways are contracted resulting in shorter alpha pathways from the emitter 218Po and 214Po to the nuclei of the basal and secretory cells of the airway epithelium. Synergistically with the higher deposited progeny number, this usually results in higher absorbed cell nucleus doses for the asthmatic subject.
For asthma, the airway covering mucus is much thicker, than for the healthy subject resulting in some shielding effect especially in the big bronchial airways, but this usually cannot fully compensate for the above mentioned dose-elevating factors (higher deposited number and shorter alpha pathways).
Conclusion: Present results indicate that health status of the subjects is important in radon dosimetry. Inhaling the air with the same radon concentration, in asthma, usually much more energy is absorbed in the nuclei of the radiation sensitive secretory cells of the airway epithelium.
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