

CHRONIC SMOKER LUNG DOSIMETRY OF RADON PROGENY

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Abstract: The purpose of this study is to determine how cigarette smoking affects the dose of radon progeny in the lungs. An extensive survey of the Medical Literature suggests that about six physiological parameters may influence deposition as well as clearance of radon progeny and thus, affecting the resulting doses received by sensitive bronchial target cells. The calculations reflect a difference between the dose of a nonsmoker lung and the lung of the heavy long-term smoker, of a multiplicative factor 1,8.

Key words: radon, dosimetry, smoker, lung.

1. INTRODUCTION

Inhalation of airborne short-lived radon progeny in the indoor and outdoor environment yields the greatest amount of natural radiation exposure to the public. When radon decays it forms its short-lived progenies (^{218}Po to ^{214}Po). The progenies can be collected electrostatically on tiny dust particles, water vapors, oxygen, trace gases in air and other solid surfaces. These dust particles (aerosols) can easily be inhaled and attached to the bronchial epithelium, producing a high local radiation dose. Alpha radiation is one of the most harmful ionizing radiation due to its high Linear Energy Transfer (LET). It can induce DNA doublestranded breaks and the development of cancer. The attachment of the progenies with the aerosols in the air depends upon the size of aerosols that varies with the ventilation rate, cleanliness and living styles of the inhabitants. Typically, the estimated absorbed dose to the critical cells of the respiratory tract, per unit of radon exposure are derived from the analysis of information on aerosol size distribution, unattached fraction, breathing rate, fractional deposition in the airways, mucous clearance rate, and location of the target cells in the airways [1].

In the case of internally deposited radionuclides, direct measurement of the energy absorbed from ionizing radiation emitted by the decaying radionuclides is rarely, if ever, possible. Therefore, we use the dosimetric models.

Various authors have attempted to evaluate the dose to the respiratory tract due to the inhalation of radon daughters. Many publications have dealt with the issue of radon dose in reference to the lung derived from physical dosimetry. A significant problem in internal radiation dosimetry is the discrepancy between the radiation dose from exposure to radon inferred from epidemiological studies and the higher dose calculated using the Human Respiratory Tract Model (HRTM) adopted by the International Commission on Radiological Protection (ICRP) [2]. The agreement between these two assessments by radically different approaches is surprisingly good; therefore, it is useful for comparative dosimetry.

The main objective of this study is to make a comparison between the doses of radon progeny in a normal lung and in a smoker lung. Accordingly, will calculate the dose of the smoker lung using the stochastic model detailed presented in [3, 4] in which we insert new parameters analyzing the physiological changes of the lung in chronic smokers as cigarette smoke destroys the normal function of the lung and the self-protection mechanism [5]. It is worth mentioning that used model takes into account the variability of the physiologic, morphometric and histological parameters of the lung.

For the heavy, long-term smoker group the specialty literature discusses the differences between appropriate effects and appropriate values. The effects in this group are chronic and can highly influence the deposition, clearance, as well as, activity values.

2. METHODS

For the present calculations, the stochastic dosimetry model IDEAL-DOS [4] was used, which considers the effects of intra- as well as inter-subject variations of morphological, physiological and histological parameters involved in lung dosimetry, applying Monte Carlo techniques [6]. The dosimetric model consists of three parts: *i* – the original deposition model IDEAL (Inhalation, Deposition, Exhalation of Aerosol in/from the Lung) [7], *ii* – the bronchial clearance model [8, 9], and *iii* – the bronchial cellular dose model [10, 11].

The stochastic deposition model simulates the random walk of inspired particles through a random airway geometry, represented by a sequence of asymmetrically dividing airway bifurcations [12]. Upon inhalation, linear airway dimensions are randomly selected from their distributions and correlations at each airway bifurcation. The probability that a particle selects the major or minor

daughter airway at a given bifurcation is proportional to the splitting of the airflow, assumed to be proportional to distal lung volume. Deposition of particles in bronchial airways is computed by analytical equations for different physical deposition mechanisms, such as diffusion, impaction and sedimentation, i.e. the deposition probabilities of individual particles are given by their average probabilities. In case of a deposition event, the particle continues its path with decreased statistical weight. Deposition fractions for bronchial airway generations are typically based on 10,000 to 100,000 simulations.

The filtering efficiency of nasal passages for submicron particles was considered by empirical equations derived from in vivo measurements [13]. Deposition by Brownian motion in upper bronchial airways was determined by the empirical equation proposed by Cohen and Asgharian [14], to account for enhanced deposition due to developing flow. Outside the range of flow rates and airway dimensions of this relationship, i.e., in more peripheral airways, equation for diffusion deposition under parabolic flow conditions was applied [15]. The magnitude of deposition by inertial impaction in upper bronchial airways was calculated according to Yeh and Schum [16].

The stochastic bronchial clearance model considers both fast and slow bronchial clearance phases. Due to conservation of mass, average mucus velocities in a given airway generation in asymmetrically branching airways are proportional to their respective diameters and mucus velocities in individual bronchial airways are normalized to a tracheal mucus velocity of 5.5 mm/min [2]. The dependence of the magnitude of the slow bronchial clearance fraction with a half-time of 10 days on geometric particle diameter is modelled by an empirically-derived relationship [17]. Half-times for the transport through epithelium into blood are assumed to be 10 hours for attached progeny and 1 hour for the unattached fraction [18]. Connecting the stochastic clearance model to the stochastic deposition model and considering the radioactive decay of the three short-lived radon progeny allows the calculation of the radon progeny activities retained in a given airway generation. Assuming steady-state conditions for continuous radon exposures, alpha-emitting ^{218}Po and ^{214}Po surface activities, obtained by dividing the activity retained in a given generation by the total mean surface area of that generation, are computed and subsequently normalized to an exposure of 1 WLM.

The targets of interest in the bronchial epithelium are the basal and secretory cells, assumed to be the progenitor cells of lung carcinomas [2]. The mean depths of these critical cells in the epithelium are based on the data of Mercer *et al.* [19]. Dose as a function of depth in bronchial epithelium is computed for uniform ^{218}Po and ^{214}Po activities on cylindrical bronchial airway surfaces, considering both near wall and far wall contributions. The output of the dosimetric model program presents basal and secretory cell doses (in mSv/WLM), normalized to a cumulative exposure of 1 WLM.

Our model presumptions are made on following suggests out coming from an extensive survey of the literature about the physiological parameters which are changed by cigar smoke and may affect deposition, clearance and resulting dose received by the sensitive bronchial target cells.

For the heavy, long-term group the specialty literature conclude to appropriate effects with appropriate values, the effects in this group are chronic and can influence very high the deposition, clearance, as well as activity values. The following physiological parameters may affect the dose received by the sensitive bronchial target cells [2, 3, 20-26]:

- i.* Mucus production by increased thickness of the mucus layer;
- ii.* Mucociliary clearance by decreased velocity as well as cilia killed;
- iii.* Lung volumes and capacities a breathing pattern more frequent and deeper together with a changed vital capacity and volumes;
- iv.* Airway obstruction and structural alveolar changes by bronchitis (inflammation of airways, mucus hipersecretion) as well as by emphysema (loss of elasticity, alveolar collapse, alveoli structure destruction);
- v.* Cellular changes of epithelial tissue manifested by thickening of bronchial epithelium by loss of ciliated cells and their replacement by squamous epithelium, basal-cell hyperplasia or dysplasia and goblet cell metaplasia;
- vi.* Penetrability of bronchial epithelium due to increased viscosity of mucus as well as to bronchial epithelial lesions.

3. RESULTS AND DISCUSSION

For the purpose of comparison between the doses of a normal and a smoker's lung, we have utilized the total effective dose normalized by 1 WLM, calculated for the layers that contain the basal and secretory cell nuclei. For the normal lung we have calculated the effective dose equivalent to 13.1056 mSv/WLM, in accordance with the ICRP66 approach for the lung morphometry.

Initially, we have calculated the dose by separately changing each parameter. This procedure was conducted to observe the manner in which the parameters influence the dose.

The parameters that were altered during the process include: *I* – the volume of the lung, *ii* – the thickness of the mucus layer, *iii* – the breathing cycle period, *iv* – the clearance velocity on different values. The variability of the dose by volume in a smoker's lung is insignificant when we use the values of the lung volumes as found in literature. The dose is higher if the residual capacity is smaller. We have calculated the dose for two different values, one from the ICRP 66 (a–Tables 1, 2) and the other (b–Tables 1, 2) which can be considered as the

smallest typical adult lung functional residual capacity cited from spirometric measurements. The increased thickness of the mucus layer is directly related to the higher mucus production, which does not have a protective role, as expected, in the case of radon progeny; the result is a higher dose.

The hyperplasia of the radiosensitive secretory cells makes these cells a superior target for the radiation. The higher breathing frequency brings a higher amount of particles in the lungs that can be deposited in the airways. The clearance in a smoker's lung is almost completely dissolved because of the impairment of the cilia muscles. Consequently, the clearance velocity is reduced and, as a result, the dose is higher. The clearance of the mucus also means a clearance of the particles. If the clearance is reduced the amount of harmful particles is higher. Each parameter was calculated to form an individual mean value. The mean values were used to calculate the dose, which included all changes of the physiological parameters in the chronic smoker's lung. These changes are presented in Table 1.

The heavy, long-term smoker group in which all the protective mechanisms of the lung are destroyed include as follows: the cilia are killed, the mucus cannot be cleared anymore and thus it obstructs the tiny airways. Due to this destroyed mechanism the radioactive progenies remain deposited within the lungs.

Table 1

Most influent values of the lung input parameter changed by chronic smoking [21–26]

Parameter	Normal values	Changes of the normal values in the chronic smoker
Thickness of mucus layer	5 μm	5 times thicker
Clearance velocity	5.5 mm/min	1.1 mm/min
Breathing pattern	12 breath/min	20 breath/min
Lung volume FRC	3300 cm^3	a. 2650 cm^3 b. 3300 cm^3

Due to the forced cough clearing mechanism, the probability of lesions appearing in the epithelia is eminent; the radioactive progenies can interact with the radiosensitive target cells directly. The higher breathing frequency of the chronic smoker increases the number of inhaled radon progeny. In result to these hypotheses, the dose is approximate two times higher then in the case of the normal lung (Table 2). The dose where calculated for four different particle sizes. The total dose was calculated as the weighted mean value of the doses of each particle size.

Table 2

Effective bronchial doses rates of non-smokers and chronic smokers

Particle size (μm)	Relative frequency of the size distribution	Effective dose rate mSv/h by 1WL for chronic smoker	Effective dose rate mSv/h by 1WL for the normal lung
		a. b.	
0.0009	0.08	0.2161 0.1843	0.1599
0.05	0.258	0.2663 0.2279	0.1370
0.25	0.644	0.0877 0.0743	0.0442
1.5	0.018	0.0648 0.0507	0.0274
Mean effective dose rate		a. b. 0.1436 0.1215	0.0771

4. CONCLUDING REMARKS

The most important effects of cigarette smoke on the lung physiology, which are affecting the dose of radon progeny, are as follows: decrease of the mucus clearance velocity, thickening of the mucus layer, cells metaplasia and higher breathing frequency.

The chronic exposure to tobacco smoke induces changes in the breathing frequency, which can result in an increased inhaled dose. Also induced changes in the epithelial structure, the metaplasia of the goblet cells into inflammatory cells, which are situated very close to the contact surface of the epithelia with the air as well as with the radon progeny carried by air. The ciliated columnar cells are forever lost and as a result all the toxins become impossible to be cleared from the lung. This creates lesions into the epithelium and the lesions are a perfect path for the radon progeny to attack the basal cells.

Another effect that is important for understanding the higher dose in the case of chronic smoker is the basal cell hyperplasia so the target cell is larger and the impact probability with the alpha particle is higher.

All these effects should be taken into consideration in future, in dosimetry researches but also in medical studies and practices.

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