



**DEPOSITION ANALYSIS OF ATTACHED
FRACTION OF RADON PROGENY IN THE
HUMAN RESPIRATORY TRACT**

By

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This Work is Dedicated
to
My parents whose loving memories still guide me
through difficult times.

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Acronyms and Abbreviations Used

Acronyms

IAEA ≡ International Atomic Energy Agency

NCRP ≡ National Council on Radiation Protection

NORM ≡ Naturally Occurring Radioactive Material

WNA ≡ World Nuclear Association

UNSCEAR ≡ United Nations Scientific Committee on the Effects of Atomic Radiations

US EPA ≡ United States Environmental Protection Agency

WHO ≡ World Health Organization

BEIR ≡ Biological Effects of Ionizing Radiation

IARC ≡ International Agency for Research on Cancer

ICRP ≡ International Commission for Radiological Protection

EPA ≡ Environmental Protection Agency

IARC ≡ International Agency for Research on Cancer

HRTM ≡ Human Respiratory Tract Model

DCF ≡ Dose Conversion Factor

Abstract

In this work, the regional deposition analysis of attached fraction of radon have been studied. So as to find the regional deposition of the inhaled activity, a fortran code is used. The parameters and the assumptions used in the code were developed by ICRP. The regional deposition from modified parameters were compared with ICRP model deposition. According to the comparison, it was observed that the deposition of attached fraction in the lower (trachea-bronchial) regions of the respiratory tract was found small in contrast their deposition in the extrathoracic (ET) region becomes dominant as aerodynamic diameter of aerosols increases.

Introduction

Humans, animals and plants have been exposed to natural radiation since the creation of life. More than 3.5 billion years ago, when the living organisms appeared on the Earth, the level of natural radiation was about three times higher than its current level. Radon is major sources of background radiations. It contribute about 55% of the natural radiation dose to humans. [1].

Radon is responsible for the majority of the mean public exposure to ionizing radiation. It is often the single largest contributor to an individual's background radiation dose (55%), and is the most variable from location to location [2]. Radon and its short-lived decay products (^{218}Po , ^{214}Pb , ^{214}Bi and ^{214}Po etc.) in buildings is the major source of public exposure to natural radioactivity, making up almost 55 percent of the worldwide mean effective dose [1].

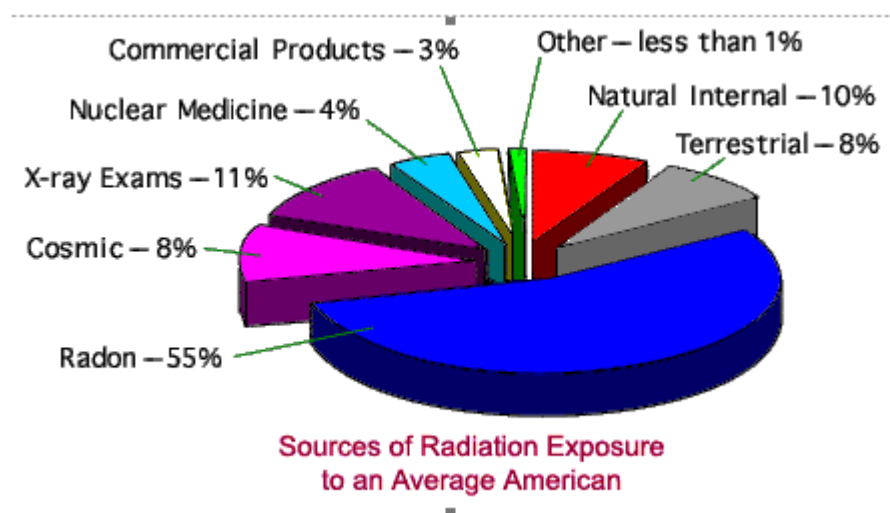


Figure 1: Major sources of background radiation

The relationship between radon exposure and lung cancer has already been demonstrated in epidemiological studies performed on cohorts of miners. Radon comes from the natural (radioactive) breakdown of uranium in soil, rock and water and gets into

the air we breathe. Because of radon is radioactive gas it produces a number of decay products called radon progeny once it has been deposited inside the lung by emitting highly ionizing alpha radiations [3].

A lot of work has been done to determine the doses in the human lungs due to short-lived radon progeny. Since the dose on lung tissue can not be measured directly [3], many lung models were developed to deal with the determination of dose delivered by alpha particles in the lungs because of their low range and relatively high and discrete energy (6 MeV for ^{218}Po and 7.69 MeV for ^{214}Po).

One single most important mechanisms by which the lung is exposed to radon daughters is deposition of aerosols in the different parts of the human respiratory tract. Deposition of aerosols that are inhaled into the lung can be attached to radioactive progeny of radon. These attached radon progeny are called radioactive aerosols and are major contributors to the lung dose.

In this work comparison is made between the predictions of different models and deposition fractions of the inhaled aerosols in the five regions of respiratory tract.

Rationale of The Study

Even though radon concentration varies from place to place and from season to season, it is found elsewhere. Further radon is a major reason for higher incidence of lung cancer among miners in the world. In light of the developing mining industry, in our country, it is imperative to assess radon level in air and develop methods measurement and analysis of parameters that determine the radiation dose due to radon. The knowledge generated can be used as a starting point for radiation protection and future studies.

Objectives of the Study

The major purpose of this study is to study the deposition of attached radon progeny predicted by developed mathematical model with the ICRP model.

Specific Objectives

The specific objectives are:

1. to study the deposition effect of newly modified ICRP parameters in the respiratory tract.
2. comparison of the resulting regional deposition with the ICRP66 model.

Review of Related Literature

1.1 Radon and its Physical Properties

1.1.1 The Element Radon

Radon is a chemical element with symbol Rn and atomic number 86. It is a radioactive, colorless, odorless, and tasteless noble gas. So that it cannot be detected without special equipment. It has melting point of -71°C and boiling point of -61.8°C . It occurs naturally as an intermediate step in the normal radioactive decay chains through which thorium and uranium slowly decay into lead; radon itself is a decay product of radium. Its most stable isotope, ^{222}Rn , has a half-life of 3.8 days. Since thorium and uranium are two of the most common radioactive elements on Earth, and since their isotopes have very long half-lives, on the order of billions of years, radon will be present in nature long into the future in spite of its short half-life as it is continually being regenerated. Thus, uranium and radon, will continue to occur for millions of years at about the same concentrations as they do now.

Radon is an inert gas therefore it is a noble gas. It is the last member of the noble gas family. The term inert means incapable reacting with other substances. Radon does not react with air, water and others, but its decay daughters are electrically charged so that they are reactive. This is chemical property of radon [4].

1.1.2 Radon Isotopes

Radon has no stable isotopes. Even though there are 37 radioactive isotopes have been characterized with atomic mass ranging from 193 to 229, it has three well known isotopes, the first isotope ^{222}Rn is found from decay chain of uranium-238 which is the most stable isotope with half-life of 3.8 days. Radon is also generated in the other two series. However, these isotopes of radon are of lesser radiological

<i>RadonIsotopes</i>	Half-life	Decay Series	Name
^{222}Rn	3.8 d	^{238}U	Radon
^{220}Rn	56 sec	^{236}U	Thoron
^{219}Rn	3.96 sec	^{235}U	Actinon

Table 1.1: Isotopes of radon

importance since there concentration is small compare in air [4]. The thorium series generates ^{220}Rn , which is also called thoron obtained from Th-232 series. ^{220}Rn has a half-life of 56 s and therefore has a much greater chance to decay before becoming airborne. The third isotope is called Actinon, ^{219}Rn , is a noble gas produced by the decay of radium, ^{223}Ra , which is a member of uranium-235 decay series. It has 3.96 second half-life [4].

1.1.3 Radioactivity of Radon

Radon is naturally occurring radioactive noble gas that has been found from the decay chain of uranium isotopes (^{238}U , ^{236}U and ^{235}U). It is present in trace amounts in all rocks and soil. After the decaying of ^{222}Rn , the next members of the decay chain are ^{218}Po , ^{214}Pb , ^{214}Bi and ^{214}Po which are known as the short-lived decay products of radon. Radon and its decay products are found in variable concentrations indoors, outdoors, and in mining environments.

1.1.4 Sources of Radon

Radon is radioactive gas which comes from the natural breakdown of uranium in soil, rock, building materials, groundwater and mining areas. Confined areas of house such as basements where air is not move freely, some openings and holes of the homes are also sources of radon. Nevertheless, the ground is the major radon source. The lower air pressure indoors gives rise to a pressure-driven flow of radon-rich soil air into the indoor environment through cracks in the bottom slab and cellar walls. Certain rocks and soil, such as some granite and shales, contain more uranium than others. However, ground with moderate contents of uranium or radium can also give high indoor radon concentrations. The inflow depends largely on the building construction and the permeability of the ground materials [5].

Building materials made from soil (e.g. clay bricks) or rock always contain uranium and radium. The content is usually low, but materials may have high concentrations of radium-226, for example alum shale concrete and building materials made of volcanic tuff, gypsum waste, etc [2]. The radon concentration can reach several

thousand becquerels per liter (Bq/l) in water from drilled wells in regions with granite rock, for example in Finland and Sweden. This contributes to indoor radon and to exposure via ingestion.

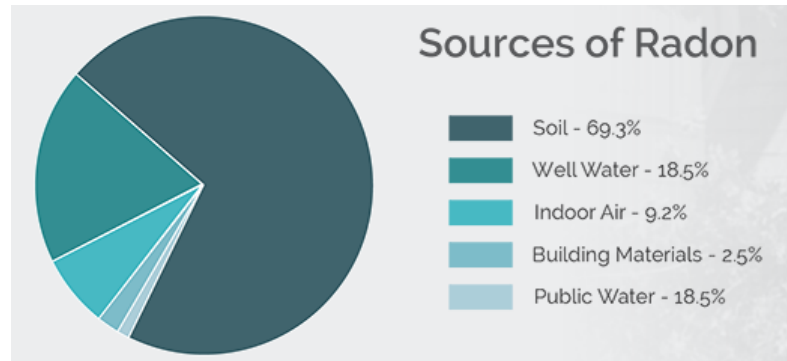


Figure 1.1: Major radon contributors

1.1.5 Radon Decay Products

All of the heavy elements (Z greater than 83) found in nature are radioactive and decay by alpha, beta or gamma emission. When radon in air decays, it forms a number of short-lived radioactive decay products (called radon progeny), which include polonium-218, lead-214, bismuth-214 and polonium-214. All are radioactive isotopes of heavy metal elements and all have half lives that are much less than that of radon. Two of the alpha emitting daughters of ^{222}Rn (^{218}Po and ^{214}Po) contribute over 90% of the total radiation dose attributable to exposure to radon.

Radon is descendant of one of the naturally occurring radioactive series that starts with ^{238}U . Uranium and its first five daughters are solids that remain in the soil, but the fifth daughter ^{226}Ra decays into ^{222}Rn . This daughter, called radon, is a noble gas, not bound chemically in the material where its parents resided. The chart below lists all decay products of radon gas (radon-222) in their order of appearance. They are called radon progeny (radon daughters). Each radioactive element on the list gives either alpha radiation, beta radiation and sometimes gamma radiation too. The transformation continues up to stable lead-206, the last element in the list, which is not radioactive, it doesn't decay.

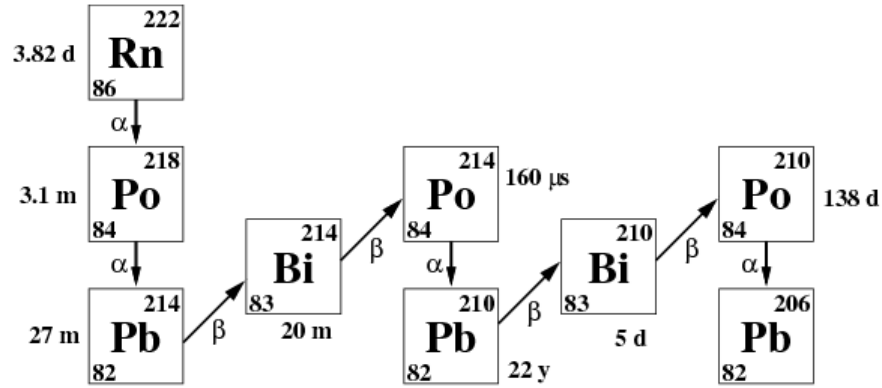
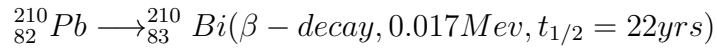
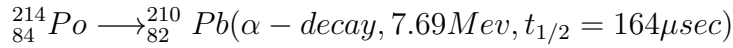
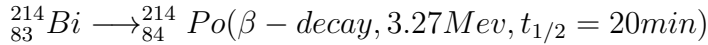
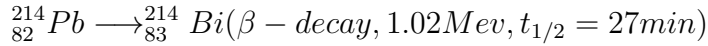
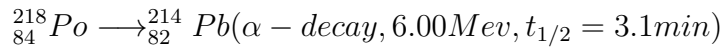
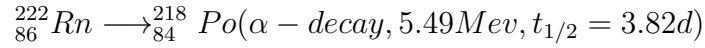


Figure 1.2: Radon decay chains

The general expression of the above graph, the type of decay which is performed and the amount of radiation released is given as follows;



etc. down to stable ${}_{82}^{206}\text{Pb}$.

1.2 Radon Decay and Aerosols

1.2.1 Aerosols in Air

The Earth's atmosphere is a two-phase system consisting of gases and particles (solid or liquid). This means the entire atmosphere is, by definition an aerosol. Aerosol refers to suspension of liquid or solid particles in a gaseous medium and they are formed by the conversion of gases to particles or by the disintegration of liquids or solids into finer constituents [6].

When the particles are all the same in size, an aerosol is termed monodisperse. This is extremely rare in nature. Generally, particles vary in size, and this is called polydisperse. When its particles are chemically identical, an aerosol is called homogeneous. When particles are spherical, their radius or diameter can be used to describe their size. Since most particles are not spherical, however, other parameters must be used. Often the diameter is defined in terms of particle settling velocity and diffusion coefficient. All particles with similar settling velocities or diffusion coefficients are considered to be the same size, regardless of their actual size, composition or shape.

When a radioactive nuclide decays, electrons are stripped from the parent atom by its recoil and decay products are formed as positive ions. These ions can attract liquid and even solid material, thus forming clusters of atoms or particles in the submicron region ranging from 0.0005 to 10 μm [7].

The gas radon (^{222}Rn) is emitted from the ground into the atmosphere, where it decays and forms daughter products, isotopes of polonium, bismuth and lead, which either remain airborne until they decay, or are deposited by diffusion to the ground.

1.2.2 Attached and unattached radon decay products

Radon decay products appear in two size modes, these are,

1. attached mode and
2. unattached mode

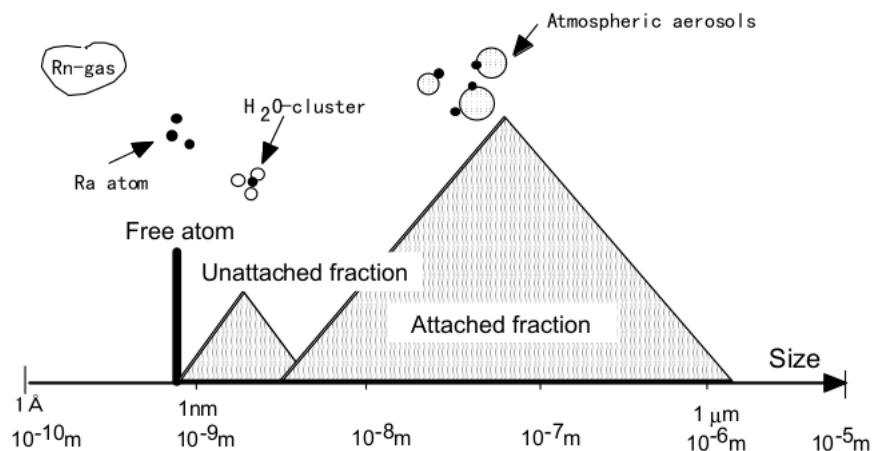


Figure 1.3: Size distribution of radon decay products

Attached Radon Decay Products

The decay products of radon (^{222}Rn) are in solid state and become attached to aerosols and dust particles in the air due to their electrostatic charge. They frequently collide with and attach themselves to large dust particles within the air inside a dwelling [3]. The effective size of such attached decay products ranges from 0.05 micrometer to a few micrometers in diameter or greater than $1\mu\text{m}$.

The rate of attachment of radon progeny depends on the following factors:

1. aerosol concentration of the surrounding
2. the electrostatic charge of the progeny
3. humidity of the surrounding environment

Depending on the concentration of dusts in the room air, up to 80 % of the decay products will attach to dust particles. So, the attached fraction of radon decay products are defined as the ions or atoms of radon decay products that are attached to aerosol particles. The particle attached fraction is normally considered to consist of three different modes. The accumulation mode with a median diameter of the activity weighted size distribution between 100 - 400 nm. A nucleation mode between 10 - 100 nm with non- aged aerosol, where the particles (e.g. candle burning, and natural gas heating) are produced by nucleation from the gas-phase. A third mode with larger particles (coarse mode, particles $> 1\mu\text{m}$) is sometimes present in polluted dusty areas e.g. at work places. The attached fraction of radon can be found in three modes; nucleation, accumulation and course mode depending on the size. Raising aerosol concentration increases equilibrium factor, because the attachment process becomes much faster than the loss of atoms by plateau. Relative humidity also modifies the condition of attachment of free fraction to aerosols by changing the total number of condensation nuclei; a high level of humidity (often close to 100 percent in under ground mines) reduces the proportion of free fraction [8].

Unattached Radon Decay Products

Unattached fraction of radon decay products are the short-lived radon progeny especially those of ultrafine progeny smaller than 2 nm in diameter. Unattached Fraction of radon decay products are in small clusters or attached to very small Particles (1-10) nanometer in diameter. Depending on the concentration of dust in

the room air, up to 20 % of the decay products will not attach to dust particles and will remain in free [3]. They tend to attach to other polar molecules in the air, such as water vapor and other atmospheric gases, and are believed to exist, by making small and highly diffusive ion clusters.

The fraction of unattached (free) ions, though with important contribution to the dose in the lung, was determined by several methods and considered small (about 15%), as compared to the fraction of the attached once. The fraction of unattached nuclei is an important determinant of the dose received by the target cells; as the unattached fraction increases, the dose also increases, because of the efficient deposition of the unattached fraction in the airway.

1.3 Units of Measurements in Radon

Radon concentration is the amount of radioactivity per unit volume of space, i.e activity per liter. Radon concentration is usually measured in the atmosphere in becquerels per cubic meter, which is an SI derived unit. As a frame of reference, typical domestic exposures are about 100 Bq/m³ indoors and (10–20) Bq/m³ outdoors. In the US, radon concentrations are often measured in picoCuries per liter (pCi/l) with,

$$1 \text{ pCi/L} = 37 \text{ Bq/m}^3,$$

The mining industry traditionally measures exposure using the working level (WL) index, and the cumulative exposure in working level months (WLM): one WL equals any combination of short-lived ²²²Rn progeny (²¹⁸Po, ²¹⁴Pb, ²¹⁴Bi and ²¹⁴Po) in a liter of air that releases 1.3×10^5 MeV of potential alpha energy. One WL is equivalent to 2.08×10^{-5} joules per cubic meter of air (J/m³). The SI unit of cumulative exposure is expressed in joule-hours per cubic meter (Jh/m³). One WLM is equivalent to 3.6×10^{-3} Jh/m³. An exposure to one WL for one working month (170 hours) equals 1 WLM cumulative exposure. That is, 1 WLM is 1WL for 170 hours.

One working level:

$$1WL = 1.3 \times 10^5 \text{ MeV/L} = 2.08 \times 10^{-5} \text{ Jm}^{-3}, \quad (1.1)$$

One working level month:

$$1WLM = 1WL \times 170h = 3.54 \times 10^{-3} \text{ Jh/m}^3, \quad (1.2)$$

Potential alpha energy is the total amount of alpha energy ultimately emitted during the decay of radon progeny chain upto the stable lead (^{206}Pb).

Radon dose D is the amount of energy deposition from radon and its progeny per unit exposure mass, for example per unit human body, per unit human lung. It measures average density of energy absorbed by the mass of the absorbing tissue. Its SI unit is J/Kg. The special name given for this unit is Gray (Gy). But in cgs system its unit is Rad.

$$100 \text{ Rad} = 1 \text{ J/Kg} = 1 \text{ Gy},$$

Dose equivalent H the product of the absorbed dose D by W_R called radiation weight factor. It is the quantity which measures both the amount energy absorbed by the target mass and also the biological effectiveness of the absorbed energy.

$$H = D \times W_R, \quad (1.3)$$

Its SI unit is Sievert (Sv).

$$1 \text{ Sv} = 100 \text{ Rem} = 1 \text{ J/Kg},$$

It is the quantity which measures the risk of that typical radiations. The risk on the tissue is also depends on the tissue type. ICRP introduces the idea of effective dose E.

$$E = H \times W_T, \quad (1.4)$$

where W_T , is called tissue weight factor, it is the sensitivity of the target tissue for the incoming radiation, different tissue has different sensitivity for radiations.

The total effect on human body can be calculated by summing the effective dose over all human organs.

$$E = \sum_T H \times W_T \times W_R, \quad (1.5)$$

Types of radiation	W_R	Tissue type	W_T
γ -rays and x-rays	1	Breast	0.05
β -partices	1	Bone Marrow	0.12
Thermal neutrons	5	lung	0.12
Fast neutrons	10	skin	0.01
proton	10	stomach	0.12
α -particles	20	liver	0.04

Table 1.2: Radiation and tissue weight factors according to ICRP 103

1.4 Radon Mitigations

Radon mitigation is any process used to reduce radon gas concentrations in the breathing zones of occupied buildings, homes, offices, schools, and water supplies. In confined spaces such as homes and office buildings, radon can accumulate to harmful levels. If the concentration of radon goes above the Action level attempts should be made to reduce it because of its adverse effect on human health. In this context, the Environmental Protection Agency (EPA) report that if a person is exposed to an indoor radon level of 4 pico- Curies per litre (4 pCi/l) or 148 Becquerel per cubic meter ($148 \text{ Bq}/\text{m}^3$), the probability of developing lung cancer is 13 - 50 persons per 1000. On the way, international commission for radiation protection (ICRP) recommends $200 \text{ Bq}/\text{m}^3$ as an action level. So one should take an action to reduce its concentration. Radon levels in room air can be lowered in a number of ways, from sealing cracks in floors and walls to changing the flow of air into the building [9].

The six principle ways of reducing the amount of radon entering a house are:

1. Improving the ventilation of the house;
2. Sealing of cracks and other openings on the walls;
3. Increasing under-floor ventilation;
4. Installing a radon pump system;
5. Opening of windows, doors and vents of the house (called natural ventilation)
6. House or room pressurization uses a fan to blow air into the basement

1.5 Health Effects of Radon

When radon decays after inhalation or ingestion, it releases energy that can damage DNA in the cells of sensitive organs like lungs and stomach and can cause cancer. Thus, naturally occurring radon in buildings has been identified as a human lung carcinogen and is considered to be the second leading cause of lung cancer after smoking tobacco [10].

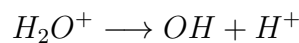
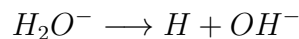
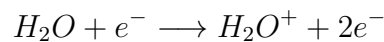
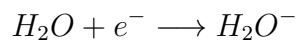
Radon (^{222}Rn), an inert radioactive gas, occurs naturally and exists everywhere in the atmosphere. When radon progeny are inhaled, a part of them is deposited onto the walls of the respiratory tract. Two of the radon decay products, ^{218}Po and ^{214}Po , emit alpha particles that impart their energies to lung tissues, which is associated with the risk of lung cancer [2].

Radon is an established human lung carcinogen based on experimental evidence of mutagenesis studies in cell culture and laboratory animals and epidemiologic cohort studies on uranium miners and case-control studies on the general public. The adverse health effects of exposure to radon are caused primarily by damage due to alpha-particles. The possible effects will depend on exposure level. The main danger from high radon exposure is an increased risk of lung cancer. Radon as a noble gas is rapidly exhaled after being breathed in; however, radon progeny, combine with other molecules in the air and with particles of dust, aerosols or smoke, and readily deposit in the airways of the lung. While lodged there, the progeny emit ionizing radiation in the form of alpha particles, which can damage the cells lining the airways [1]. Experiments have confirmed that ionizing radiation affecting bronchial epithelial cells could cause cancer. Lung cancer risk is much higher when radon exposure is combined with smoking. According to the BEIR IV report of the US national academies of sciences, for men exposed to radon at work, smokers were 10 times more likely to get lung cancer risk than non-smokers.

Radon has a short half-life (3.8 days) and decays into other solid particulate radium-series radioactive nuclides. Two of these decay products, polonium-218 and 214, present a significant radiologic hazard. If radon is inhaled, the decay products, whether attached to aerosol particles or unattached, will largely be deposited on the surface of the respiratory tract. If dust or aerosol (attached progeny of radon) is inhaled that already carries radon decay products, the deposition pattern of the decay products in the respiratory tract depends on the behavior of the particles in the lungs. Smaller diameter particles diffuse further into the respiratory system,

whereas the larger tens to hundreds of micron-sized particles often deposit higher in the airways and are cleared by the body's mucociliary staircase [10]. Deposited radioactive atoms or dust or aerosol particles continue to decay, causing continued exposure by emitting energetic alpha radiation with some associated gamma radiation too, that can damage vital molecules in lung cells, by either creating free radicals or causing DNA breaks or damage, perhaps causing mutations that sometimes turn cancerous [1]. In addition, through ingestion and blood transport, following crossing of the lung membrane by radon, radioactive progeny may also be transported to other parts of the body.

In human body, there is huge amount of water, radiations like alpha (or any other ionizing radiations) interact with water. The electron produced by ionization energy loss of radiation have interaction with water molecules.



All these reactions are possible, the result of the reaction (H_2O^+ , H_2O^- , H_2O_2) reacts with the body cells in our body. Especially H_2O_2 is more dangerous to body cells chemically which produces more damage to our body.

Using various modeling approaches, results of lung cancer risk in miners were used to project lung cancer risk for the general population exposed to residential radon. The results suggested that (10 - 15)% of the total lung cancer deaths in the US could be attributed to radon exposure in homes, making radon the second leading cause of lung cancer death after tobacco smoke.

Deposition Mechanisms in Human Respiratory Tract

2.1 Human Respiratory System

The respiratory tract is the part of the anatomy of the respiratory system involved with the process of respiration. In order to understand the deposition of aerosols in the respiratory tract, it is useful to understand aspects of human respiratory system. The human respiratory anatomy can be divided into three compartments or regions [11]. The Figure 2.1 below shows the three regions of the respiratory tract.

2.1.1 Extrathoracic (ET) Regions

The extrathoracic airways are divided into two compartments, ET_1 which includes the anterior nose, and ET_2 which includes the posterior nasal passages, larynx, pharynx, and mouth. This site is also referred to as the head and neck airway region. The main function of the nasal passages is olfaction and heating, humidifying and filtering of the inhaled air. This region of the human respiratory tract serves as an important first stage filter for inhaled particles entering the lung [12].

2.1.2 Trachea-bronchial (TB) Region

It includes the trachea (called generation zero) which bifurcates at the carina, behind the sternum into the two main bronchi of which each leads to a lung. In the lung the bronchial (BB) (consists from generation 0-8) divides further into a sequence of smaller and smaller airway bronchi called bronchiole (bb) (which corresponds from generation 9-16) which have a diameter of about 0.5 mm. The first approximately 16 bifurcations (generations) belong to the Trachea-bronchial region. The epithelium of the trachea primarily consists of ciliated cells ending with mucus secreting goblet cells and specialized mucus secreting glands [12]. The

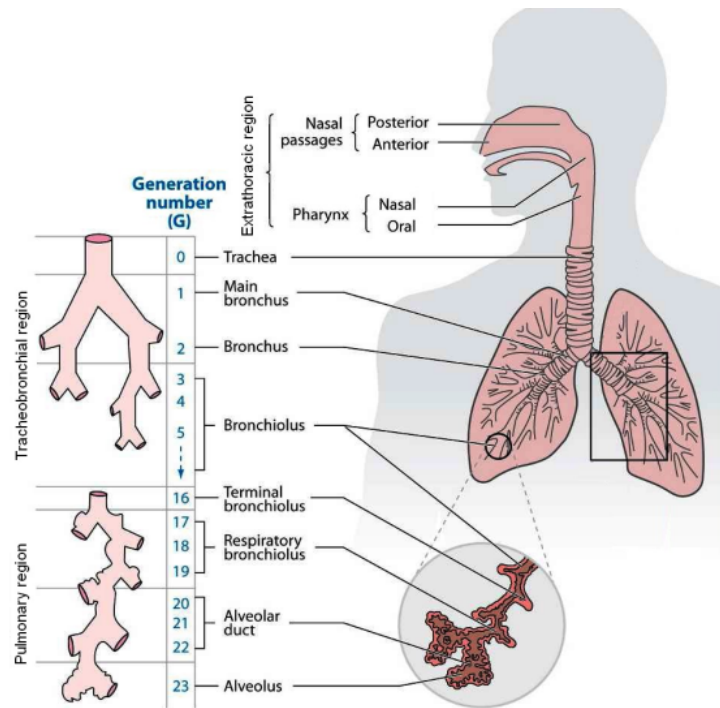


Figure 2.1: Parts of human respiratory system

cilia reveal synchronized beating patterns in order to propel mucus and deposited matter up to the larynx where they are either expelled or ingested. The main function of this region is to further humidify and filter air and conduct it to the gas exchange region.

2.1.3 Alveolar Interstitial (*AI*) Region

It is called gas-exchange region (entrance of oxygen into the body and the exit of carbon dioxide) includes the terminal of bronchioles and the alveolar ducts and sacs. The alveolar are surrounded by millions of pulmonary capillaries. Venous blood, which is pumped through these capillaries, takes up oxygen and expels CO_2 in the gas exchange region due to diffusion gradients between the blood and the inspired air under the relaxation and contraction of diaphragm [12].

Generally, the radon progeny can be inhaled through nose or mouth, pass through pharynx and larynx to trachea zero generation (has no airways), and then enters to the bronchial regions which consist from (0- 17 generation) and passes into gas exchange area (alveolar regions) which starts from generation 17 up to generation 23.

2.2 Assumptions of ICRP Model

It is based on dividing the human respiratory tract into compartments. During the past 4 decades, the International Commission on Radiological Protection, ICRP, has published three different mathematical models to describe the deposition, clearance, and dosimetry of inhaled radioactive materials in the respiratory tract. The models make it possible to calculate the absorbed doses expected to be received by different parts of the respiratory tract and describe mathematically the expected absorption and translocation of portions of the deposited radionuclides to other organs and tissues beyond the respiratory tract. Due to it is based on dividing the respiratory tract into regions, this model is called a deterministic regional compartment model [13].

Before dealing the mathematical descriptions of particle deposition and clearance and the associated dosimetry, it is important to note that the models have changed in structure, expanding from two airway regions in the 1959 model to five airway regions in the 1994 models.

The 1959 model had a very simple structure in which the respiratory tract was divided into an upper respiratory tract, URT, and a lower respiratory tract, LRT. No specific anatomical sites were assigned to the URT and LRT.

In the 1979 model, the respiratory tract was divided into the three airway regions, Extrathoracic (ET); tracheobronchial, T-B; and the alveolar intestinal (AI) regions. The ET region extended from the anterior nose to the larynx, and the T-B region included the trachea and bronchial tree through the terminal bronchioles. The AI region was the remaining, nonciliated gas-exchange region.

The newest model, the 1994 version, the respiratory tract is divided into five airway regions as shown in figure 2.2. The extrathoracic airways are divided into two compartments [14] ET1, the anterior nose, and ET2, the posterior nasal passages, larynx, pharynx, and mouth. The bronchial region, (BB, consists of the trachea, generation 0 and bronchi, airway generations 1-8) and the bronchiolar region, (bb: airway generations 9-15), consists of the bronchioles and terminal bronchioles. The last region, the alveolar-interstitial region AI, where gas exchange is performed, consists of the respiratory bronchioles, the alveolar ducts, alveoli, and the interstitial connective tissue.

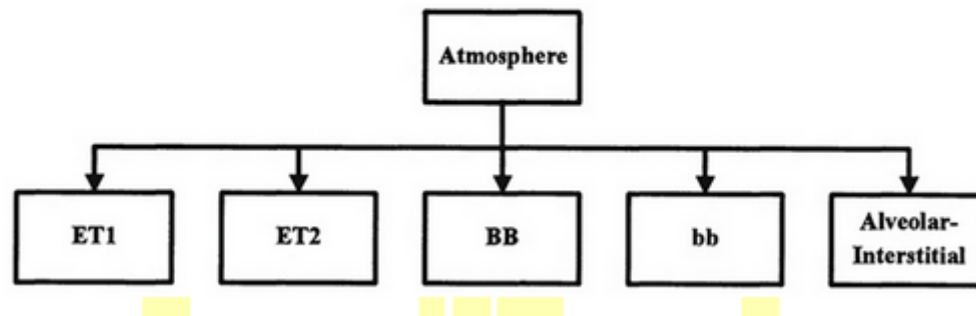


Figure 2.2: The five regions of respiratory tract

2.3 Morphometry of Human Lung

In the HRTM the respiratory tract is represented by five regions, based on differences in radio-sensitivity, deposition and clearance. The extrathoracic (head and neck) airways (ET) are divided into ET1, the anterior nasal passage, and ET2, which consists of the posterior nasal and oral passages, the pharynx and larynx. The thoracic regions of the lungs are Bronchial (BB), Bronchiolar (bb), and Alveolar-Interstitial (AI, the gas exchange region).

The bronchial region of the human lung consists of a sequence of bifurcating tubes, which decrease in diameter and length as they penetrate deeper into the lung, until the terminal bronchioles are reached. All airways beyond the terminal bronchioles are surrounded by alveoli. In the ICRP respiratory tract model, the tracheobronchial airway system is divided into only two anatomical compartments: a central bronchial (BB) and a more peripheral bronchiolar (bb) region [13].

2.4 Deposition of Radon Progeny

Aerosol deposition in the human respiratory tract is determined by biological factors such as lung morphology, breathing rate, and breathing frequency and physical factors such as fluid dynamics, particle properties, and deposition mechanisms.

If radon is inhaled, the decay products, whether attached to aerosol particles or unattached, will largely be deposited on the surface of the respiratory tract. A fraction of them is deposited in the lung, depending on the size, density, shape, charge, and surface properties of the particles of the aerosols and the breathing pattern of the individual. Aerosol deposition in the compartments of the HRTM is

determined by using semi-empirical equations, which are essentially based on the experimental deposition data [11].

The deposition model evaluates the fraction of activity in the inhaled air that is deposited in each region. Deposition in the ET regions was determined mainly from experimental data [12]. For the lungs, a theoretical model was used to calculate particle deposition in each region, and to quantify the effects of the subjects lung size and breathing rate. The deposition of an inhaled radon progeny is performed through three mechanisms as shown in figure 2.3 below. The first is inertial impaction, in which a droplet fails to turn a corner and impacts the wall of the airway. The second is sedimentation, in which the droplets or particles rain out under the influence of gravity. Finally, there is diffusion caused by Brownian motion, which results in eventual collisions of the droplets with the airway wall. Generally we classified them into two groups of deposition processes, i.e Thermodynamic and Aerodynamic deposition.

2.4.1 Thermodynamic Deposition

Thermodynamic deposition is a method of deposition for particles less than $1\mu\text{m}$ diameter in size. Those particles are slowly deposited due to diffusion and they are often called diffusion or Brownian deposition. Since particles are so smaller of order $1\mu\text{m}$, such Brownian diffusion is performed in lower regions (alveolar region) of the respiratory tract as they are smaller they can reach up to alveolar regions [15].

Deposition by diffusion is caused by Brownian motion or a larger diffusion coefficient. Diffusion is the deposition mechanism for small particles ($< 0.5 \mu\text{m}$) due to collision with air molecules. Diffusion may cause the particle to move across the streamline and deposit upon contact with the airway wall. Deposition by diffusion increases with decreasing particle size and flow rate. Increased diffusion deposition occurs in the alveoli region because of longer residence time and smaller airways.

This type of deposition is caused by the random movement of the radon progeny in the air tract. The distance of the particle traveled by diffusional transport increases with decreasing particle size and increasing breathing rate of the individual. During their diffusion process, when they touch the walls of the airway tube, they will deposit there. So generally the deposition of particles by diffusion decreases with increasing of particle size and becomes negligible for larger particles.

2.4.2 Aerodynamic Deposition

Aerodynamic deposition is more important for larger particles and there are two types of processes belonging to this group. These are

1. impaction or inertial deposition
2. gravitational sedimentation

Impaction or inertial deposition, this deposition process takes place around bent when the air stream changes the direction, some of the airborne progeny with larger mass cannot adjust their directions of movement sufficiently quickly because of their inertia, and impact onto the wall of the airway tube. In the upper air ways (extrathoracic region) the air-speed is high enough to cause particles to deposit by impaction. Deposition through inertial impactation is more effective in upper (extrathoracic) regions.

Particles with enough momentum (product of the mass and velocity) are affected by centrifugal force at the points where the airflow suddenly changes direction, colliding with the airway wall. This mainly happens in the first 10 bronchial generations, where the air speed is high and the flow is turbulent. This phenomenon mainly affects particles larger than 10 micrometer, which are mostly retained in the extrathoracic region [12].

The second aerodynamic deposition process is gravitational sedimentation of particles. The motion of larger particles in the respiratory tract is influenced by gravity. Sedimentation, or settling under the force of gravity, is an important deposition mechanism for particle in the respiratory tract. Gravitational sedimentation because of it gravity dependent so that alveoli at the base of the lung are relatively compressed compared alveoli are more compliant ventilation is greatest near to the bottom of the lung and became progressively reduced near the top.

Sedimentation deposition becomes dominant in the bronchiolar and alveolar regions where air flow decelerates and it increases with an increase in particle size and a decrease in flow rate.

Sedimentation represents deposition under the action of gravity. During sedimentation, a particle acquires its terminal settling velocity v when gravitational forces are balanced by viscous resistive forces of the gas. The terminal settling velocity for spherical particles, v is then,

$$v = \frac{\rho d^2 g c(d)}{18\eta}$$

where, where g is the gravitational constant, d is particle diameter, ρ is particle density, η is viscosity and $c(d)$ is Cunningham slip correction factor.

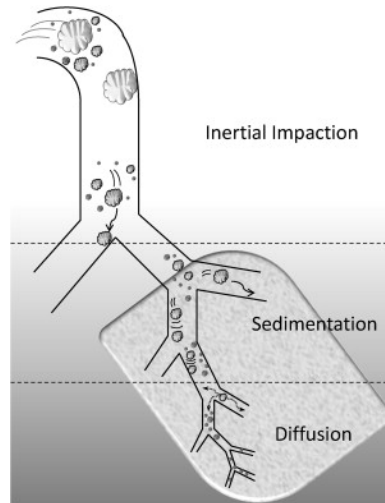


Figure 2.3: Aerosol deposition mechanisms in the respiratory system

2.5 Total Deposition Patterns in Human Airways

The shape of deposition patterns strongly depends on the inhaled particle sizes, breathing pattern and the lung airway geometry. Generally speaking, lung deposition is a superposition of two separate deposition patterns - sedimentation and impaction for larger particles and diffusion for nano-sized particles [16]. The deposition of submicron particle increases with particle diameter, as the deposition through sedimentation and impactation increase with diameter. The deposition of nano-sized particles decreased with increasing particle size. The total deposition reaches a minimal deposition efficiency at the intersection between the two patterns. With further decrease in particle size into the nanometer scale, deposition picks up again as one proceeds towards the alveolar domain [12].

The deposited fraction is given as a function of particle diameter. Minimal deposition occurs for particles in the size range between $0.1 \mu\text{m}$ and $1 \mu\text{m}$, but it increases equally as well with increasing as with decreasing particle diameter and reaches almost 100% for $10 \mu\text{m}$ or 0.001 mm particles. Considering the deposition mechanisms already described, the dependence of total deposition on particle diameter becomes clear. For particles in the size range of $0.1 \mu\text{m}$ to $1 \mu\text{m}$, particle displacement

related to either of the three deposition mechanisms is minimal. With increasing particle size, displacement by sedimentation and impaction increasingly rises so that total deposition is enhanced. For particles less than $0.1 \mu\text{m}$, diffusional deposition increases and therefore causes deposition to increase [17].

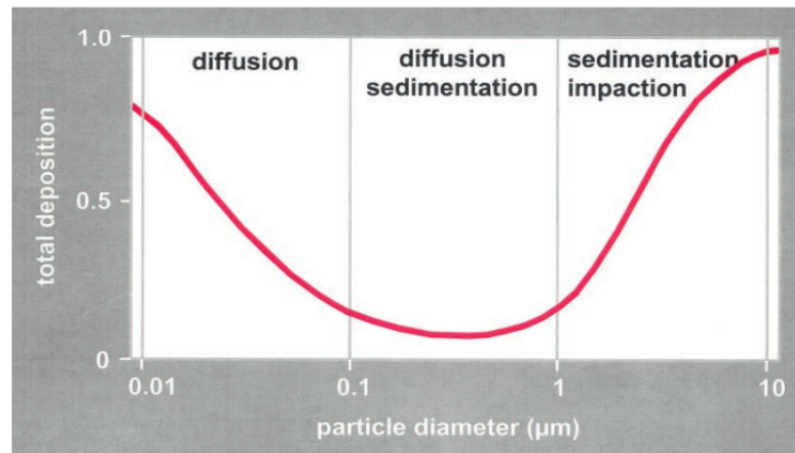


Figure 2.4: Total deposition pattern of inhaled particles in the respiratory tract

2.6 Factors Affecting the Deposition of Radon Products

There are many conditions or parameters that we need to consider in model developing. Even if the inspired concentration of radon progeny were similar, the dose deposited in the lungs may vary. Factors such as metabolic rate, breathing pattern, and lung structure determine the deposition of radon progeny and may differ among individuals (children versus adults).

Aerosol size is one factor which determines the regional deposition of radon progeny in the respiratory tract. A major factor governing the effectiveness of the regional deposition mechanisms is the size of the inspired particles. The effective aerodynamic diameter is a function of the size, shape, and density of the particles and affects the magnitude of forces acting on them. For example, inertial and gravitational effects increase with increasing particle size, whereas the displacements produced by diffusion decrease. The importance of particle size cannot be overemphasized. It is featured prominently in most discussions of aerosol deposition in the respiratory tract. A particle's velocity in response to gravitational or inertial forces increases as the square root of its density. The breathing condition of a person is also another factor. The breathing condition for a reference worker at light work defined as an

adult, nasal breathing male, with 31.25 % sitting and 68.75 % light exercise, which yield average breathing rate (volume of air per given time) of $0.78 \text{ m}^3/\text{h}$ [14].

Tidal volume, target cell depth, thickness of target cell layer and breathing frequency are also another parameters that we need to consider. Tidal volume is the volume of air that can be inhaled or exhaled only in a single breath. larger tidal volumes result in higher particle deposition in the human lung as particle-laden air penetrates deeper into the lung. Tidal volume of air varies from person to person, it depends on gender and age. Breathing frequency is the number breaths you takes in a minute. Functional residual capacity is the volume of air left in the lungs after the end of normal tidal volume expiration. Lower flow rates also result in higher particle deposition in the peripheral lung as velocities are slower and particles have more time to deposit by sedimentation or diffusion.

The density of the unattached progeny was taken as $1 \text{ g}/\text{cm}^3$, while that of the attached progeny as $1.4 \text{ g}/\text{cm}^3$. The half-life for transfer to blood (clearance rate) is taken as 600 min. Hygroscopic growth factor is also another factor. Hygroscopicity of the the aerosols is the property of the particle due to warm humidity in the respiratory tract, absorbs water and grows in size in the respiratory tract. Since they are hygroscopic and rapidly change their particle size in the lung, they are delivered at high mass concentrations. Humidity of the environment also determines the attachment of radon progeny with aerosols.

2.7 Deposition Calculation

Deposition of aerosols can be takes place both during inhalation and exhalation of radon through aerodynamics and thermodynamics deposition as shown in figure 2.5. The total deposition on each regions of the lung is the sum of the deposition during inhalation and exhalation.

Deposition of aerosols depends on the transit time of inhalation and exhalation. Transit time is different from region to region. Transit time in bronchial (BB) is given by:

$$t_B = \frac{V_D(BB)}{V} \left[1 + \frac{0.5V_T}{FRC} \right], \quad (2.1)$$

Transit time in bronchiolar (bb) is given by:

$$t_b = \frac{V_D(bb)}{V} \left[1 + \frac{0.5V_T}{FRC} \right], \quad (2.2)$$

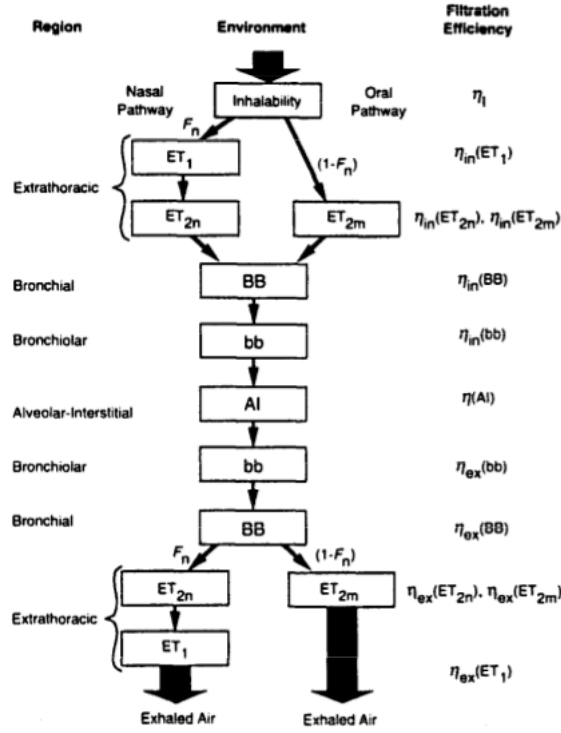


Figure 2.5: Aerosol deposition during inhalation and exhalation

Transit time in Alveolar interstitial (AI) is given by:

$$t_{AI} = \frac{V_T - V_D(ET) - \left(V_D(BB) + V_D(bb) \right) \left(1 + \frac{0.5V_T}{FRC} \right)}{V}, \quad (2.3)$$

where: V_T is tidal volume, $V_D(bb)$ is dead space in bb, $V_D(BB)$ is dead space in BB, FRC is functional residual capacity and V is total volumetric flow rate. Dead space is the volume in the respiratory tract where the air inside it does not participate in gas exchange in alveoli.

Thermodynamic deposition efficiency of each region of respiratory tract is given by:

$$\eta_{th} = 1 - e^{-aRp}, \quad (2.4)$$

and deposition efficiency of each region resulting from impactation and gravitational settling of aerodynamic aerosols is given by:

$$\eta_{ae} = 1 - e^{-aRp}, \quad (2.5)$$

where a , R and p are the three parameters, which has different value during inhalation and exhalation in different regions of the lung as tabulated in table 3.2. R is aerosol diameter, volumetric flow rate, transit time and airway diameter

of lung dependent. Thermodynamic diameter d_{th} and aerodynamic diameter d_{ae} of aerosols can be expressed as shown in equation 2.6 and 2.7 respectively.

$$d_{th} = \frac{KTC(d)}{3\pi\mu D}, \quad (2.6)$$

where k is boltzmann constant, T is temprature, D is diffusion cofficent, $C(d)$ is Cuningham correction and μ is air viscosity.

$$d_{ae} = d_e \sqrt{\frac{\rho C(d_e)}{\chi \rho_o C(d_{ae})}}, \quad (2.7)$$

where d_e is particle diameter, $C(d_e)$ is Cuningham correction can be expressed interms of mean free path and particle size,

$$C(d_e) = 1 + \frac{\lambda}{d_e} [2.514 + 0.8e^{-0.55\frac{d_e}{\lambda}}], \quad (2.8)$$

Where ρ is density and χ is shape factor, and finally the deposition efficiency of each filter or region of the respiratory tract in one stage (either inhalation or ehalation phase) is represented by two components, η_{ae} which arises from the aerodynamic deposition processes of impaction and gravitational settling, and η_{th} which arises from the thermodynamic process of particle diffusion by Brownian motion. The total deposition efficiency of each region will be :

$$\eta = (\eta_{ae}^2 + \eta_{th}^2)^{1/2}. \quad (2.9)$$

To find the total deposition on each lung regions, one need to find the volumetric fraction of the region from the amount of activity inhaled per one breath. Volumetric fraction is a fraction of the inhaled air (tidal volume) which passes through each regions. Arrive and deposition is different. The volumetric fraction of each region is given from equation 2.10 to 2.12.

$$V_{BB} = 1 - \frac{V_D(ET)}{V_T}, \quad (2.10)$$

$$V_{bb} = 1 - \frac{V_D(ET) + V'_D(BB)}{V_T}, \quad (2.11)$$

$$V_{AI} = 1 - \frac{V_D(ET) + V'_D(BB) + V'_D(bb)}{V_T}, \quad (2.12)$$

where,

$$V'_D(BB) = V_D(BB) \left(1 + \frac{V_T}{FRC}\right), \quad (2.13)$$

$$V'_D(bb) = V_D(bb) \left(1 + \frac{V_T}{FRC}\right), \quad (2.14)$$

Total deposition on each region during inhalation and exhalation can be found by multiplying deposition fraction, volumetric fraction and amount of inhaled activity separately. Finally the total deposition on the region is the sum of deposition due to inhalation and exhalation.

The deposition fraction D_{frac} along the five regions of the respiratory tract will be the ratio of total deposition on each region to the total volume of activity inhaled,

$$D_{frac}(i) = \frac{D_{inh}(i) + D_{exa}(i)}{A_{inh}}, \quad (2.15)$$

The percent in deposition along each region from the intake activity can be expressed as

$$D_{frac}(i) = \frac{D_{inh}(i) + D_{exa}(i)}{A_{inh}} \times 100\%, \quad (2.16)$$

where $D_{inh}(i)$ is deposition of aerosols on region i of respiratory tract during inhalation phase, $D_{exa}(i)$ is deposition of aerosols on region i during exhalation phase and A_{inh} is the total inhaled activity.

Simulation Method

3.1 Computer Code

For the estimation of regional deposition of attached fraction of radon progeny, this paper uses a fortran code which runs according to the assumptions of ICRP model. The computer codes are thus designed to simplify the complicated numerical analysis of deposition fraction in bronchial (BB), bronchiolar (bb), alveolar interstitial (AI) and extrathoracic (ET) regions of the respiratory tract. Regional deposition of attached fraction of radon depends on biological, physical and enviromental factors.

Biological factors such as,

- breathing rate
- breathing frequency
- functional residual capacity
- tidal volume
- volumetric flow rate
- airway dimension
- anatomic dead space

Physical factors such as,

- size of aerosol
- density of aerosol
- shape factor of aerosol.

Recommended parameters	Values according to ICRP
Tidal volume(V_T)	866.66 mL/ breath
Breathing frequency(F_R)	15/min
Breathing rate (V)	0.78 m ³ /h
Functional residual capacity(FRC)	3300 mL
Density(ρ)	3 g/cm ³
Shape factor(χ)	1.5
Temperature(T)	37 ⁰ c
Viscosity of air(η)	1.835 × 10 ⁻⁵ g/cm.s

Table 3.1: The values of input parameters for the code

All those exposure conditions should be given to the computer code as input parameters. The values of the input parameters are described by ICRP committee for a reference adult nose breather. These values are tabulated in table 3.1.

The anatomic dead space of the regions of the lung affects the regional deposition efficiency of the inhaled activity. Dead space is the volume of inhaled air in each region of the lung that does not participate in the gas exchange process. The dead space in extrathoracic (ET), bronchi (BB), and bronchiolar(bb) region is 50 ml, 49 ml and 47 ml respectively. The diameter of trachea (d_0), bronchi (d_9) and terminal bronchiole (d_{16}) is also another factor. The regional deposition of aerosols is also depends upon the diameter of airways within the respiratory tract. The aerodynamic and thermodynamic deposition of particulate material within ET1, ET2, BB, bb and AI region depends on d_0 , d_9 and d_{16} with a value according to ICRP 1.65 cm, 0.165 cm and 0.051 cm respectively. It is the average airway diameter of various airway generations in each region.

Using mathematical formulas of volumetric fraction, transit time and deposition efficiency which are applied along each region and the input parameters given in table 3.1, the code calculates the regional deposition fraction of attached fraction of radon aerosols as a function of particle size. Generally,

- The code takes the biological and physical exposure conditions which affect the regional deposition as an input parameters.
- To investigate the relation between regional deposition and aerodynamic diameter of aerosols, the fortran code uses a do loop statement to iterate the equivalent aerodynamic diameter.

- Under each iteration the code calculates the activity deposited on each region by multiplying deposition efficiency with volumetric fraction of the region.
- Finally, the ratio of activity deposited on each region to the total activity inhaled will be the deposition fraction along each regions.

3.2 Regional Deposition Calculation Method

Humans can be exposed for both internal and external radiations. To determine the health effects from external radiations different instrumental measurements can be applied. But since the dose from internal radiation exposure is difficult to measure directly, different lung models were developed. For the estimation of the dose delivered by alpha particles in the lung, this paper uses the assumptions of ICRP model. The dose on the lung is the result of the regional deposition of radon progeny in the respiratory tract. The fortran code which implements the assumptions of ICRP is applied to calculate the regional deposition fraction of attached fraction of radon aerosols.

The concentration level of radon is measured in nuclear physics laboratory. The results of the experiment shows that the highest activity of radon concentration was recorded 27.53 Bq/m^3 and minimum value of radon concentration was recorded 5.59 Bq/m^3 with a mean value of 14.2 Bq/m^3 . The concentration of radon were measured for 21 consecutive days by closing the room and windows over the night and the results of the measurement were found below the action level that WHO (WHO2008) recommended (100 Bq/m^3). So it was found riskless. Since 14.2 Bq/m^3 amount of radon gas is diluted with air, the total amount of concentration of radon that will be inhaled in a single breath will be mean value of radon concentration times tidal volume of the reference person.

$$AINHALED = Conc_{Rn} \times V_T.$$

Regional deposition fraction of attached fraction of radon on each region depends on deposition efficiency of each regions. Deposition efficiencies of each region is defined as, from the given amount of inhalation how much of them will be filtered there before passing to the next regions of the respiratory tract. The deposition of large size aerosols (attached fraction of radon) is expressed in terms of aerodynamic deposition through gravitational settling and inertial impaction. As a result

the deposition efficiency of each region can be found as following, where i in each equation represents each regions of the respiratory tract.

The deposition efficiency of extrathoracic ET1 region is given by,

$$\eta_{ET1} = 0.5\left(1 - \frac{1}{1 + a_i R_i^{p_i}}\right).$$

The deposition efficiency of extrathoracic ET2 region is given by,

$$\eta_{ET2} = 1 - \frac{1}{1 + a_i R_i^{p_i}}.$$

The deposition efficiency in the bronchi, bronchiolar and alveolar region is given by,

$$\eta = 1 - e^{-a_i R_i^{p_i}}.$$

Following the inhalation of a volume of aerosols, deposition of aerosols takes place in different regions of the respiratory tract which depends on the deposition efficiencies of each region. The deposition efficiencies of each region is expressed in equation above interms of three parameters a , p and R . The value of a , p and R varies from region to region also varies during inhalation and exhalation and there values are given in table 3.2 above. R is breathing rate, transit time, airway diameter and diameter of aerosol dependent.

Although deposition along each region can takes place during inhalation phase and exhalation phase, the code calculates the deposition during inhalation phase first and then it calculates the deposition during exhalation phase after the gas exchange takes place in alveolar regions.

The expression for parameter a , p and R for aerodynamic deposition as a result of large size particles through inertial and sedimentation deposition of aerosols is given below.

The deposition efficiencies of each region depends explicitly on the average transit time of inhaled and exhaled air through each region. The transit times are denoted by t_B , t_b , and t_{AI} , for the BB, bb, and AI regions, respectively. As transit time between inhaled and exhaled air increased the deposition of aerosols along each region also increased. Transit time depends on dead space, tidal volume, functional residual capacity and breathing rate. Its value in bronchiolar, bronchi and alveolar regions are given in equation 2.1 to equation 2.3.

Filter	Region	a	p	R
1	ET1	3×10^{-4}	1	$d_{ae}^2 V S F_t^3$
2	ET2	5.5×10^{-5}	1.17	$d_{ae}^2 V S F_t^3$
3	BB	4.08×10^{-6}	1.152	$d_{ae}^2 V S F_t^{2.3}$
4	bb	4.08×10^{-6}	1.173	$(0.056 + t_b^{1.5}) \times d_{ae} t_b^{-0.25}$
5	AI	$0.146 \times S F_A^{0.98}$	0.6495	$d_{ae}^2 t_{AI}$

Table 3.2: Recommended algebraic expressions for aerodynamic deposition

So as to find the deposition fraction along each region the volumetric fraction of each region must be known. Volumetric fraction is the fraction of the total inhaled volume that will reach on each regions of the lung in a single breath. It depends on tidal volume, functional residence capacity and dead space of the region. Dead space is a little portion or volume of the inspired volume of air which can hold some volume of inhaled air. The air in dead space is not participate in gas exchange. The amount of dead space increases the deposition process. The volumetric fraction of each region is given from equation 3.1 to equation 3.3 . It is noted that the volumetric fraction in extrathoracic region, in ET1 and ET2 is considered nearly one.

$$\phi_{BB} = 1 - \frac{V_D(ET)}{V_T}, \quad (3.1)$$

$$\phi_{bb} = 1 - \frac{V_D(ET) + V'_D(BB)}{V_T}, \quad (3.2)$$

$$\phi_{AI} = 1 - \frac{V_D(ET) + V'_D(BB) + V'_D(bb)}{V_T}, \quad (3.3)$$

where,

$$V'_D(BB) = V_D(BB) \left(1 + \frac{V_T}{FRC}\right),$$

$$V'_D(bb) = V_D(bb) \left(1 + \frac{V_T}{FRC}\right),$$

Once the deposition efficiencies η_i and volumetric fraction ϕ_i of region i of the respiratory tract is known, the deposition fraction D_i of region i can be the product of the two.

$$D_i = \phi_i \times \eta_i.$$

The computer code uses a do loop that allows variation of diameter of attached fraction aerosols from $1\mu\text{m}$ to $10\mu\text{m}$, and at each diameter the code calculates the deposition efficiencies and percentage of regional deposition.

The activity deposited in extrathoracic (ET1) region during inhalation from the total amount of activity inhaled is given as

$$D_{inh}(ET1) = \eta_{ET1} \times AINHALED,$$

The activity after ET1 or brought to ET2 should be total volume of activity that are inhaled AINHALED minus the activity deposited in ET1.

$$AKT = AINHALED - D_{inh}(ET1).$$

All the activity that brought to ET2 will not deposit on ET2. The activity deposited on ET2 is the product of deposition efficiencies of ET2 and volumetric fraction that are brought to ET2.

$$D_{inh}(ET2) = \eta_{ET2} \times AKT.$$

The activity after ET2 or brought to BB should be total volume of activity that are inhaled AINHALED minus the activity deposited in ET2 and ET1.

$$AKT = AINHALED - D_{inh}(ET1) - D_{inh}(ET2).$$

The activity deposited in BB is

$$D_{inh}(BB) = \eta_{BB} \times AKT \times \phi_{BB}.$$

The activity after BB or brought to bb should be total volume of activity that are inhaled AINHALED minus the activity deposited in BB, ET2 and ET1. The activity that are deposited in bb is the product of the deposition efficiency of bb, volumetric fraction of bb and the volume of activity that are brought to bb.

Similarity produces is applied to find the deposition on alveolar interstitial (AI) regions. Once the inhaled air reaches the alveolar regions the gas exchange takes place. The oxygenated blood is transported to parts of the body through pulmonary arteries and the deoxygenated gas will be exhaled. During the exhalation phase of deoxygenated gas from alveolar region to the external environment, the deposition of aerosols can take place along each region of the respiratory tract.

The activity after AI back to bronchiolar (bb) region during exhalation is the difference between the activity deposited on each region to the total inhaled air.

$$AKT = AINHALED - D_{inh}(ET1) - D_{inh}(ET2) - D_{inh}(BB) - D_{inh}(bb) - D_{inh}(AI).$$

And hence the amount of activity deposited in bronchiolar (bb) region during exhalation phase can be expressed as,

$$D_{exh}(bb) = \eta_{bb} \times AKT \times \phi_{bb}.$$

The code computes the deposition along each regions during exhalation phase. Finally the total deposition on each region is the sum of the deposition fraction during inhalation phase plus deposition fraction during exhalation phase. The total deposition fraction in the ET1 region can be expressed as,

$$D_{ET1} = \frac{D_{inh}(ET1) + D_{exh}(ET1)}{AINHALED}. \quad (3.4)$$

The total deposition fraction in the ET2 should be,

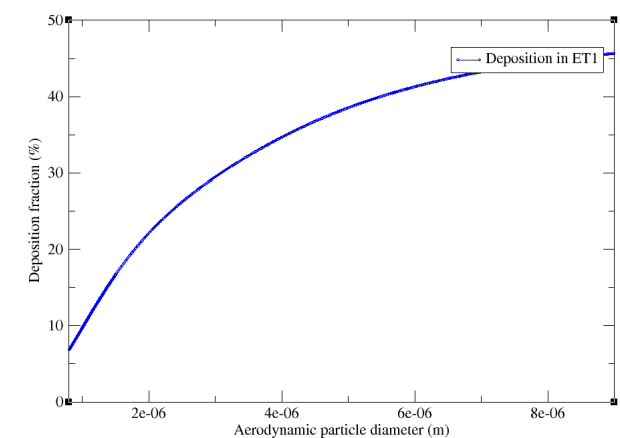
$$D_{ET2} = \frac{D_{inh}(ET2) + D_{exh}(ET2)}{AINHALED}. \quad (3.5)$$

In similar way the total deposition fraction of the other regions of the respiratory tract can be determined. Deposition fraction is the ratio of deposition on each region to the volume of activity that are inhaled as shown in equation 2.15 and 2.16. The deposition fraction on each region indicates how much fraction of the inhaled activity will deposited in that given region.

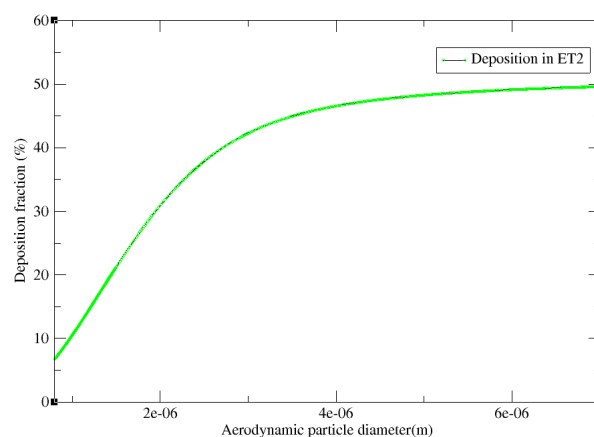
Result And Discussion

4.1 Results

As shown in the graph 4.1, as diameter of aerosols increases from $1 \mu\text{m}$ to $9.99 \mu\text{m}$, the percentage deposition fraction in extrathoracic regions, in ET1 region increases from 6.53% to 48.43% and in ET2 region increases from 7.3% to 50.33%.



(a)



(b)

Figure 4.1: Deposition of attached fraction of radon progeny in extrathoracic region

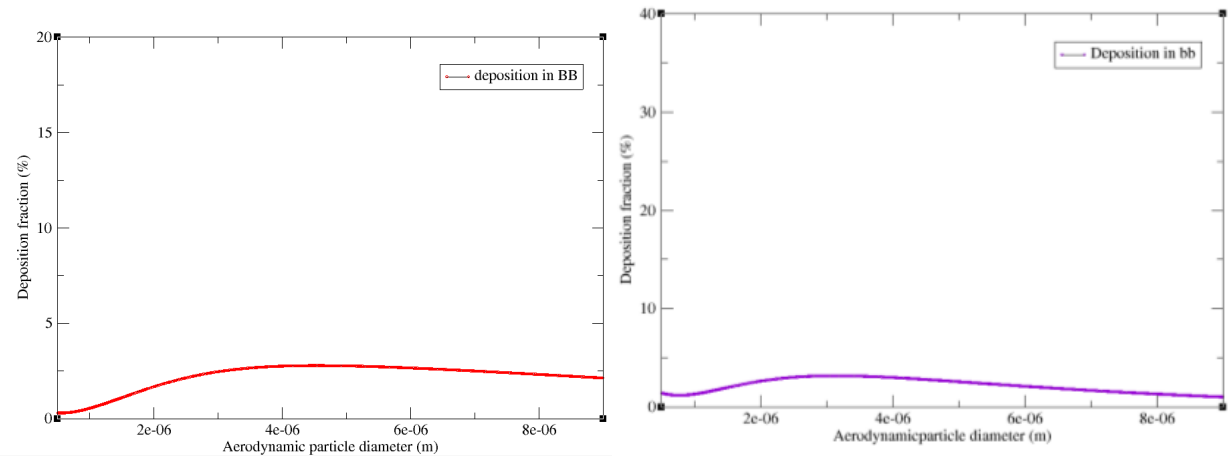


Figure 4.2: Deposition of attached fraction of radon progeny in bronchi and bronchiolar region

The aerodynamic deposition of attached fraction of radon aerosols along the bronchial regions (bronchi (BB), and bronchiolar (bb) region) is found small and almost constant as shown in graph 4.2. For 1 μm diameter aerosols the percentage deposition is found 0.82%, around 5 μm , 2.56% and at 9 μm the deposition is predicted about 1.93% in bronchi (BB) region. In bronchiolar region (bb) for 1 μm aerosols the deposition predicted is 1.5 % and at 9 μm about 0.88% is predicted.

As shown in the graph 4.3 the deposition of attached fraction of radon aerosols in alveolar region decreased as the size of aerosols increased. The deposition of small size attached fraction of radon aerosols is found maximum in this region as compared to other regions of the respiratory tract.

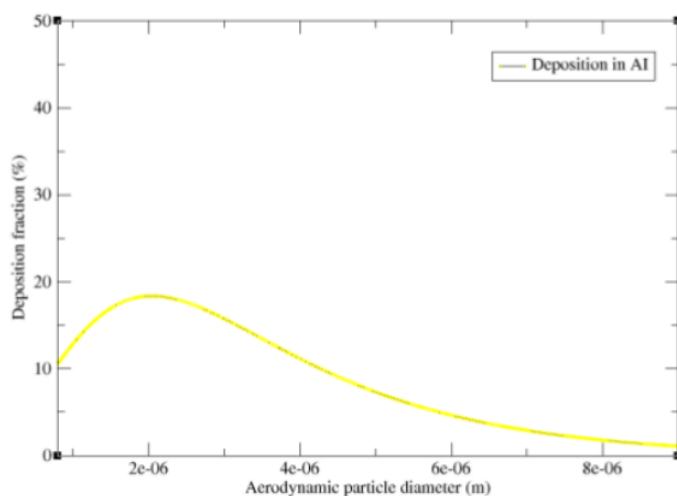


Figure 4.3: Deposition of attached fraction of radon progeny in alveolar region as function of aerodynamic diameter

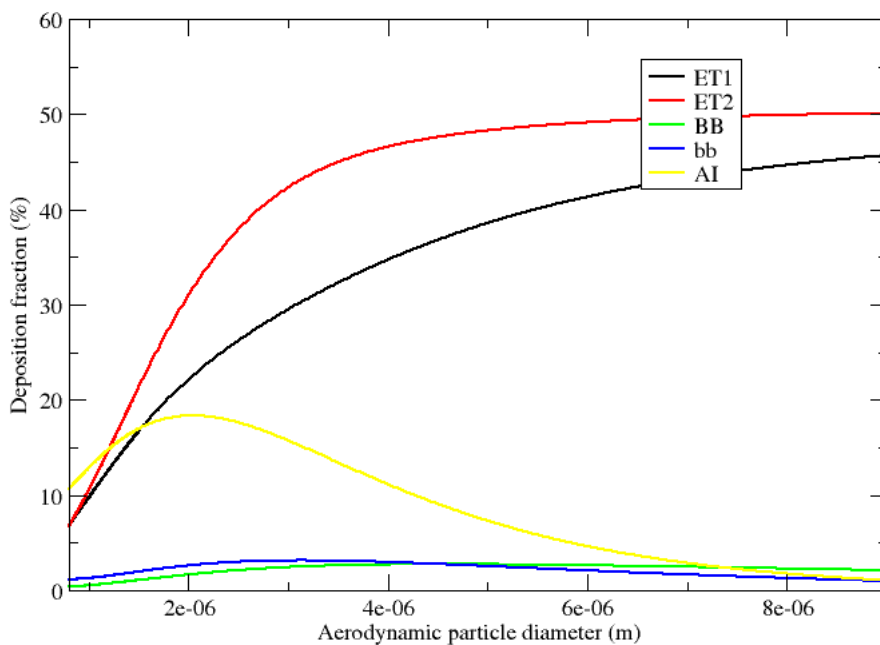


Figure 4.4: Deposition on each regions of the respiratory tract

4.2 Discussion

The attached fraction of radon is primarily deposited through aerodynamic deposition mechanism. The deposition in the upper regions (extrathoracic region) of the respiratory tract is performed through inertial impaction and the deposition in lower regions is using gravitational settling (sedimentation) mechanisms.

As the aerodynamic diameter of the attached fraction of radon aerosols increases as shown in the graph 4.1, the deposition in extrathoracic (ET) region becomes larger and larger. This is due to that during the inhalation, the air speed in the extrathoracic region is high which implies the inertia of attached fractions with larger mass becomes high (product of high mass and also high speed). Moreover, since during inhalation, the airflow undergoes several direction changes in the extrathoracic region, it is difficult for the particles to change directions around the curvatures of the air streams in the extrathoracic regions. Many large particles do not turn with airstream, but prefers to travel in the initial directions and hence they will impact or stick to the surfaces of the turnings and deposited there through inertial impaction.

As diameter of aerosols increases from $1 \mu\text{m}$ to $9.99 \mu\text{m}$, the deposition fraction in extrathoracic regions, in ET1 region increases from 6.53% to 48.43% and in ET2 region increases from 7.3% to 50.33%. This is due to the fact that as the diameter of the particles increases, their size also increases, in addition to this the speed of the inhaled particles in extrathoracic region is relatively high which implies the particles momentum increases. As a result the particles will be deposited through inertial impaction. For any further increase the particle size, it is observed that the deposition in extrathoracic region will dominant in contrast the deposition in other regions of the respiratory tract becomes negligible.

In fact the nasal hairs in the extrathoracic regions contributes for the filtering process. Most of the large particles are trapped by the nasal hairs which avoids those particles from penetrating into the lung volume. As a result larger particles will deposited in the extrathoracic regions.

As shown in the graph 4.3, the deposition fraction in alveolar interstitial (AI) region is high for small size aerosols as compared to other regions of the respiratory tract. The deposition of small size attached fraction of radon is performed through gravitational deposition, they can reach upto the alveolar regions as they have small size through the inhaled air. But as the size of aerosols increases from 2

μm to $7.11 \mu\text{m}$ the deposition process in alveolar region decreases from 18.3% to 2.75% and becomes almost negligible for large size aerosols. In the curve, the increase in total deposition in alveolar region is shown upto $2 \mu\text{m}$, but for the deposition greater than $1 \mu\text{m}$ diameter, aerodynamic deposition becomes dominant, and hence instead of deposition in alveolar regions, the deposition in extrathoracic region increases rapidly. And therefore the deposition of attached fraction of radon aerosols in alveolar region gets decreased.

In another way, following the extrathoracic (ET) region, the flow rate (speed) of aerosols in the inhaled air is slightly decreased, this will increase the gravitational settling of the particles. When the speed of air is slow, the residence time is becomes longer and hence gravitational sedimentation will takes place in the alveolar region. As particles velocity becomes slower, the particles will have more time to deposit by sedimentation. Gravitational sedimentation becomes more effective in alveolar region. But as the diameter of the aerosols gets larger and larger, the particles will not have a chance to pass the extrathoracic region. The inertial deposition becomes dominant and the deposition in alveolar region will approach to zero.

The aerodynamic deposition of attached fraction of radon along the trachea-bronchial regions (bronchi (BB), and bronchiolar (bb) regions) is small and almost constant. There is still deposition of attached fractions through inertial impaction in bronchi (BB) region as trachea is divided into left and right bronchi, the inhaled aerosols will impact around this branching angle when airway changes direction. Gravitational settling deposition is performed in the bronchiolar (bb) region as shown in the graph of 4.2. As particles are travel through air, the gravitational force eventually overcome their motion, as a result particles will settle down to the lower surface of the lung (bronchioles (bb)).

Normally, the deposition of attached fraction of radon is performed through inertial impaction and gravitational sedimentation. The small size part of attached fraction of radon is deposited through gravitational sedimentation in the alveolar region but as size of aerosols increased the influence of inertial impaction becomes more effective. Generally, because of larger particles of attached fraction of radon aerosols are effectively collected in extrathoracic region through impaction and small size aerosols are deposited in alveolar region through gravitational effect, and hence the deposition in bronchial region does not show any further increase or decrease. In addition to the effects of particle size and density, the ciliated mucus in the

bronchiolar regions contributes for the deposition process. As the aerosols flow within the inhaled air in the bronchiolar region, the mucus will capture the incoming aerosols.

Moreover, the deposition of attached fraction of radon aerosols is dominantly performed in extrathoracic (ET) region. Their deposition in the lower regions of the respiratory tract (called lung cancer site) is low. The particles which are deposited in the extrathoracic (ET) region can be removed through coughing, sneezing and swallowing, and hence they will expelled or ingested. Among the deposited aerosols in ET2 region almost all will cleared by transport to the gastro-inertstial (GI) tract on the same way the deposited aerosols in ET1 region are cleared to the outside enviroment. Trachea-bronchial region is the main target regions for the lung cancer. The decreased deposition in trachea-bronchial region, however, will lead to a decreased dose suplement to the lung.

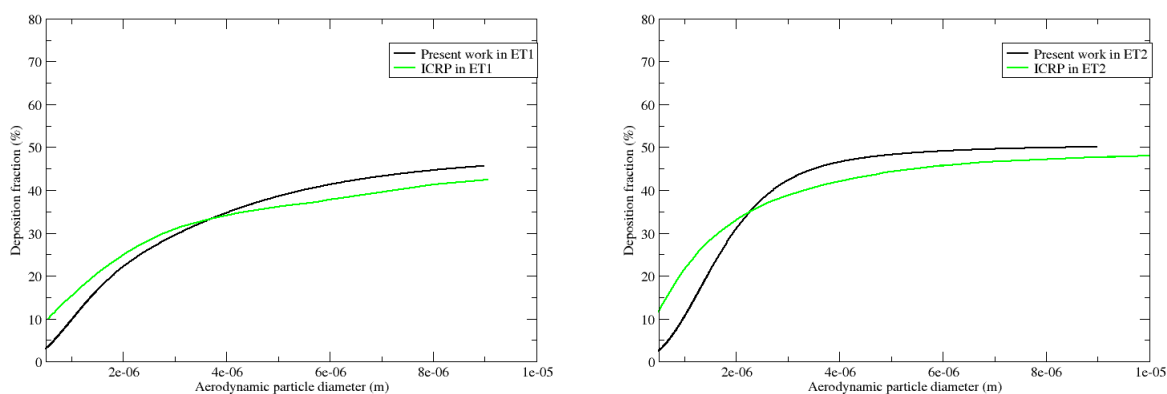


Figure 4.5: Extrathoracic region comparison

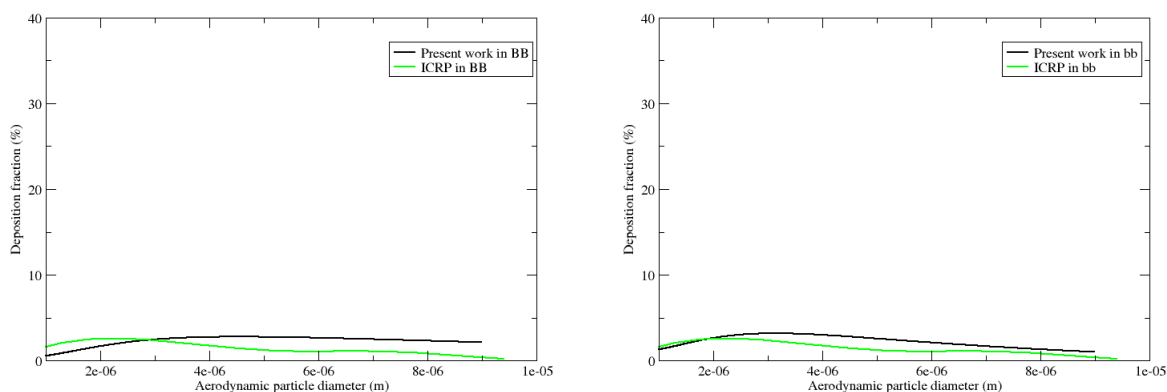


Figure 4.6: Bronchial region comparison

4.3 Comparison with ICRP66 model

The distribution of aerosol deposition in different regions of the respiratory tract for 100% nose adult breather is simulated. The result of the deposition of aerosols on each regions of the respiratory tract is compared with the graph developed by the ICRP committee. The results of the two plots in each region shows a similar deposition pattern with diameter of aerosols as indicated in graph 4.5, 4.6 and 4.7.

The deposition in extrathoracic (ET) region in both plot increases with aerosol size in similar way the deposition in alveolar region gets decreased with aerosol size. As shown in the figure 4.5, the ICRP66 model predicts slightly larger deposition for aerosol diameter in the range from 1 μm to around 2 μm in extrathoracic regions.

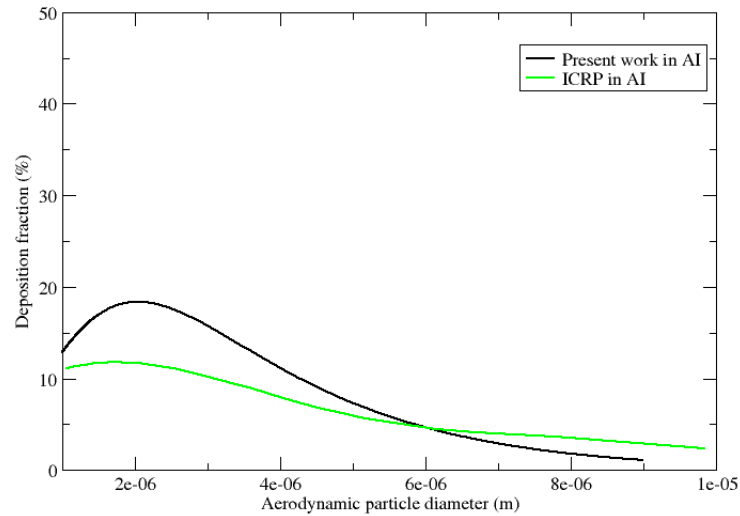


Figure 4.7: Alveolar region comparison

For the diameter greater than around $2 \mu\text{m}$, the ICRP66 model predicts under estimated deposition fraction. But for the diameter around $2 \mu\text{m}$ agreement in deposition fraction is observed between the two plots.

In bronchial region, an over estimated deposition fraction is predicted by ICRP66 model for aerosol diameter range from $1 \mu\text{m}$ to around $2 \mu\text{m}$ as compared to the present work as shown in figure 4.6. The two predictions agrees in the diameter approximately around $2 \mu\text{m}$. In alveolar regions for aerosol diameter between $1 \mu\text{m}$ to around $6 \mu\text{m}$, the present work predicts under estimated deposition fraction where as for a diameter greater than $6 \mu\text{m}$ the ICRP66 model predicts slightly larger deposition fraction as shown in figure 4.7.

This little discrepancy between the present work and ICRP66 mode is due to a modification of parameters. The data for ICRP 66 plot in each regions above is generated by extracting the regionaldeposition plot of ICRP 66 publication which is based on breathing rate of $1.2 \text{ m}^3/h$ (sitting and light exercise). But the simulated graph (black one) along each region is plotted at a flow rate of $0.78 \text{ m}^3/h$. The two deposition plots were compared to see the deposition deviation. The other parameters were kept constant in both cases. This is because in our simulation a best estimated newly modified ICRP parameter values are used, so as to fit with the experimental measurements. The result obtained in this study is also in agreement with the theory explained in the review literature.

Conclusion And Recommendations

5.1 Conclusion

Many lung models were developed to study the deposition of aerosols in the respiratory system. In this work, a comparison of a deposition estimated by newly modified parameters and ICRP66 model deposition were performed. The resulting deposition from the modified breathing rate aerosols in the extrathoracic, bronchial and alveolar region were compared with the deposition predicted by ICRP66. According to the comparison, the deposition predicted by experimentally modified parameters is slightly differ from ICRP 66 deposition prediction.

5.2 Recommendations

The regional deposition of attached fraction of radon progeny from modified parameters in each regions of the respiratory tract was determined following the inhalation. It is recommended to compare the deposition from the modified parameters along each regions with the experimental data. In the future, the regional deposition of the inhaled attached fraction of radon progeny calculated in this paper can be used for further study of different aspects of radon and its decay products in the respiratory tract. The deposition data obtained in this work allows to study about the clearance of the deposited activity in the respiratory tract. All the deposited radioactive material will not contribute for the regional dose effect as the body will have its own clearance mechanism against foreign particles.

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DECLARATION

ADDIS ABABA UNIVERSITY
COLLEGE OF NATURAL AND COMPUTATIONAL SCIENCES
DEPARTMENT OF PHYSICS

MSc Thesis

Deposition Analysis of Attached Fraction of Radon Progeny in The Human
Respiratory Tract

Name of Candidate: Guadie Degu Belete

I the under signed declare that the thesis is my original work and no part of it can be claimed as an intellectual property of anybody else except me and my advisors.

Signature: _____