



AN ABSTRACT OF THE THESIS OF

Jason D. Hout for the degree of Master of Science in Radiation Health Physics presented on September 2nd, 2010.

Title: Development of a Dose Conversion Factor from Uptakes of Po-210 through Smoking using a Modified ICRP 30 Lung Model.

Abstract Approved: \_\_\_\_\_

Kathryn A. Higley

A thesis presented on the determination of factors related to deposition, clearance, and dosimetry of the ICRP 30 lung model as related to smoking. Variations in calculations used to determine radiation doses leave a need to determine a standard method to determine absorbed dose from smoking. Standard parameters are used based upon recent studies, and the ICRP 30 lung model is used because of the fact that most regulatory bodies are not yet familiar with new ICRP lung models. Po-210 was chosen as a radionuclide to validate the model due to the presence of a dose conversion factor in regulatory literature, available data as related to smoking, and recent attention brought by the Litvinenko spy case. Results show a consistency with published scientific findings, with a dose conversion factor for the tracheobronchial region of  $3.71 \times 10^{-3}$  Sv/Bq. More attention is required however to produce a more accurate dose conversion factor to account for shortcomings inherent within this model.

Development of a Dose Conversion Factor from Uptakes of Po-210 through Smoking  
using a Modified ICRP 30 Lung Model

by

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I understand that my thesis will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my thesis to any reader upon request.

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Jason D. Hout, Author

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Development of a Dose Conversion Factor from Uptakes of Po-210 through Smoking  
using a Modified ICRP 30 Lung Model

## **Introduction**

The International Commission on Radiological Protection (ICRP) lung model is the basis for most regulatory guidance for radionuclide uptakes through inhalation in the United States. This model was derived based upon a simplistic model given in ICRP 2 (International Commission on Radiological Protection, 1959) and modifications made to the model based on findings of the Task Group on Lung Dynamics (Task Group on Lung Dynamics, 1966), formed by the ICRP to develop a more accurate model. The task group's findings were published in 1966, and the improved lung model, given in ICRP 30 (International Commission on Radiation Protection, 1979), was published in 1979.

One notable exception to the way the ICRP 30 lung model estimates radiation dose is the act of smoking. With the ICRP 30 lung model, most of the dose received is to the pulmonary region of the lung. In smokers it has been shown that a majority of lung cancers are produced in the tracheobronchial region (American Cancer Society, 2009). Given this data, it stands to reason that the ICRP 30 model could have some flaws when it comes to determining the dose from smoking.

Major radioactive constituents of cigarette smoke are from the Thorium and Radon decay chains. Po-210 was chosen as a representative radionuclide to model

due to its presence in literature related to smoking, relatively long half life, and notoriety from the recent poisoning of Alexander Litvinenko, the Russian spy who died from Po-210 poisoning in 2006 (Gardiner, 2007).

### **The ICRP Lung Model**

The ICRP 30 lung model uses the recommendations of the Task Group on Lung Dynamics (TGLD) as the basis for the deposition and retention model. The ICRP 30 model is essentially the same but some minor differences between the two exist as new data and discoveries prompted changes to the TGLD model prior to the publishing of ICRP 30. When changes between the two exist, the ICRP 30 change is taken to represent the model.

Deposition into the human lung has three main assumptions: that the dust particles follow a log-normal distribution in size, that the activity state of the person inhaling the particles affects deposition, and that the aerodynamic properties of the dust, along with the physiology and anatomy of the pulmonary system govern deposition (Task Group on Lung Dynamics, 1966). The main method of classifying particle size distributions is by the aerodynamic median activity diameter (AMAD).

The size, or AMAD of the dust distribution is most important for the nasopharyngeal (N-P) and pulmonary regions of the model, whereas the

tracheobronchial region generally stays the same (about 0.08 deposition from 0.2 to 10 $\mu$ m AMAD). As AMAD increases, the deposition in the N-P region increases, while deposition in the pulmonary region decreases. The converse also holds true for this model. The main reason for this behavior is that when particles increase in size, it becomes more difficult to traverse the arduous path through the N-P region to reach other areas of the respiratory tract (Task Group on Lung Dynamics, 1966).

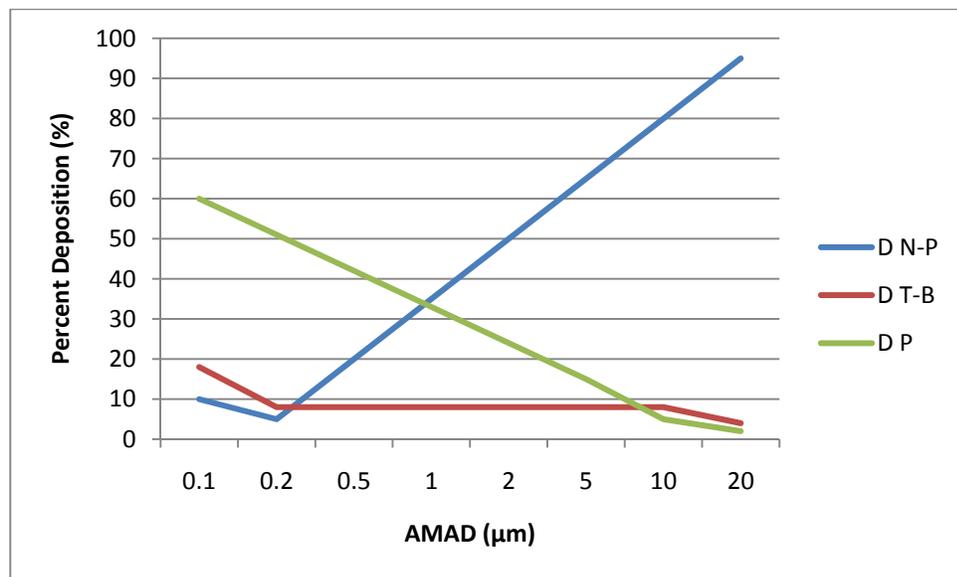


Fig. 1. Deposition of dust in the respiratory system (International Commission on Radiation Protection, 1979).

The ICRP lung model makes the assumption that the inhaled dust will be taken up through the nose, vice the mouth (International Commission on Radiation Protection, 1979). TGLD gives the reason for this as it is too difficult to determine deposition through the mouth due to variations in shape of the mouth affecting actual deposition. Dust inhaled through the nose is deposited in a logarithmic relationship to its AMAD.

$$N = -0.62 + 0.475 \log(D_a^2 F)$$

Eq. 1. Nasal deposition relationship (Task Group on Lung Dynamics, 1966).

$$NP = N_i + N_e(1 - N_i)[1 - (TB' + P')(1 - V_n/V_t)]$$

Eq. 2. Total N-P deposition fraction (Task Group on Lung Dynamics, 1966).

Nasopharyngeal deposition is assumed to follow Eq. 1, above. This relationship was determined empirically. In Eq. 1,  $N$  is the fraction of particles which are deposited into the nose,  $D_a$  represents the aerodynamic diameter (AMAD), while  $F$  stands for the inhalation flow rate in l/min. Hygroscopic effects have not been observed in the N-P region for nasal breathing (Task Group on Lung Dynamics, 1966), and are assumed to not take place due to the short transit time of particles.

Eq. 1 is assumed to apply to both inhalation and exhalation. Total nasal deposition is estimated by using Eq. 2. Here,  $NP$  is total deposition in the N-P region;  $N_i$  and  $N_e$  are the fractions of deposition in the N-P region during inhalation and exhalation, respectively;  $TB$  is tracheobronchial deposition;  $P$  is the pulmonary deposition fraction;  $V$  is the tidal volume;  $V_n$  is the volume of the N-P region. Eq. 2 applies to aerosols with AMADs between 0.1  $\mu\text{m}$  and 20  $\mu\text{m}$ . For any distribution greater than 20  $\mu\text{m}$ , it is assumed that total deposition occurs in the N-P region (Task Group on Lung Dynamics, 1966).

The activity state in the ICRP lung model is assumed to be for an individual in working conditions, such as a laboratory or industrial setting (International Commission on Radiation Protection, 1979). TGLD gives three different tidal volumes (TV) of 750, 1450, and 2150 cm<sup>3</sup>. The lowest activity state is representative of mild to moderate activity (Task Group on Lung Dynamics, 1966). The number of respiratory cycles is held constant at 15 cycles per minute. A cycle is considered inhalation followed by exhalation with no pause. ICRP 23, Reference Man, gives the minute volume of the lungs to be 20.0l/min during light activity, which correlates to a TV of 1250 cm<sup>3</sup> (International Commission on Radiological Protection, 1974).

		Diameter of Sphere ( $\mu$ )									
Tidal Volume	Location	0.01	0.06	0.20	0.60	1.0	2.0	3.0	4.0	6.0	10.0
750 cm <sup>3</sup>	N-P	0	0	0	0	0.036	0.406	0.552	0.654	0.799	0.992
	T-B	0.307	0.068	0.027	0.020	0.027	0.051	0.071	0.084	0.091	0.007
	P	0.506	0.585	0.281	0.204	0.250	0.346	0.308	0.238	0.103	0.002
1450 cm <sup>3</sup>	N-P	0	0	0	0	0.275	0.522	0.665	0.773	0.923	1.0
	T-B	0.256	0.051	0.017	0.019	0.027	0.050	0.064	0.069	0.043	0
	P	0.676	0.711	0.334	0.215	0.242	0.330	0.250	0.150	0.033	0
2150 cm <sup>3</sup>	N-P	0	0	0	0.068	0.371	0.607	0.736	0.844	1.0	1.0
	T-B	0.208	0.035	0.015	0.021	0.030	0.056	0.067	0.062	0	0
	P	0.746	0.653	0.294	0.209	0.226	0.285	0.195	0.092	0	0

Fig. 2. Deposition Fractions for differing TV's (Task Group on Lung Dynamics, 1966).

At the time of the TGLD report, it was almost impossible to determine an empirical relationship for tracheobronchial deposition. The TGLD based its deposition model for the T-B region off of studies done by Findeisen and by Gormley and Kennedy (Task Group on Lung Dynamics, 1966). A respiratory cycle of 4 seconds was assumed, with a pause prior to exhalation of 0.2 seconds. Inhalation took place for 1.74 seconds while exhalation was 2.06 seconds. Calculations were then determined for tidal volumes of 700, 1400, and 2100 cm<sup>3</sup>. An assumption was made that the first 50 cm<sup>3</sup> of air inhaled into the T-B region was air that was previously in the pulmonary region. After multiplying by a factor that adjusted for this assumption, the 50 cm<sup>3</sup> was added onto the tidal volume.

Deposition in the pulmonary region was based on the work of Wilson and LaMer, as well as Hatch (Task Group on Lung Dynamics, 1966). Results from Wilson and LaMer were shown using mouth breathing through a tube, while Hatch's results were through nasal breathing. Both studies assumed a 92% humidity in the lungs, which was proven to be 99% later. TGLD calculated results based upon 99% humidity and charted them along with the experimental data.

Particle size, and thus deposition, is affected by the absorption of moisture in the air (Task Group on Lung Dynamics, 1966). Equation 3 shows a correction factor used to account for changes in mass diameter due to humidity within the respiratory tract. In this equation  $D_s$  is the equilibrium diameter,  $D_c$  is the dry particle diameter,  $\rho_c$  and  $\rho_s$  are the densities of the dry particle and droplet of solution,  $M_w$  is the molecular

weight of water,  $M_c$  is the molecular weight of the dry particle,  $n$  is the effective number of ions produced by dissolution of a solute molecule, and  $H$  is the relative humidity.

$$\frac{D_s}{D_c} = \left\{ \frac{\rho_c}{\rho_s} \left[ 1 + \frac{M_w}{M_c} \left( \frac{n - 1 + H}{1 - H} \right) \right] \right\}^{1/31}$$

Eq. 3. Effect of humidity on particle size (Task Group on Lung Dynamics, 1966).

The lung clearance model for ICRP 30 was derived from experimental data and clinical studies. This model expands upon the three compartment deposition model and splits each compartment in two (with the exception of the pulmonary region which has four different clearance mechanisms), providing for different mechanisms to clear foreign material from the respiratory system. Also interacting with the clearance model is the blood (transfer compartment), gastrointestinal system, and the lymphatic system (International Commission on Radiation Protection, 1979).

There are four processes for removal of dust from the respiratory system, endocytosis, the ciliary movement of mucus, absorption, and lymphatic draining. Endocytosis is a process in which a cell absorbs the foreign material into the membrane. This process can allow radioactive material to remain in the area for a longer amount of time, or through cell processes remove the material from the area. This is thought to be based upon the chemical properties of the material. This process tends to occur more readily for smaller particles, but there is no established limit on

the size in which the process does not affect particle removal (Task Group on Lung Dynamics, 1966).

The second process by which dust may be removed is through the ciliary process. This process acts much like an escalator, trapping dust within the mucus and then transporting it up the system to the esophagus. This process tends to be a slower process, moving particles at rates of mm/min to cm/min. Since the mucus layer is continually moving, the removal effects of this process are immediate. Half times for this process are thought to be in minutes (Task Group on Lung Dynamics, 1966).

Absorption occurs when the radioactive material complexes with a protein or ligand, and thus becomes affected by the biologic processes specific to that organic compound. This process is thought to be responsible for a rapid partial clearance of material, usually occurring within the first few hours. Material is generally cleared to the blood, liver, or excreted as urine. At the time of the TGLD report, there was no substantial evidence to theorize where this type of process could be occurring in the lung. The idea being that since it is a very rapid clearance to the GI tract, blood, or other system, the specific process would not need to be known (Task Group on Lung Dynamics, 1966).

Lymphatic draining occurs as part of the pulmonary clearance process. Since the kinetics of removal were not very well known at the time of the TGLD report, it was generally accepted that the lymphatic system acted as a pump, with flows up to

over 1000ml/day in a person, based upon canine studies (Task Group on Lung Dynamics, 1966).

Based upon these four types of removal methods, TGLD derived ten different clearance mechanisms, each represented by a letter between a and j. Specifically:

(a) is the rapid uptake of material deposited in the N-P region to the transfer compartment;

(b) is the rapid clearance of material in the N-P region by ciliary-mucus transport;

(c) is the rapid absorption of material in the T-B region into the transfer compartment;

(d) is the ciliary-mucus clearance from the T-B region;

(e) is the translocation of material from the pulmonary region to the transfer compartment;

(f) is a combination of endocytosis within the ciliary-mucus escalator and the escalator action itself to remove material from the pulmonary region. This action takes the material through the T-B region and into the GI tract;

(g) is the same process as (f) but is a much slower removal rate primarily attributed to the characteristics of the dust deposited;

(h) is a slow removal of material to the lymphatic system and is regarded as similar to (g) with the lymph transport replacing the ciliary-mucus escalator;

(i) is the pathway from the lymphatic system to the transfer compartment;

(j) is a portion of the lymphatic system in which material is retained indefinitely.

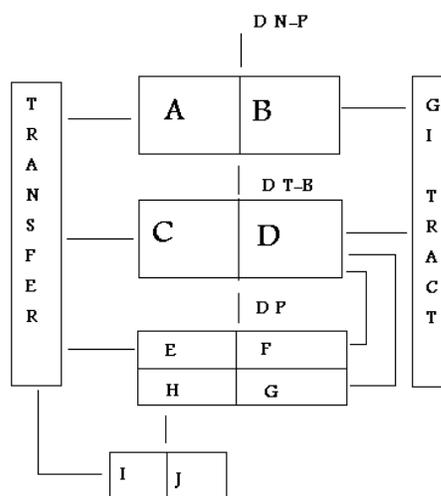


Fig. 3. Diagram of the lung model (International Commission on Radiation Protection, 1979).

To correctly model lung clearance, there must be specific data, based upon chemical interactions, that defines the rates at which material is cleared through each process. There is not a complete description of in vivo reactions of all chemical compounds at this time. However, TGLD attempted to define clearance based upon available studies at the time. This resulted in all elements being grouped into three classes, based upon their chemical form (Task Group on Lung Dynamics, 1966).

The three classes that all elements are grouped into in this lung model are defined by their biological clearance time. Class D compounds are expected to have half times of less than 1 day and clear the body within ten days; class W compounds have half times between a few days and a few months, and are generally clear of the body within 100 days; class Y compounds have half times of longer than a few months, and range from clearing the body in greater than 100 days to infinite retention (Task Group on Lung Dynamics, 1966).

Region	Compar- tment	Class					
		D		W		Y	
		T Day	F	T Day	F	T Day	F
N-P ( $D_{N-P} = 0.30$ )	a	0.01	0.5	0.01	0.1	0.01	0.01
	b	0.01	0.5	0.4	0.9	0.4	0.99
T-B ( $D_{T-B} = 0.08$ )	c	0.01	0.95	0.01	0.5	0.01	0.01
	d	0.2	0.05	0.2	0.5	0.2	0.99
P ( $D_P = 0.25$ )	e	0.5	0.8	50	0.15	500	0.05
	f	n.a.	n.a.	1	0.4	1	0.4
	g	n.a.	n.a.	50	0.4	500	0.4
L	h	0.5	0.2	50	0.05	500	0.15
	i	0.5	1	50	1	1000	0.9
	j	n.a.	n.a.	n.a.	n.a.	$\infty$	0.1

Fig. 4. Lung clearance rates and fractions (International Commission on Radiation Protection, 1979).

## **Method**

Research was conducted via the internet and visits to the Oregon State University Library to compile a more accurate picture of how the lungs react to smoke inhalation. When possible, factors based on empirical results were used and the use of assumptions were minimized. A personal computer was used for internet searches to find journal articles and internal cigarette corporation memos. Calculations were performed by hand with the use of a handheld calculator. No computer modeling software was used. This method is primarily aimed to achieve reproducible results without the need of sophisticated computer software.

In essence, the same lung model as ICRP 30 was used, with changes related to situations specific to smoking. Since the stochastic risk to the lung is only based upon the T-B region and the pulmonary region, radiation doses based upon the average concentration of Po-210 in cigarettes were calculated for these regions only. However, in order to obtain a more complete model of smoking, deposition in the N-P region was calculated.

The ICRP 30 supplement lists the annual limit of intake (ALI) and derived air concentration (DAC) values for various radionuclides, making an assumption that the AMAD of the particles inhaled is 1 $\mu$ m. When broken down to a dose conversion factor, the dose to the lung from inhalation of Po-210 is  $1.3 \times 10^{-5}$  Sv/Bq (International Commission on Radiation Protection, 1979).

Martell (Martell, 1983) determined that the Radon progeny within cigarette smoke has an AMAD between 1 and 2 $\mu\text{m}$ . A more recent study conducted by Dickens, et. al (C. Dickens, 2008), showed results averaged between 0.15 and 0.17  $\mu\text{m}$ . Bernstein (Bernstein, 2004) reviewed many publications and determined found that particle size ranged between 0.18 and 0.34  $\mu\text{m}$ . Gower and Hammond (S. Gower, 2007) suggested a size distribution with a median between 0.1 and 1.0  $\mu\text{m}$ . Since particle deposition depends heavily on the AMAD, it would make the most sense to assume a range of 0.2 to 2  $\mu\text{m}$  diameter.

A problem with the results in AMADs shown are that there was no adjustment for hygroscopic growth. Hygroscopic growth is the effect that occurs when a dry particle enters a humid environment and absorbs moisture, having the effect of increasing the original particle size. Asgharian (Asgharian, 2004) found that 1  $\mu\text{m}$  AMAD particles will experience hygroscopic growth by a factor of 5, increasing size to 5  $\mu\text{m}$  by the time it reaches the T-B region. Thus deposition calculations will be assumed for an AMAD of 1 to 10  $\mu\text{m}$  for the T-B and pulmonary regions, and utilize a 0.2 to 2  $\mu\text{m}$  AMAD for the N-P region.

Another effect that is not accounted for in ICRP modeling is coagulation. This has the effect of increasing individual particle size when particle density is high, primarily due to individual particles combining to form larger particles. Since cigarette smoke has a high density of particles, this effect is certainly a factor (S.

Gower, 2007). Due to the lack of literature available regarding compensation for this effect, it cannot be fully accounted for.

A major flaw that cannot be accounted for completely with this particular model is the effects of cloud motion on cigarette smoke. Martonen and Musante (T. Martonen, 2000) as well as Gower and Hammond (S. Gower, 2007) have convincing evidence that cigarette smoke tends to act like a cloud when inhaled versus individual particles. This has the net effect of a simulated enlarging of the AMAD of particles. Martonen and Mustante (T. Martonen, 2000) found that 0.443 AMAD particles will tend to have a deposition fraction of 0.99 in the TB region when simulated under cloud mechanics. Since cloud modeling requires a great length of computational time an increase in AMAD will be used for each region that simulates the effects of cloud mechanics.

A major assumption that the ICRP 30 model makes is that there is only nasal breathing during inhalation and exhalation. The cigarette was designed to be inhaled through the mouth. Generally, the diameter of a filtered cigarette is between 7-8 mm. Puff velocities, range between 1 and 2.5 l/min. Martell (Martell, 1983) stated the average puff velocity as 1.1 l/min, while results from Dickens, et. al, (C. Dickens, 2008) show ranges between 1.65 and 2.44 l/min. Dickens, et. al (C. Dickens, 2008), performed a study on 7 smokers and measured puffing behavior and particle size. The results of this are shown in figure 5.

Subject	Total Puff Volume (ml)	Mean Puff Volume (ml)	Average Puff Duration (s)	Average Puff Flow (mL/s)	CMD (nm)
1	572	95 ± 2	3.3 ± 0.2	29.5 ± 2.4	154 ± 5
3	576	72 ± 1	1.7 ± 0.3	40.7 ± 1.7	156 ± 4
9	376	63 ± 13	1.9 ± 0.3	35.0 ± 2.9	159 ± 9
12	400	57 ± 1	1.7 ± 0.1	34.5 ± 1.1	159 ± 1
102	717	119 ± 7	2.6 ± 0.5	27.5 ± 3.0	160 ± 8
105	440	73 ± 4	1.5 ± 0.2	35.0 ± 0.9	172 ± 7
112	441	55 ± 5	3.3 ± 0.2	29.5 ± 2.4	156 ± 1

Fig. 5. Puffing behavior and particle size (C. Dickens, 2008).

From the data in figure 6, a tidal volume directly related to the act of smoking can be determined. The tidal volume is an important factor in determining the deposition characteristics of smoke. From the results above it can be determined that on average it takes between 6 and 8 puffs to smoke a cigarette. However, the National Institute on Drug Abuse (National Institute on Drug Abuse, 2009) states that an average smoker will take around 10 puffs on a cigarette and will spend approximately 5 minutes smoking. Using the NIDA results would yield a frequency of 2 puffs/min. TGLD uses a 15 breath/min frequency, and data shown by Stuart (Stuart, 1984) of various studies in deposition show between 14 and 15 breath/min, thus a 15 breath/min frequency should be used, with 2 of those 15 breaths being puffs on a cigarette.

The act of puffing on a cigarette is unique compared to regular breathing in that the smoker will tend to inhale the cigarette smoke into his or her mouth, hold the smoke there for a small period of time, inhale the smoke deeply into the lungs, and

then exhale (C. Dickens, 2008). This has an effect of greatly increasing the tidal volume for a given activity level. Hinds (W. Hinds, 1983) showed that a 750 ml tidal volume would have a corresponding 1870 ml tidal volume when puffing on a cigarette. Robinson and Yu (R. Robinson, 2001) determined changes in deposition based on changes in puffs. The results of their work are in figure 6. The inhalation per puff for purposes of this model shall be a total puff time of 10 seconds, comprised of an inhalation time of 6 seconds, a hold time of 2 seconds, and an exhalation time of 2 seconds.

Increase In	Effect on Total Deposition Fraction
Puff volume from 25 mL to 75 mL	Negligible
Puff time	1.0% increase/sec
Inhalation time	2.8% increase/sec
Pause (breath-hold) time	1.9% increase/sec
Exhalation times	3.9% increase/sec

Fig. 6. Changes in deposition fraction (R. Robinson, 2001).

Tidal volumes are calculated based upon the observed respiratory flow rate and the frequency of breathing. It will be assumed that smokers do not decrease nor increase the flow rate of inhalation during times at which they are not taking puffs on the cigarette. This is an adequate assumption due to the correlation between tidal volume and activity level. It can be safe to say that a person would normally be at a resting activity level while smoking. TGLD gives no values for a resting tidal volume

but places a value for light activity at 750 ml. Of note is that ICRP 23 places a 750 ml tidal volume as a resting value. For the purposes of this model, a 500 ml tidal volume will be assumed to be a resting level, corresponding to a 33 ml/min minute volume during non puff times. This corresponds to a tidal volume of 1250 ml while puffing.

Since the ICRP 30 model did not address oral deposition, adjustments must be made to account for inhaling a cigarette through the mouth. The two major means of deposition within the oral cavity are diffusion and impaction. Diffusion tends to predominate in smaller diameter distributions, whereas impaction is the major method of deposition in larger diameters. Cheng (Cheng, 2003) derived an equation that addresses oral deposition in equation 4. The basis of this equation was derived from using human subjects and assessing deposition by breathing through a tube. Such a shape of the mouth should be similar to cigarette smoking, and therefore should be fairly accurate in determining deposition. Deposition values for 0.5 and 2  $\mu\text{m}$  are 0.0 and 0.014, respectively. Results from Mitsakou et al. (C. Mitsakou, 2007) support Cheng's conclusions.

$$E_o = 1 - e^{(-0.000278d_a^2Q - 20.4D^{0.66}Q^{-0.31})}$$

Eq. 4. Oral Deposition (Cheng, 2003).

For cigarette smoking, the deposition in the tracheobronchial region could very well be the most important parameter to determine an accurate radiation dose. Most tumors related to cigarette smoking occur in this region (American Cancer Society, 2009), yet historically deposition models have estimated between an 8-10% deposition

(International Commission on Radiation Protection, 1979) (Task Group on Lung Dynamics, 1966). Chan and Lippman (T. Chan, 1980) provided an equation that better estimates T-B region deposition. This equation adds a factor called bronchial deposition size, which is a factor accounting for the physical characteristics of bronchial constriction based upon empirical data. The BDS for smokers is 1.02, whereas for nonsmokers it is 1.20. These values of BDS assume that there is no bronchial disease present.

$$TB = 8.14 \times 10^{-4} [D^2 Q / (BDS^3)] + 0.037$$

Eq. 5. TB region deposition (T. Chan, 1980).

Pulmonary deposition tends to follow T-B region deposition for smaller particle diameters, but tends to fall off at higher diameters due to the increased deposition in the T-B region. This is primarily due to impaction in the trachea at higher diameters, and at lower diameters sedimentation has more of an effect, depositing preferentially into the pulmonary region. Sedimentation is the primary cause of deposition in the pulmonary region due to air flow essentially going to zero where the alveoli have a large cross sectional area. Also, the increase in efficiency in the T-B region for particles larger than 4  $\mu\text{m}$  causes a corresponding decrease in the particles reaching the pulmonary region. No available information was found on an empirical equation for the deposition of particles in the pulmonary region, thus pulmonary deposition will be assumed at 0.20.

Clearance from the N-P region will not be addressed further in this paper.

Main reasons for not addressing N-P clearance are the lack of publications related to the subject and that the dose received to the N-P region is not relevant to the committed effective dose to the lungs. Since the N-P region does not contribute to the radiation dose received by the lungs, clearance methods for the N-P region can be assumed to not change from the ICRP model.

Clearance from the tracheobronchial and pulmonary regions are usually grouped together in more recent literature. Martell (Martell, 1983) describes a clearance model of soluble and insoluble materials, while Lourenco, et al. (R. Lourenco, 1970), describes clearance related strictly to smokers for 2  $\mu\text{m}$  monodisperse particles. Neither of these studies differentiate between the pulmonary and T-B regions.

Exposure to cigarette smoke can have an effect on clearance of particles. Chan, et al. (T. Chan, 1980), found that in "normal" smokers, clearance rates are either enhanced or there is no effect. In addition, chronic smokers may have large variability in clearance rates. However, in chronic smokers that have quit, an improvement in clearance has generally been seen, which may suggest a slight reduction in clearance rates.

Lourenco (R. Lourenco, 1970) performed a study between clearance rates on smokers and non smokers. Results from that study show that while after the first 24 hours clearance rates are generally the same between the two groups, from deposition

to 24 hours after deposition clearance rates are markedly different. Non smokers generally clear material much faster than smokers within the first day, showing an almost exponential clearance rate, while smokers have a more linear clearance rate. Lourenco indicated that a possible cause for the slower clearance rates could be a slower mucociliary escalator, which is plausible given that cigarette smoke is a ciliostatic substance.

Martell's (Martell, 1983) clearance model relied mainly on whether a particle was soluble or insoluble. Soluble particles tended to have faster clearance times, whereas insoluble particles were removed very slowly, at a half time around 172 minutes for smokers. This type of model allowed for complete decay of some of the radon daughter products. Martell's assumption regarding smoke depositing in localized hot spots led to estimated doses of 2-5 rad per cigarette smoked to the T-B region.

Given the data discussed previously and the knowledge of the ICRP clearance model, it might be an accurate assumption to assume that the rapid uptake of dust from the T-B region to the blood does not exist in the same fashion for smokers as assumed in the model. For purposes of dose estimation in this document, it is assumed that this type of clearance has the same effective clearance rate as the slower clearance process from the T-B region. Pulmonary clearances are assumed to be the same as the ICRP model due to a lack of a more rapid clearance rate from one removal method to the

other, and lack of information regarding pulmonary clearance. For the purposes of this model, clearance rates for regions other than mentioned shall be class W rates.

## Results

Deposition was plotted as a function of AMAD for the nasopharyngeal and tracheobronchial regions. Results are shown in figures 7 and 8. Pulmonary deposition was not plotted due to lack of data and assumptions previously made by the author.

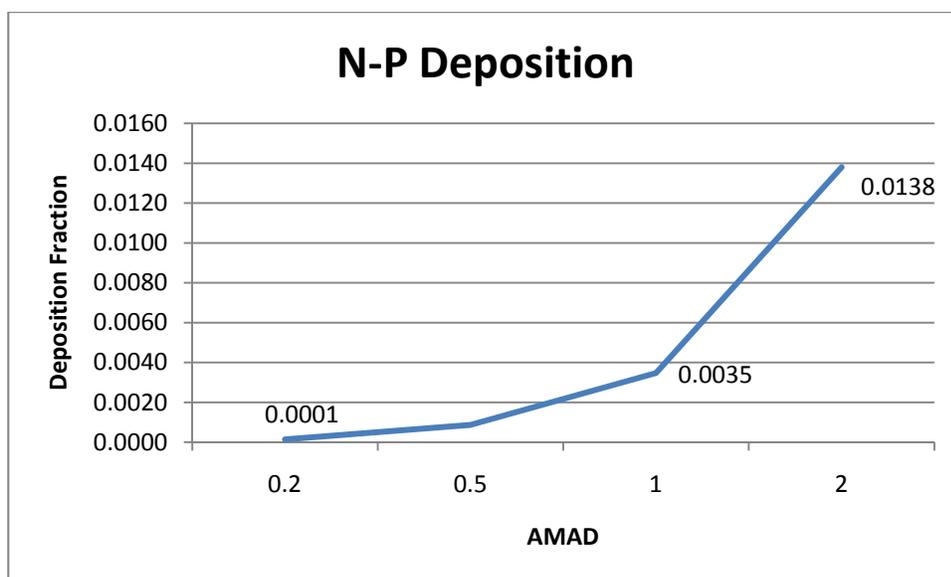


Fig. 7. Calculated N-P Deposition.

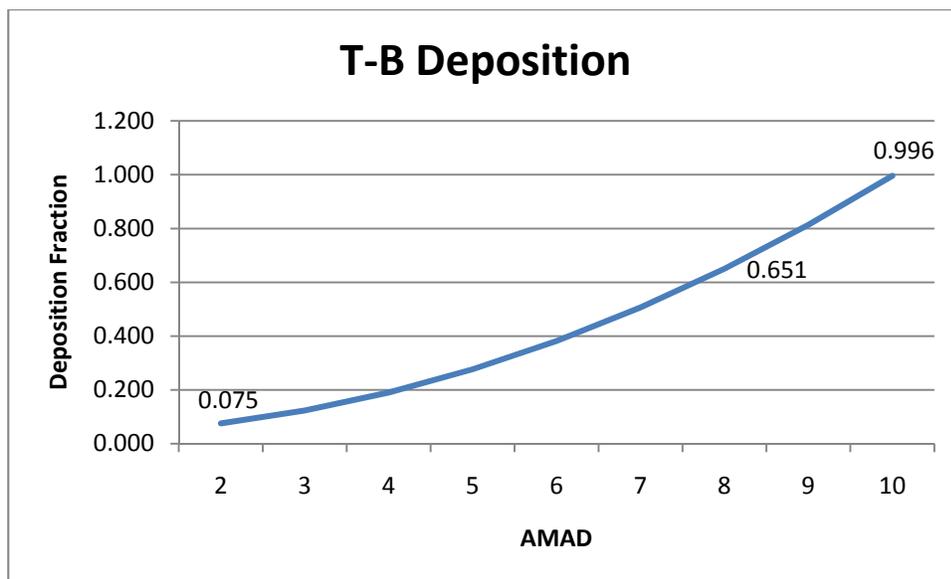


Fig. 8. Calculated T-B Deposition.

Calculations were performed to determine radiation dose to the lung based off of variations of the model described in the previous chapter. Results are shown in tables 1 through 6. Various parameters were adjusted to account for possible changes in cloud mechanics, tracheobronchial clearance, and pulmonary deposition.

Committed Effective Doses to the Lung and Tracheobronchial Region  
Based Upon 1  $\mu\text{m}$  AMAD Inhaled, Hygroscopically Growing to 8  $\mu\text{m}$   
AMAD and Depositing 20% Into the Pulmonary Region

Activity (Bq)	H <sub>50</sub> total lung	H <sub>50</sub> T-B region
1	1.04E-05	8.34E-06
0.222	2.75E-06	1.85E-06
0.008	6.49E-07	6.67E-08
0.00629	6.33E-07	5.24E-08

Doses are in Sv

Table 1.

Committed Effective Doses to the Lung and Tracheobronchial Region  
Based Upon 0.2  $\mu\text{m}$  AMAD Inhaled, Hygroscopically Growing to 2  
 $\mu\text{m}$  AMAD and Depositing 20% Into the Pulmonary Region

Activity (Bq)	H <sub>50</sub> total lung	H <sub>50</sub> T-B region
1	1.02E-05	2.25E-06
0.222	2.70E-06	5.00E-07
0.008	6.47E-07	1.80E-08
0.00629	6.31E-07	1.42E-08

Doses are in Sv

Table 2.

Committed Effective Doses to the Lung and Tracheobronchial Region  
Based Upon 2  $\mu\text{m}$  AMAD Inhaled, Hygroscopically Growing to 10  
 $\mu\text{m}$  AMAD and Depositing 0% Into the Pulmonary Region

Activity (Bq)	H <sub>50</sub> total lung	H <sub>50</sub> T-B region
1	4.21E-07	1.05E-05
0.222	9.34E-08	2.34E-06
0.008	3.37E-09	8.42E-08
0.00629	2.65E-09	6.62E-08

Doses are in Sv

Table 3.

Committed Effective Doses to the Lung and Tracheobronchial Region  
Based Upon 1 $\mu$ m AMAD Inhaled, Hygroscopically Growing to 8  $\mu$ m  
AMAD and Depositing 20% Into the Pulmonary Region, With No  
Removal From the Tracheobronchial Region

Activity (Bq)	H <sub>50</sub> total lung	H <sub>50</sub> T-B region
1	2.41E-04	5.78E-03
0.222	5.40E-05	1.28E-03
0.008	2.50E-06	4.62E-05
0.00629	2.08E-06	3.63E-05

Doses are in Sv

Table 4.

Committed Effective Doses to the Lung and Tracheobronchial Region  
Based Upon 0.2  $\mu$ m AMAD Inhaled, Hygroscopically Growing to 2  
 $\mu$ m AMAD and Depositing 20% Into the Pulmonary Region, With No  
Removal From the Tracheobronchial Region

Activity (Bq)	H <sub>50</sub> total lung	H <sub>50</sub> T-B region
1	7.25E-05	1.56E-03
0.222	1.65E-05	3.46E-04
0.008	1.15E-06	1.25E-05
0.00629	1.02E-06	9.82E-06

Doses are in Sv

Table 5.

Committed Effective Doses to the Lung and Tracheobronchial Region  
Based Upon 2  $\mu\text{m}$  AMAD Inhaled, Hygroscopically Growing to 10  
 $\mu\text{m}$  AMAD and Depositing 0% Into the Pulmonary Region, With No  
Removal From the Tracheobronchial Region

Activity (Bq)	H <sub>50</sub> total lung	H <sub>50</sub> T-B region
1	2.92E-04	7.29E-03
0.222	6.47E-05	1.62E-03
0.008	2.33E-06	5.83E-05
0.00629	1.83E-06	4.59E-05

Doses are in Sv

Table 6.

## **Discussion**

Results in the previous chapter were tabulated using expected regional values for Po-210 concentration in mainstream smoke (0.222 (China), 0.008 (Italy), and 0.00629 (United States) Bq). Regional values were provided to illustrate the variation in dose one can receive due to regional fluctuations of natural radon in the world. Tracheobronchial deposition per AMAD was a slightly different shape than modeled elsewhere, but smoking inhalation changes were the primary cause of the different shape.

Martell (Martell, 1983) estimates that the dose to small, isolated hotspots in the tracheobronchial region range from 0.16-0.32 Gy from Po-210. Estimating that the doses received are over a span of 25 years, and that an average smoker will smoke one pack a day, this results in a maximum dose per cigarette of  $1.75 \times 10^{-6}$  Sv. Results shown in tables 1 through 6 average the expected dose over the entire mass of the T-B region vice just localized hotspots. However, when this factor is accounted for, Martell's estimations based off of findings from Little, et al. (J. Little, 1965), are in agreement with doses for tables 4, 5, and 6. This indicates a strong argument for little to no removal of deposits of Po-210 within the T-B region.

Based off of the results and the agreement with Martell, another calculation was performed for a  $7 \mu\text{m}$  initial deposit in the T-B region (0.507 deposition) with zero clearance from the T-B and zero deposition into the pulmonary region. The result was a dose to the T-B region of  $8.24 \times 10^{-4}$  Sv, which is even closer to Martell's estimate ( $7 \times 10^{-4}$  Sv after factor corrections). Given that Martell's estimate was based off of actual Po-210 concentrations found in bronchial bifurcations, it is probable that this model is somewhat accurate in predicting dose. The resulting dose conversion factor is  $3.71 \times 10^{-3}$  Sv/Bq to the T-B region. This model assumes 0.35% deposition into the mouth and oral cavities, 50.7% deposition into the T-B region, and 0% deposition into the pulmonary region. A 51% total deposition is also in agreement with Dickens, et al (C. Dickens, 2008). Error is not provided due to the lack of error data in studies that led to this dose conversion factor. However, it is noted that in

human subjects there is a wide variability in biological factors present from one human to another and further exploration should be made to validate results.

There are many dilemmas with this model that cannot be improved upon without the presence of more empirical data and better modeling capabilities. A smoke cloud model would provide best results in deposition estimation. More empirical data to determine effective clearance rates on smokers would be highly valuable in determining an accurate dose conversion factor. Also of note is that pulmonary deposition should not be zero given the way a smoker inhales, but should be a low value due to impaction on the trachea and bronchi.

Obviously, this type of model has its shortcomings and will not accurately predict the occurrence of cancer unless a smaller mass is used than the T-B region, due to the nature of deposition in bronchial bifurcations. A more sophisticated computer system, which would be able to model all factors associated with smoking would be the most desired method for producing accurate results. The results and findings in this paper are meant to bridge the gap between a more incorrect model, and a more correct one. Results here should not be used in any official capacity to estimate radiation dose, but can be used as a rule of thumb to help provide validation to already concluded work.

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