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A. C. James
A. Birchall

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Pacific Northwest Laborartory
Richland, Washington 99352

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IMPLICATIONS OF THE ICRP TASK GROUP'S PROPOSED LUNG MODEL FOR INTERNAL DOSE ASSESSMENTS IN THE MINERAL SANDS INDUSTRY

by

ANTHONY C. JAMES
Pacific Northwest Laboratory, Richland, WA 99352, U.S.A.

ALAN BIRCHALL
National Radiological Protection Board, Chilton, Oxfordshire, OX11 ORQ, U.K.

ABSTRACT

The ICRP Task Group on Respiratory Tract Models for Radiological Protection is proposing a model to describe the deposition, clearance, retention and dosimetry of inhaled radionuclides for dose-intake calculations and interpretation of bioassay data. The deposition model takes into account new data on the regional deposition of aerosol particles in human lung and the inhalability of large particles. The clearance model treats clearance as competition between mechanical transport, which moves particles to the gastro-intestinal tract and lymph nodes, and the translocation of material to blood. This provides a realistic estimate of the amount of a given material (such as mineral sand) that is absorbed systemically, and its variation with aerosol size. The proposed dosimetry model takes into account the relative sensitivities of the various tissue components of the respiratory tract. A new treatment of dose received by epithelia in the tracheo-bronchiolar and extrathoracic regions is proposed. This paper outlines the novel features of the task group model, and then examines the impact that adoption of the model may have on the assessment of doses from occupational exposures to mineral sands and thoron progeny.

INTRODUCTION

The ICRP is currently engaged in revising its basic recommendations on radiation protection described in Publication 26 (ICRP 1977). This process includes review and reassessment of the system of dose limitation as well as values of primary limits. ICRP Committee 2 is preparing for a possible revision of secondary limits, which will include the annual limits on intake (ALI). To support this effort, a task group[a] on human respiratory dosimetric models was appointed to review the current lung dosimetry model and, if needed, propose a new model. The task group is proposing a model that

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(a) The members of the ICRP task group on human respiratory tract models for radiological protection are: Bill Bair (chairman), Michael Bailey, Fredrick Cross, Richard Cuddihy, Peter Gehr, Anthony James, John Johnson, Roland Masse, and Willi Stalhofen.

differs significantly in several aspects from that of ICRP Publication 30 (ICRP 1979a). The purpose of this paper is to outline these aspects of the proposed model, and to examine their implications for internal dosimetry in the processing and handling of mineral sands. It is stressed that the task group's proposals are still in draft form, and have not yet been submitted formally to the ICRP. Therefore, the model eventually adopted by the ICRP could be substantially different.

OUTLINE OF THE PROPOSED LUNG MODEL

Since the various cells and tissues of the respiratory tract are not equally sensitive to radiation exposure, and there is also a great variability in radiation doses received by the different tissues from inhaled radionuclides, the current practice of averaging dose over the lungs as a whole (ICRP 1979a) is difficult to justify. Therefore, a primary objective of the task group was a model that reflected differences in radiation sensitivities as well as the variability in dose distribution. This led to the identification of anatomical regions within the respiratory tract for which radiation sensitivities could be distinguished and for which the deposition and residence time of deposited materials could be specified (Bair 1991). The model provides for calculation of doses to these several regions.

Tissues at Risk

The target regions of the respiratory tract selected for dose calculation are those identified as the most sensitive to radiation-induced cancer and/or those that may receive the highest doses. The target tissues in these regions are:

- basal cells in keratinized epithelium of the skin in the anterior part of the nose
- basal cells in stratified squamous epithelium of the naso-oropharynx and larynx
- secretory and basal cells in ciliated epithelium of the bronchi
- secretory cells in the ciliated epithelium of the bronchioles
- epithelial cells in the alveolar-interstitium
- lymphoid cells in the thoracic and extrathoracic lymph nodes.

To estimate the relative susceptibilities of these different tissues to radiation-induced cancer, the task group assumed that induction of cancer by radiation is proportional to the spontaneous incidence in each tissue. After reviewing data from the general population on the distribution of cancers within the respiratory tract, the task group concluded that about 80% of lung cancers originate in the bronchi, 15% originate in the bronchioles, and only 5% originate from alveolar tissue. The proportion of spontaneous lung cancer that originates from nodular lymphatic tissue is considered to be extremely small (less than 0.1%). By contrast, the task group concluded that extrathoracic tissues of the respiratory tract are relatively sensitive to radiation-induced cancer. The combined incidence of cancers of the nose and nasopharynx, mouth and oropharynx, and the larynx is approximately 25% of that in lungs. The corresponding apportionment of cancer risk between these various tissues is given in Table 1. The table also gives weighting factors for dose equivalents received by each component tissue of the respiratory tract that follow from the overall value of 0.12 recommended by the ICRP for uniform irradiation of the lungs as a whole (ICRP 1977).

TABLE 1. Partition of Risk and the Proposed Weighting Factors for Dose Equivalents Received by Tissues of the Respiratory Tract

Tissue	Relative Risk		Proposed Apportionment of Weighting Factor	
Extrathoracic	0.20		0.025	
Bronchi	0.64		0.096	
Bronchioles	0.12	0.8	0.018	0.12
Alveolar Interstitium	0.04		0.006	
Lymph Nodes	0.001		0.001	

In order to examine the full implications of the task group's proposed lung model for dose assessments, which must include the effects of doses received by other organs of the body, we have assumed below the revised values of tissue weighting factors that are likely to be recommended by ICRP (ICRP 1990). These are given in Table 2.

TABLE 2. New Tissue Weighting Factors W_T Recommended in Draft by ICRP

0.01	0.05	0.12	0.20
Skin	Thyroid	Lung	Gonads
Bone Surfaces	Liver	Red Bone Marrow	
	Esophagus	Stomach	
	Breast	Colon	
	Bladder		
	Remainder(a)		

(a) The weighting factor 0.05 is to be divided between the two most highly irradiated remainder tissues.

Particle Clearance and Systemic Uptake

To model particle clearance and the associated uptake of radionuclides by the blood, the task group divided the respiratory tract into four main regions: extrathoracic (ET), bronchial (BB), bronchiolar (bb) and alveolar-interstitial (AI). Clearance from each region is treated as competition between mechanical processes and translocation (Cuddihy and Yeh 1988; Bailey et al. 1991). As shown in Figure 1, mechanical clearance transports material (mainly as particles) to the GI tract or lymph nodes (LN). Translocation refers to clearance to the blood, mainly the dissociation of particles and subsequent absorption of the dissociated material. It is assumed that mechanical clearance rates are the same for all materials, but that these are characteristically different in each region of the respiratory tract (Figure 1). Conversely, to model the systemic uptake of radionuclides from each region, it is assumed that the rate of translocation to blood of a material is the same in all regions, but that this varies characteristically with the material.

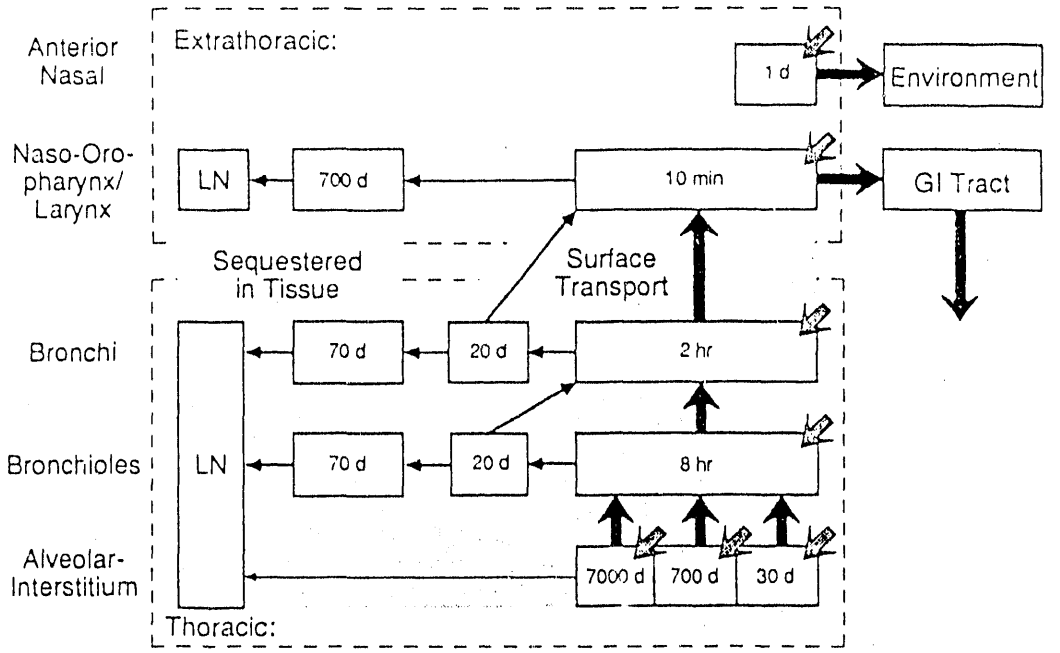

FIGURE 1. Mechanical Clearance Model Proposed by the ICRP Task Group.

The detailed structure of the mechanical clearance model (Figure 1) is determined by the need to represent all clearance pathways that may lead to significant retention of α- and ß-particle-emitting radionuclides within range of the especially sensitive target tissues in the bronchi, bronchioles and naso-oropharynx/larynx. The extrathoracic region is sub-divided into the anterior nasal passages (ET_1), which are lined by an impervious layer of skin, and the main airways (ET_2). The surface of the main airways is covered by a layer of fluid in which most particles are cleared rapidly to the GI tract. However, for dosimetry, it is assumed that a small fraction (0.05%) of material entering the naso-oropharynx and larynx by deposition from inhaled air, or by clearance from the bronchi, is sequestered by tissue in the airway wall (Snipes et al. 1983). These sequestered particles are cleared slowly to the extrathoracic lymph nodes. In both the bronchi and the bronchioles, a larger fraction (0.7%) of particles is assumed to be sequestered in the airway walls (Churg and Wright 1988; Patric'. 1989). However, of greater importance for dosimetry, recent studies of human subjects have shown that mucous clearance may be delayed for a substantial fraction of particles deposited in the bronchi or bronchioles (Stahlhofen 1989). The additional retention of particles on bronchial and bronchiolar surfaces that results from this delayed mucous clearance is represented by allocating a 20-day clearance half-time for 20% of the material deposited or cleared into these regions.

The sub-division of material deposited in the alveolar-interstitial region of the lung into the three clearance compartments shown in Figure 1 (with equal deposition in each) is introduced to represent the typical pattern of long-term retention of particles in the thorax that is measured in human subjects (Bailey 1989). The rate at which particles are cleared from alveolar-

interstitial tissue to the lymph nodes is estimated from analyses of human tissues taken at autopsy following exposures many years prior to death (Bailey et al. 1991). These autopsy data show radioactive particle concentrations in the thoracic lymph nodes that are typically ten-fold higher than in the lungs (Kathren et al. 1990).

Translocation to blood is represented by applying the same dissolution and absorption process to each compartment of the clearance model, as illustrated in Figure 2. Translocation rates for different materials exhibit a very wide range. A readily absorbed material may have a rate as great as the fastest mechanical rate (100 d^{-1}), whereas material such as mineral sand that remains in the lung for many years must have a rate less than 0.001 d^{-1}. The translocation rate of a given material may vary with time. However, since the use of time-dependent clearance functions leads to difficulties in calculations for protracted intakes and radioactive progeny, the task group proposes to represent time-dependent rates by combinations of compartments in each translocation pathway that clear at constant rates (Bailey et al. 1991). Translocation to blood is essentially a two-stage process (Figure 3). This involves the initial release of a radionuclide from a deposited particle in a form which can be absorbed into the blood, and secondly, the absorption of radionuclides once they are released, or alternatively the direct absorption of radionuclides deposited in a soluble form. For most radionuclides, absorption to blood can be regarded as effectively instantaneous, although for some important elements a significant fraction of the dissolved material is absorbed slowly as a result of binding to respiratory tract components (Cuddihy 1984). The task group will recommend translocation rates (and bound fractions when necessary) for materials that have been well studied in man or in experimental animals. In other cases, the following default values will be proposed for materials classified as D, W, and Y, respectively, in the ICRP 30 system (ICRP 1979a):

- "F" - fast translocation, with $t_{\frac{1}{2}} \approx 10$ minutes. This is represented by $s_p = 100\ d^{-1}$ (Figure 3). Almost all material deposited in BB, bb and AI, and 50% of the material deposited in ET_2 is rapidly translocated (the skin of ET_1 is assumed to be impervious).

- "M" - moderate translocation, 50% with $t_{\frac{1}{2}} \approx 3$ days and 50% with $t_{\frac{1}{2}} \approx 100$ days. This is represented by $s_p = s_{pt} = 0.1\ d^{-1}$, and $s_t = 0.005\ d^{-1}$. Small fractions of material deposited in ET_2, BB and bb are absorbed. Fractional absorption is substantial only in AI.

- "S" - slow translocation, 0.1% with $t_{\frac{1}{2}} \approx 10$ minutes and 99.9% with $t_{\frac{1}{2}} \approx 7000$ days. This is represented by $s_p = 0.1$, $s_{pt} = 99.9\ d^{-1}$, and $s_t = 0.0001\ d^{-1}$. Fractional absorption is very low, and it occurs predominantly from material deposited in AI.

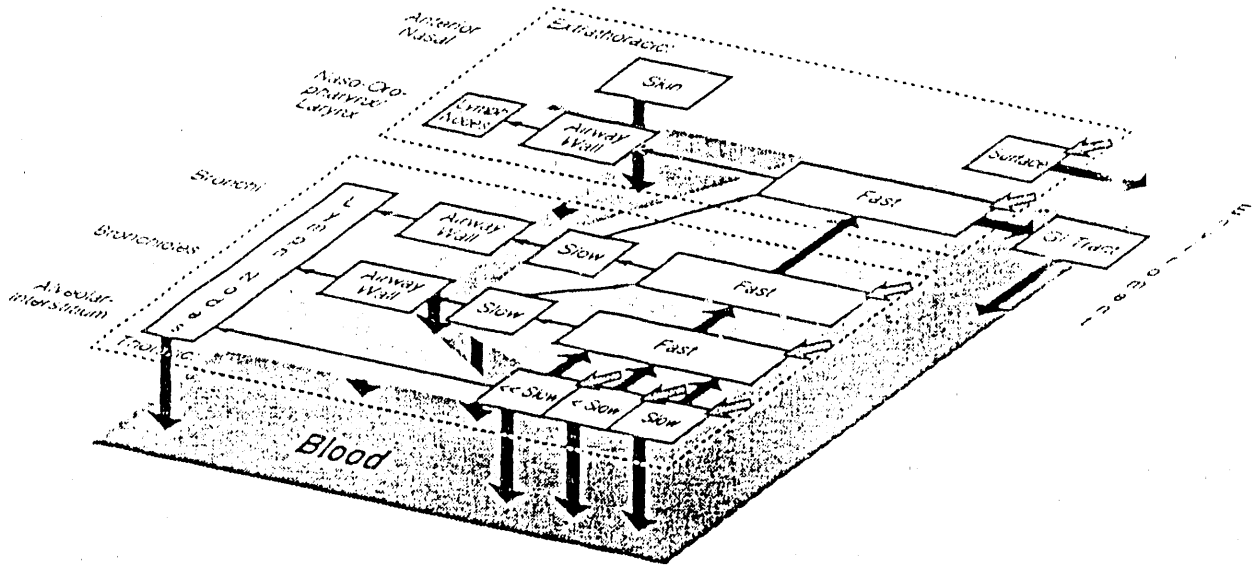

FIGURE 2. The Task Group's Combined Model of Particle Clearance and Absorption of Radionuclides into the Blood. The same "absorption function" is applied to all compartments of the clearance model, except to the compartment representing material on the surface of skin in the anterior nasal passages.

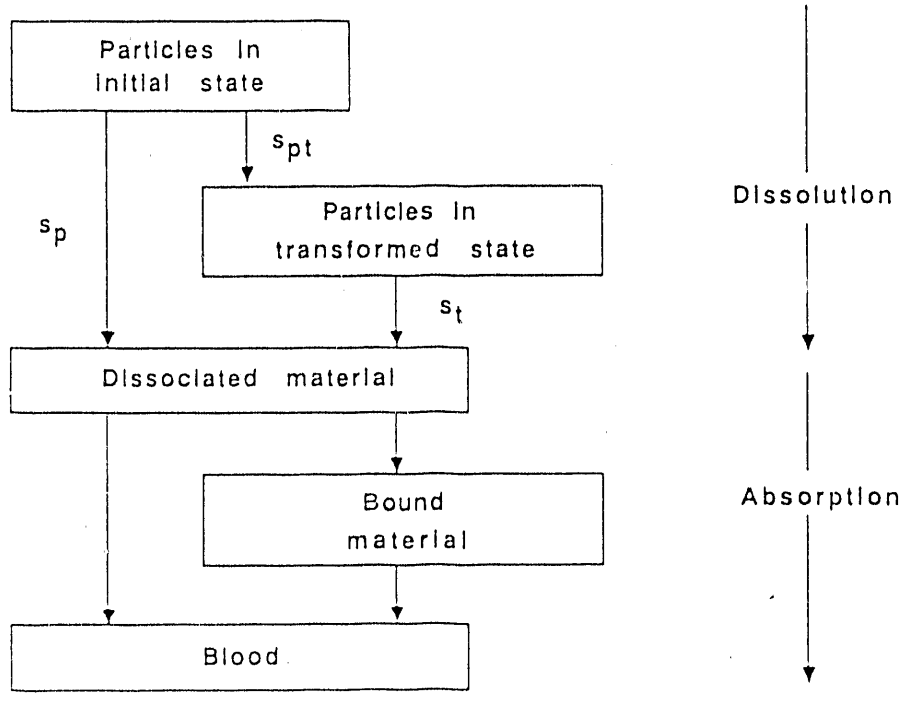

FIGURE 3. The Task Group's Model of Translocation to Blood. A single rate constant, or combinations of compartments clearing at constant rates (as appropriate), are used to represent the "absorption function" referred to in Figure 2.

Mineral sand particles deposited in the respiratory tract are expected to release all of the long-lived radionuclides present in the natural thorium decay series into the blood at rates similar to the default type "S" material. Since the translocation rates from mineral sand aerosols that arise under working conditions have not been measured (in man or experimental animals), to evaluate doses below we consider an order of magnitude range of uncertainty in the value of the dissolution rate s_t (from 0.0001 d^{-1} to 0.001 d^{-1}). To assess doses from exposure to airborne thoron progeny, the translocation rate of Pb-212 and Bj-212 deposited in the respiratory tract is represented by s_p = 0.00116 d^{-1}. This corresponds to the half-time of about 10 hours measured for uptake of Pb-212 from the respiratory tract of man (Booker et al. 1969; Hursh and Mercer 1970).

Inhalability of Large Particles

The task group model incorporates recent data on the inhalability of large particles (such as those present in airborne monazite dust). For particles with aerodynamic diameter larger than about 5 μm, the efficiency of the nose or mouth as particle samplers may be significantly less than unity (Figure 4), although this reduced inhalability does not apply for subjects exposed to very large particles in fast-moving air (Vincent et al. 1990). The task group has adopted the algebraic expression shown in Figure 4 to evaluate the intake efficiency of the nose or mouth as a function of particle aerodynamic diameter, and to correct this in situations that involve exposure to large particles at high wind speeds.

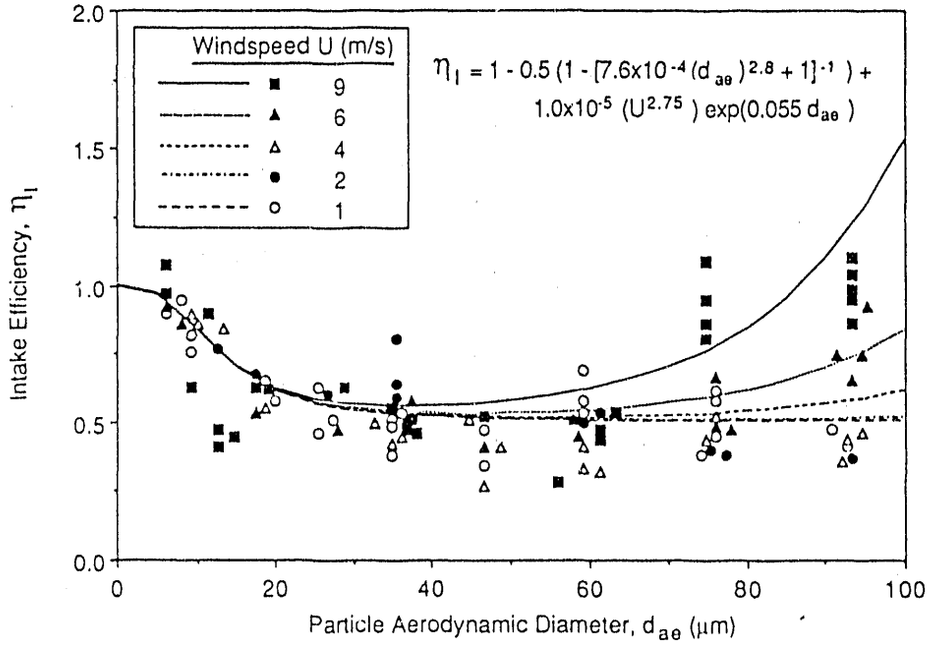

FIGURE 4. Aspiration Efficiency of the Human Head at Random Orientation to Moving Air. The data were obtained by Vincent et al. (1990) using a mannequin placed at various angles in a large wind tunnel. The curves show the representative empirical function adopted by the task group (for various windspeeds).

Regional Deposition

The task group's model of aerosol deposition in each anatomical region of the respiratory tract is based on a review of experimental data from human subjects. The model extrapolates the data to encompass the broader range of aerosol conditions and physiological factors involved in assessing doses for workers and for members of the public (of all ages). The task group has developed algebraic formulae to evaluate regional deposition in different subjects in terms of aerosol size, breathing rates, standard lung volumes, and scaling factors for airway dimensions (James et al. 1991a). Deposition in the extrathoracic regions (ET_1 and ET_2) is modeled empirically from the human data (Stahlhofen et al. 1989). To evaluate regional deposition in the thorax, the task group has applied a detailed theoretical model of gas transport and aerosol deposition in relation to airway anatomy (Egan et al. 1989). The model predicts accurately the experimental data on total deposition of aerosol particles in relation to particle size and breathing rate in human subjects. The model also predicts satisfactorily the variation of regional deposition with particle size and breathing rate measured in eight subjects who were intensively studied by Stahlhofen et al. (1980 and 1983) and by Stahlhofen (1984). However, in common with earlier theoretical models, for particles larger than about 1 μm (in the aerodynamic size-range), the task group's model predicts significantly less deposition in the tracheobronchial and alveolar-interstitial regions of the lung than the median experimental values published by Lippmann (1977), and Chan and Lippmann (1980), from their studies of a large group of subjects.

In view of these current inconsistencies between the principal experimental data, the task group has taken the conservative approach for dosimetry of adjusting the model to yield deposition values that fit the higher tracheobronchial and alveolar-interstitial deposition efficiencies measured by Lippmann and his co-workers. The effects of this adjustment are shown in Figures 5 and 6, where the theoretical values (denoted by $\Psi_{ae} = 1$) and adjusted values (denoted by $\Psi_{ae} = 3$) are compared with the published data.

These data represent the deposition efficiencies in terms of the respective fractions of particles of uniform size that deposit in each region after entering the trachea. Figure 7 shows the overall deposition in each region of the respiratory tract (ET_1, ET_2, BB, bb and AI) that is given by the task group's model (with $\Psi_{ae} = 3$), when the effects of nasal deposition, lognormal dispersion in the aerosol particle size, and inhalability of particles are included. These curves express regional deposition as a fraction of the amount of activity present in the volume of ambient air that is inhaled. The average inhalation rate of 1.5 m^3/h applies to a male manual worker who is laboring at 32% of the maximal work load for the average adult male (Roy and Courtay 1991). The corresponding values of regional deposition as a function of aerosol size, that are recommended to assess doses for a male manual worker who breathes normally through the nose, are given in Table 3. The values that apply for a worker who is a habitual mouth breather are given in Table 4 (Miller et al. 1988; James et al. 1991a).

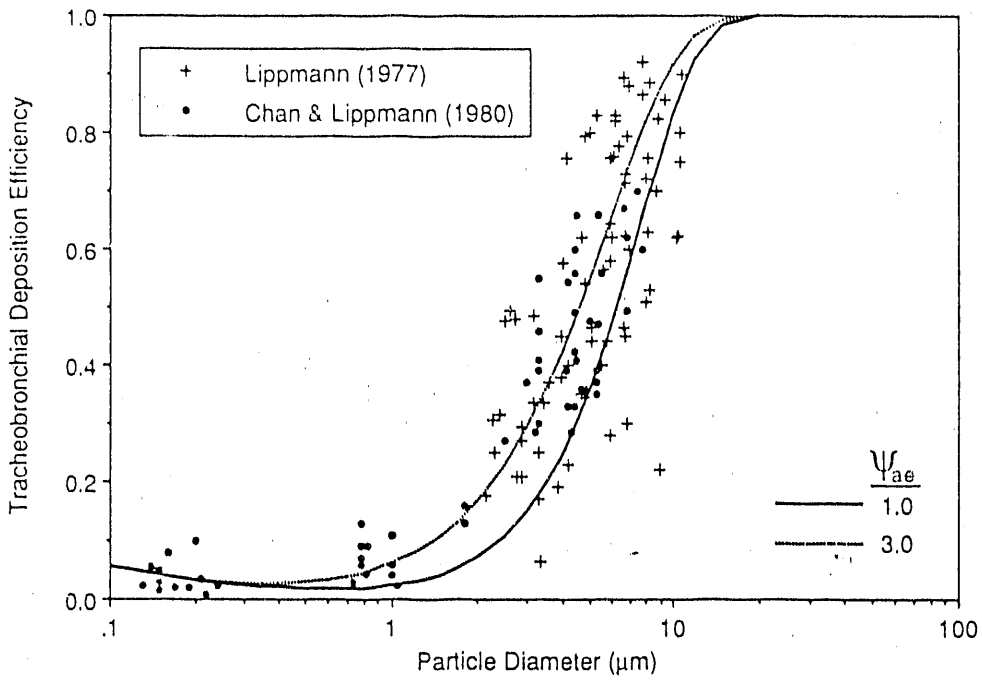

FIGURE 5. Comparison of the Tracheobronchial Deposition Efficiency Given by the Task Group's Theoretical Model with Experimental Data for Human Subjects. Values predicted directly by the model are shown as a solid curve (denoted by $\Psi_{ae} = 1$). Adjusted values obtained by setting $\Psi_{ae} = 3$ are shown by the broken curve.

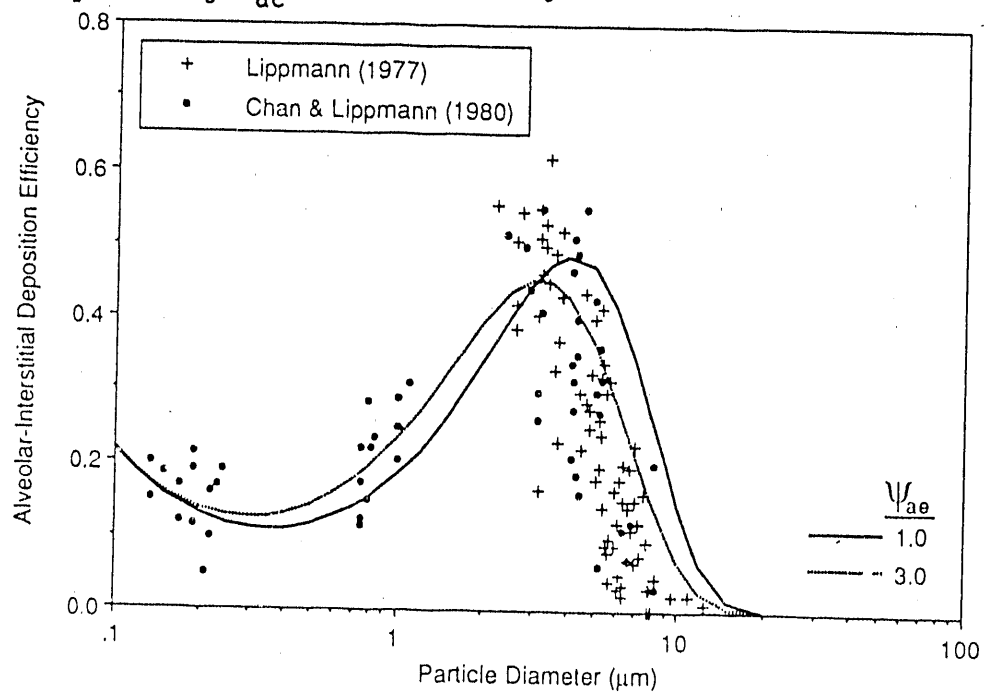

FIGURE 6. Comparison with Experimental Data of Alveolar-Interstitial Deposition Expressed as a Fraction of Activity Entering the Trachea Predicted by the Task Group Model. Details as in Figure 5.

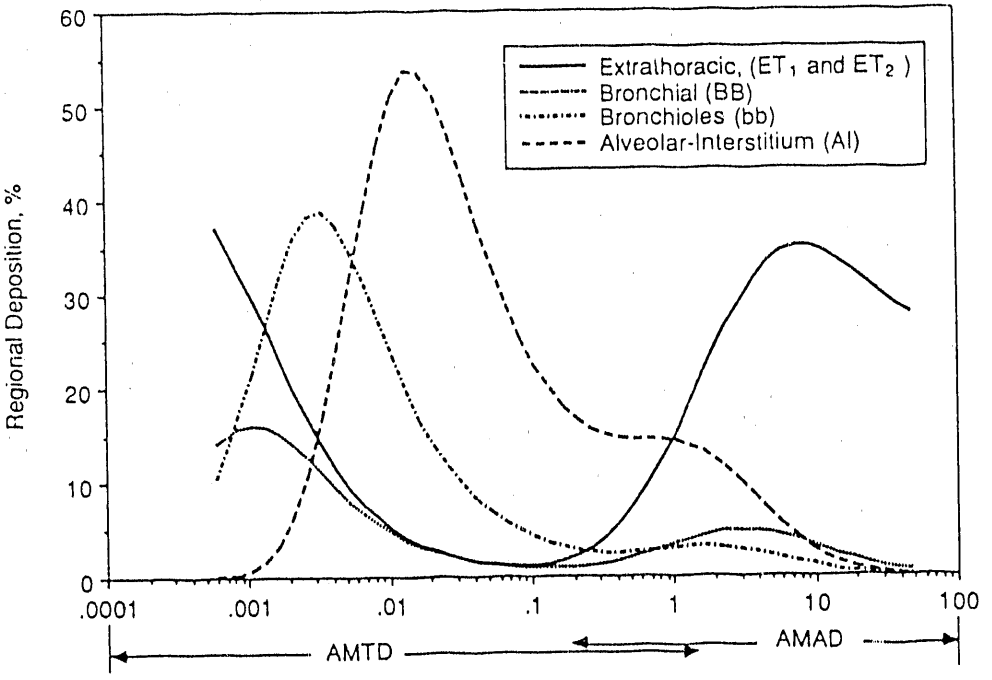

FIGURE 7. Task Group Model of Regional Deposition for a Male Manual Worker (Breathing at 1.5 m^3/h)

TABLE 3. Comparison of the Task Group's Model of Regional Deposition for a Male Manual Worker (B = 1.5 m^3/h) with the ICRP 30 Model.

AMD[a] (μm)	Regional Deposition (%)						
	Task Group					ICRP 30	
	ET[b]	BB	bb	AI	N-P	T-B	P
0.001	61.0	16.0	20.0	0.5	-	-	-
0.01	11.0	5.0	25.0	50.0	-	-	-
0.1	2.1	1.0	4.5	23.0	10.0	16.0	60.0
0.2	3.9	0.87	2.9	16.0	5.0	8.0	50.0
0.5	14.0	1.7	2.3	13.0	16.0	8.0	35.0
1.0	29.0	3.0	2.7	13.0	30.0	8.0	25.0
2.0	48.0	4.3	2.3	11.0	50.0	8.0	17.0
5.0	67.0	4.4	2.1	6.1	75.0	8.0	9.0
10.0	70.0	3.2	1.1	2.5	88.0	8.0	5.0
15.0	67.0	2.2	0.62	1.3	92.0	4.5	2.5
20.0	64.0	1.6	0.38	0.71	95.0	3.0	2.0
30.0	60.0	0.97	0.17	0.28	100.0	0.0	0.0
50.0	55.0	0.44	0.050	0.069	100.0	0.0	0.0

(a) For AMD $\leq$ 0.5 μm, deposition values in the task group model relate to activity median thermodynamic diameter (AMTD). All other values relate to activity median aerodynamic diameter (AMAD).

(b) Total deposition in ET includes equal amounts in ET_1 and ET_2 for a normal nose-breathing subject.

TABLE 4. Regional Deposition Given by the Task Group's Model for a Male Manual Worker Who is a Habitual Mouth Breather

AMD (μm)	Regional Deposition (%)				
	ET_1	ET_2	BB	bb	AI
0.001	13.0	37.0	21.0	26.0	0.61
0.01	2.3	5.8	5.1	26.0	51.0
0.1	0.40	0.91	0.97	4.5	23.0
0.2	0.47	0.78	0.96	2.8	16.0
0.5	1.5	1.9	2.7	3.0	16.0
1.0	3.5	5.0	6.3	4.4	18.0
2.0	6.8	12.0	12.0	6.1	19.0
5.0	11.0	26.0	16.0	5.9	13.0
10.0	12.0	37.0	14.0	3.7	6.7
15.0	12.0	41.0	10.0	2.3	3.7
20.0	12.0	42.0	7.7	1.5	2.2
30.0	12.0	43.0	4.6	0.72	0.94
50.0	11.0	42.0	2.0	0.23	0.26

The task group recommends simple algebraic formulae (James et al. 1991a) to generate values of regional deposition, both for workers and for the population at large (men, women, children and infants), in terms of breathing rates and lung volumes, and the activity median aerodynamic or thermodynamic diameter of the aerosols to which they are exposed. For equivalent levels of physical exertion, i.e., for sleep, rest, and light and heavy exercise, respectively (where appropriate), it is found that regional deposition predicted for these other subjects is similar to that for the reference adult male.

Dosimetry

The calculation of doses follows the method of ICRP 30 (ICRP 1979a), in which the committed dose equivalent, $H_{50,T}$, in a target tissue is determined by the energy absorbed per unit mass from the radiation emitted by each source within the respiratory tract, and in other organs of the body (James et al. 1991b). The total value of $H_{50,T}$ in any target tissue from the intake of a radionuclide is obtained by adding all contributions to $H_{50,T}$ from the radioactive transformations in all sources:

$$H_{50,T} = \sum_S U_S \, SEE(T \leftarrow S) \, Sv \qquad (1)$$

In Equation (1):

U_S is the total number of radioactive decays of the radionuclide over 50 yr in each source $SEE(T \leftarrow S)$ (in J kg^{-1}) is the specific effective energy absorbed in T per transformation in S.

$SEE(T \leftarrow S)$ is obtained by adding the contributions from all types of radiation, i, emitted by the radionuclide:

$$SEE(T \leftarrow S) = 1.6 \times 10^{-13} \sum_i \frac{Y_i E_i Q_i AF(T \leftarrow S)_i}{M_T} \; J \; kg^{-1} \; transformation^{-1} \qquad (2)$$

In Equation (2):

Y_i is the yield of radiation i per transformation of the radionuclide
E_i is the energy (in MeV) of the radiation
Q_i is the quality factor
$AF(T \leftarrow S)_i$ is the average fraction of energy absorbed in T per emission of radiation i in source S
M_T (in kg) is the mass of target tissue in T
1.6×10^{-13} is the number of joules in 1 MeV.

For intake of mineral sand (which contains a series of radionuclides) and the short-lived radon or thoron progeny, Equation (1) for the committed dose equivalent is modified to include contributions from all radionuclides j:

$$H_{50,T} = \sum_j \sum_S [U_S \sum_i SEE(T \leftarrow S)_i]_j \quad Sv \qquad (3)$$

The task group has calculated the absorbed fractions, $AF(T \leftarrow S)_i$, as continuous functions of radiation energy, E_i, for α and β particles, taking into account each combination of radioactive source and target tissue in the respiratory tract. For example, the functions calculated to represent $AF(T \leftarrow S)_i$ for α-emitting sources in the bronchial (BB) region are shown in Figure 8. As for regional deposition, the task group gives simple algebraic formulae to evaluate all absorbed fractions for short-range radiations.

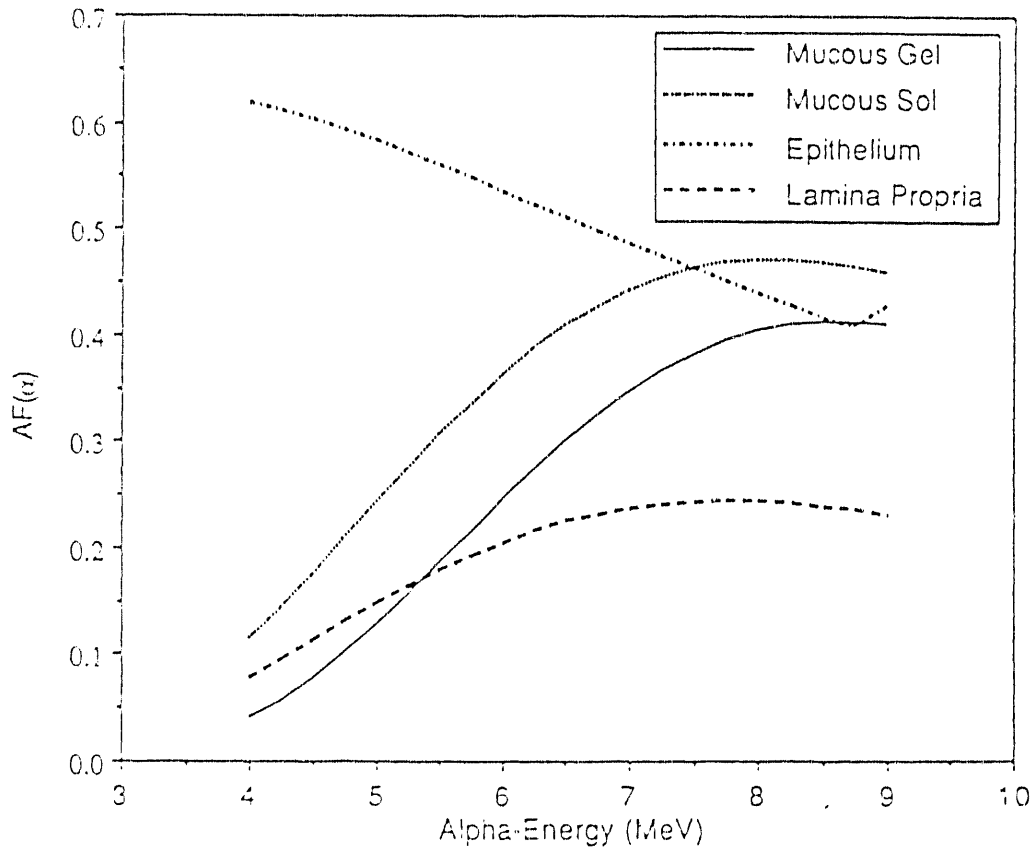

FIGURE 8. Absorbed Fractions for α-Emissions in the Bronchial Region (BB). Curves are shown for emissions in the "Mucous Gel" (representing "Fast" mucus in Figure 2), the "Mucous Sol" (representing "Slow" mucus), "Epithelium" (radionuclides chemically bound in the "Airway Wall"), and "Lamina Propria" (particles sequestered by macrophages in the "Airway Wall").

To obtain the dosimetric results given below, the task group's deposition, clearance and dosimetry models were solved for the natural thorium decay series using the integrated computer program described by Birchall et al. (1991). Weighted committed dose equivalents are obtained by applying the tissue weighting factors given earlier (in Tables 1 and 2) to $H_{50,T}$ calculated for tissues in the respiratory tract and other organs of the body. The quality factor Q_i is taken to be 20 for α-radiation and unity for ß-radiation (ICRP 1977).

IMPLICATIONS FOR DOSIMETRY OF INHALED MINERAL SAND

In this section we use the proposed task group model to evaluate the weighted dose equivalent integrated over a period of 50 years after a nominal intake by inhalation of 1 Bq α-activity in the form of mineral sand (natural thorium ore). We assume that all of the progeny of Th-232 are in radioactive equilibrium with the parent, and effectively remain in equilibrium within the particles that are deposited in the respiratory tract (Johnson 1985). Translocation and uptake of the relatively small fraction of Th-232 and Th-228 that is dissolved from ore particles while they are retained in the lung gives rise to most of the dose received by body organs. It is assumed that the shorter-lived progeny grow into radioactive equilibrium once the thorium isotopes are deposited in the skeleton and other body organs. We assume that Th-232 and Th-228 are bone surface seekers, and use the model for thorium uptake and retention in the skeleton and the rest of the body given in ICRP 30 (ICRP 1979a).

Weighted Committed Dose Equivalent

Figure 9 compares weighted committed dose equivalents from exposure by inhalation to monazite sand that are given by the proposed task group model with those given by ICRP 30 Class "Y" (as respective functions of aerosol size). It is seen that the new model gives dose conversions that are broadly similar to those derived using the ICRP 30 model (Johnson 1985). They coincide at 1-μm AMAD, and for aerosols between 15-μm and 20-μm AMAD. However, for smaller and larger aerosol sizes, the proposed model gives lower dose conversion coefficients. The marked reduction for very large aerosols arises from the combined effects of decreased particle inhalability and increased fractional deposition in the extrathoracic airways. In the new model, there is very little translocation of radionuclides from slowly dissolving particles while they are being cleared rapidly through the extrathoracic airways, whereas for Class "Y" ICRP 30 takes a fixed fraction (1%) of material deposited in the nasopharynx to be absorbed into the blood.

It is also seen from Figure 9 that the proposed model gives somewhat higher values of weighted dose equivalent per unit intake than the ICRP 30 model for aerosol sizes over the respirable range from 1-μm to 10-μm AMAD. The new values are largely independent of the actual solubility rate of the monazite sand particles, at least over the assumed range from 0.0001 d^{-1} (applicable for type "S" material) to 0.001 d^{-1} (applicable for material with solubility intermediate between types "S" and "M").

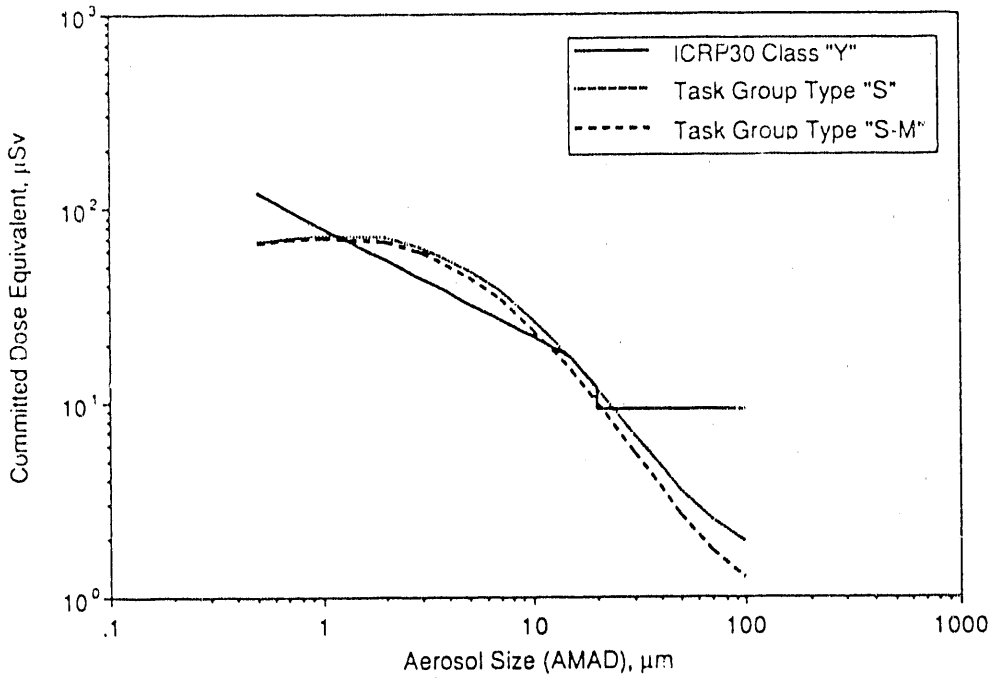

FIGURE 9. Weighted Dose Equivalent Committed Over 50 Years Following Exposure by Inhalation to 1 Bq α-Activity in Monazite Sand. Values calculated using the proposed lung model (for two different assumed solubility rates) are compared with ICRP 30 Class "Y."

Derived Air Concentration

The annual limit on intake (ALI) for inhalation of thorium ore (the Th-232 series) that is currently recommended by ICRP is 380 Bq of total α-activity. This limit is based upon the limitation of non-stochastic effects on bone surfaces, i.e., the non-stochastic limit for dose equivalent of 500 mSv/y that is committed to be received by bone surfaces. The corresponding derived air concentration (DAC) is 0.16 α-Bq/m^3 (ICRP 1986). In their draft recommendations, ICRP propose to use limitation of stochastic risk (and thus the total weighted committed dose equivalent) as the principal basis for protection standards (ICRP 1990). Table 5 gives values of the DAC that are calculated using the task group's lung model on the basis of the current stochastic dose limit of 50 mSv/y. These are generally higher than the DAC for a 1-μm AMAD natural thorium aerosol given by ICRP (1986), but not significantly so if the proposed model is applied to assess dose for a habitual mouth-breather. Values of the DAC would, of course, become proportionally more restrictive with any recommended reduction in the annual dose limit (ICRP 1990).

TABLE 5. Derived Air Concentrations for Monazite Sand, Based on the Stochastic Dose Limit of 50 mSv/y

AMAD (μm)	Normal Nose-Breather		Habitual Mouth-Breather	
	Type "S"	Type "S-M"	Type "S"	Type "S-M"
1	0.23	0.24	0.15	0.16
5	0.34	0.37	0.13	0.14
10	0.62	0.69	0.18	0.20
15	1.0	1.1	0.28	0.31
20	1.4	1.7	0.39	0.44

Tissues at Risk

Figure 10 shows the proportions of the weighted dose equivalent that are contributed by tissues in the respiratory tract and other organs of the body (for the latter, only the bone surface and red bone marrow). In the proposed model, the weighted dose equivalent (i.e., risk) is calculated to arise predominantly from irradiation of the respiratory tract, even if the solubility rate of monazite sand is assumed to be as high as 0.001 d^{-1} (intermediate between types "S" and "M" material). For aerosol sizes larger than 5-μm AMAD, about 85% of the risk of respiratory tract cancer is associated with the bronchi. The risks of bone cancer and leukemia are predicted to be relatively low, and about equal. In contrast, the ICRP 30 Class "Y" model (with ICRP 26 risk factors) predicts a risk of bone cancer almost as high as that for lung cancer. This arises partly as a result of the relatively high fraction of material transferred to blood in the Class "Y" model, and partly because of the weighting factor of 0.03 applied in ICRP 30 for bone surface dose (c.f. the proposed new value of 0.01 given in Table 2).

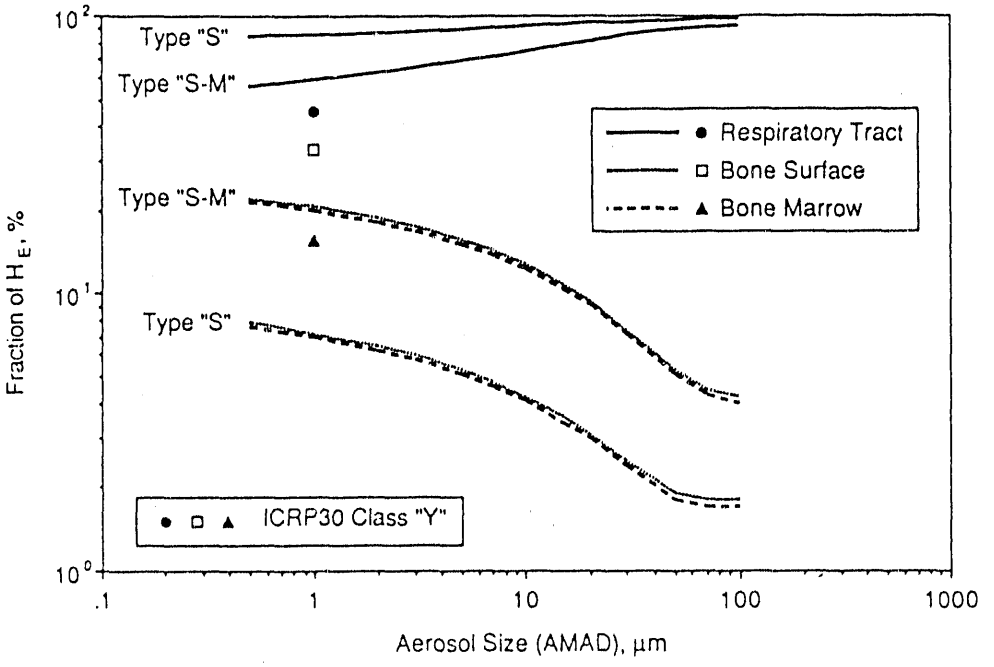

FIGURE 10. Tissue Contributions to Weighted Dose Equivalent Committed as a Result of Exposure by Inhalation of Monazite Sand. The symbols relate to a 1 μm AMAD aerosol and the ICRP 30 Class "Y" model (Johnson 1985). The curves show values given by the task group model for two different solubility rates.

Chronic Versus Acute Intake

Figure 11 compares the committed weighted dose equivalent that is calculated using the task group model for chronic exposure to monazite sand over a 50-year period with the weighted dose equivalent actually received. It is seen that for aerosols with AMAD of about 1 μm, the actual 50-year dose is approximately 70% of the committed value. However, for aerosols with AMAD of around 15 μm, the committed and actual weighted dose equivalents are similar. This is because the weighted dose is contributed mainly by material deposited in the bronchial and bronchiolar regions, and this is not subject to very long retention. Therefore, the dose is not protracted significantly beyond the year of exposure.

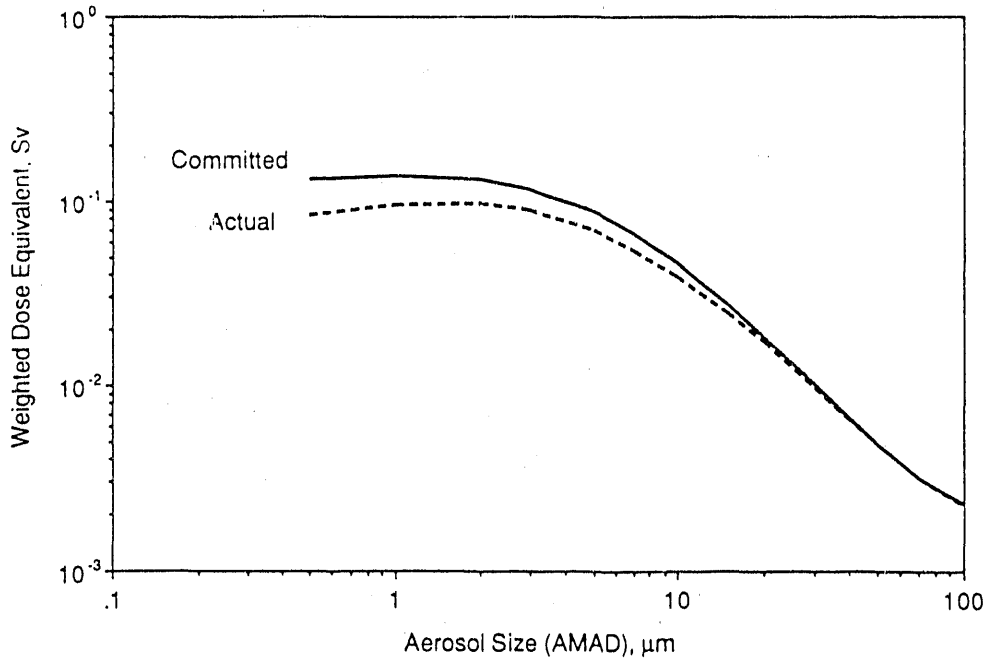

FIGURE 11. Comparison of Lifetime and Committed Weighted Dose Equivalents from 50-Year Chronic Occupational Exposure to Monazite Sand. The average rate of intake is taken to be 0.1 α-Bq/d (approximately 1/10 the ALI given in ICRP Publication 47).

Urinary Excretion

The amount of Th-232 excreted daily in urine as a function of time from the start of a continuous intake of mineral sand at the average rate of 1-Bq Th-232 α-activity per day is shown in Figure 12. Values calculated using the task group lung model with the excretion function for thorium given in ICRP Publication 54 (ICRP 1988) are compared here with daily excretion calculated using the ICRP 30 model. According to both models, urinary excretion of Th-232 at this relatively high rate of intake will not be detectable until many years of exposure have elapsed, where the minimum detectable activity (MDA) is assumed to be 0.014 Bq/d (Hewson and Hartley 1990). Furthermore, the task group model predicts more than an order of magnitude less activity excreted in urine during the early stages of chronic exposure than the ICRP 30 model.

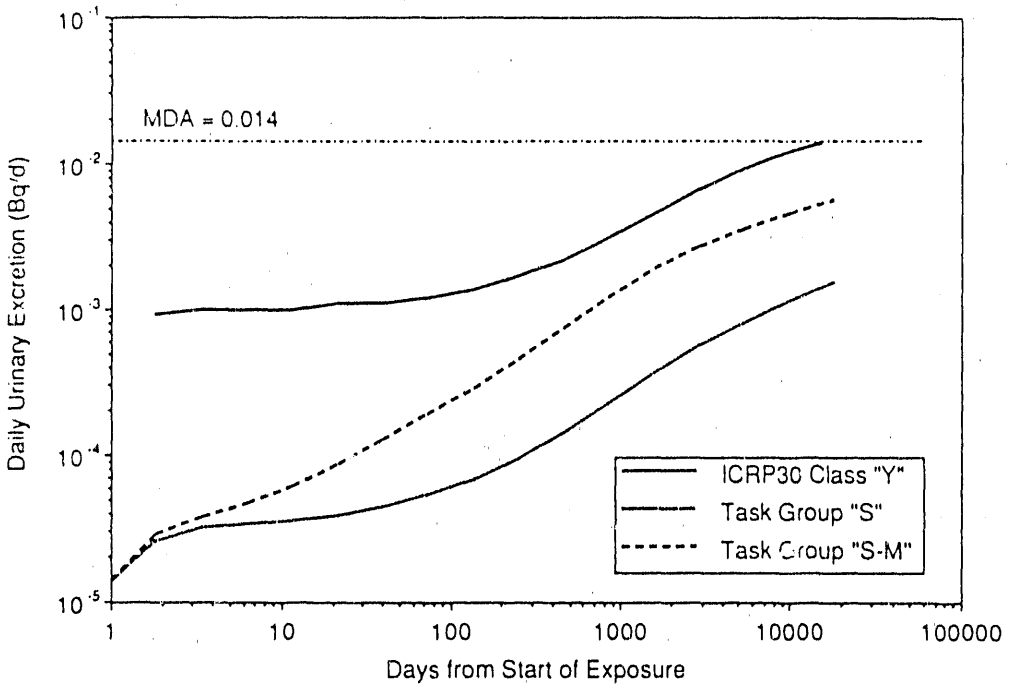

FIGURE 12. Daily Urinary Excretion of Th-232 from Chronic Intake of 1 Bq/d Th-232 Activity in Mineral Sand. This rate of intake corresponds to about 6 times the ALI given in ICRP Publication 47). In this example, the aerosol AMAD is assumed to be 10 μm. The minimum detectable activity (MDA) is indicated for comparison as 0.014 Bq/d.

Thoron in Breath

Figure 13 compares the accumulation of Th-232 activity in the lungs of a worker who is subject to chronic intake of 1 α-Bq/d of mineral sand, that is predicted by the task group model, with the minimum detectable activity based on measurement of thoron in breath (Johnson and Peterman 1984). This rate of intake corresponds to the current ALI for natural thorium given in ICRP Publication 47 (ICRP 1986). It is seen that exposure at this rate is expected to be detected after about 1 month in the case of a 1 μm AMAD aerosol, and after 6 months if the aerosol AMAD is 10 μm. In the longer term, the rate at which thorium is released from sand particles retained in the lung is expected to affect the sensitivity of this detection technique. The figure shows two curves for each aerosol size: $s_p = 0.0001\ d^{-1}$ represents material of translocation type "S" and $s_p = 0.001\ d^{-1}$ material of translocation type intermediate between "S" and "M."

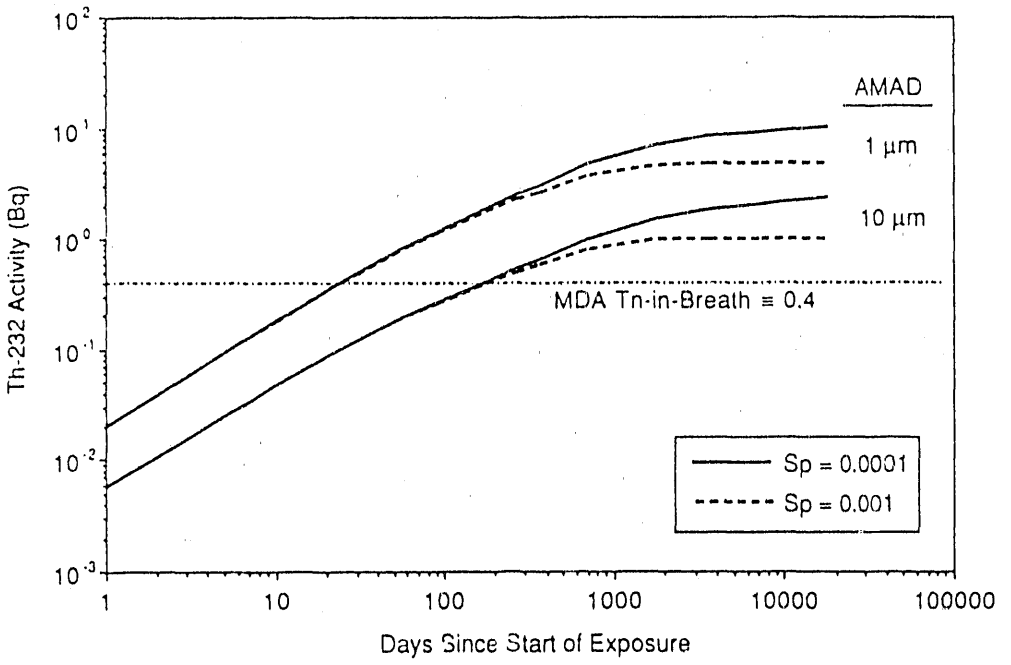

FIGURE 13. Accumulation of Th-232 Activity in the Lungs from Chronic Intake of 1 α-Bq/d Mineral Sand. The minimum detectable activity (MDA) based on measurement of thoron exhaled in the breath is shown for comparison as 0.4-Bq Th-232 activity.

IMPLICATIONS FOR DOSIMETRY OF THORON (AND RADON) PROGENY

In this final section we use the proposed lung model to calculate the weighted dose equivalent from unit exposure to the short-lived thoron or radon progeny. We consider exposure in terms of potential α-energy (ICRP 1981), which is defined as the α-energy released by total decay of the radon progeny through Po-214, or the thoron progeny through Po-212. We express exposure to potential α-energy in the familiar unit Working Level Month (WLM), where one Working Level (WL) is any combination of short-lived radon (or thoron) progeny in 1 liter of air that will result in the emission of 1.3×10^5 MeV of potential α-energy, and 1 WLM corresponds to exposure at an airborne concentration of 1 WL for a working month of 170 hours. ICRP did not use the dosimetric model of ICRP 30 to calculate dose-exposure relationships for radon and thoron progeny. Instead, ICRP 32 (ICRP 1981) adopted the dosimetric model developed for the Nuclear Energy Agency by a group of experts (NEA 1983). The currently recommended dose-exposure conversion coefficients for occupational exposures are based largely on the NEA dosimetry model. These are 10 mSv/WLM for radon progeny and 3.5 mSv/WLM for thoron progeny (ICRP 1986).

Weighted Dose Equivalent per WLM Exposure

Figure 14 shows the weighted dose equivalent calculated as a function of aerosol size for 1-WLM exposure of a male manual worker to radon or thoron progeny. Values are shown with and without adjustment of the task group's deposition model by the factor $\Psi_{ae} = 3$ (see the earlier Figure 5 and accompanying text). The symbols shown in the figure for radon and thoron progeny

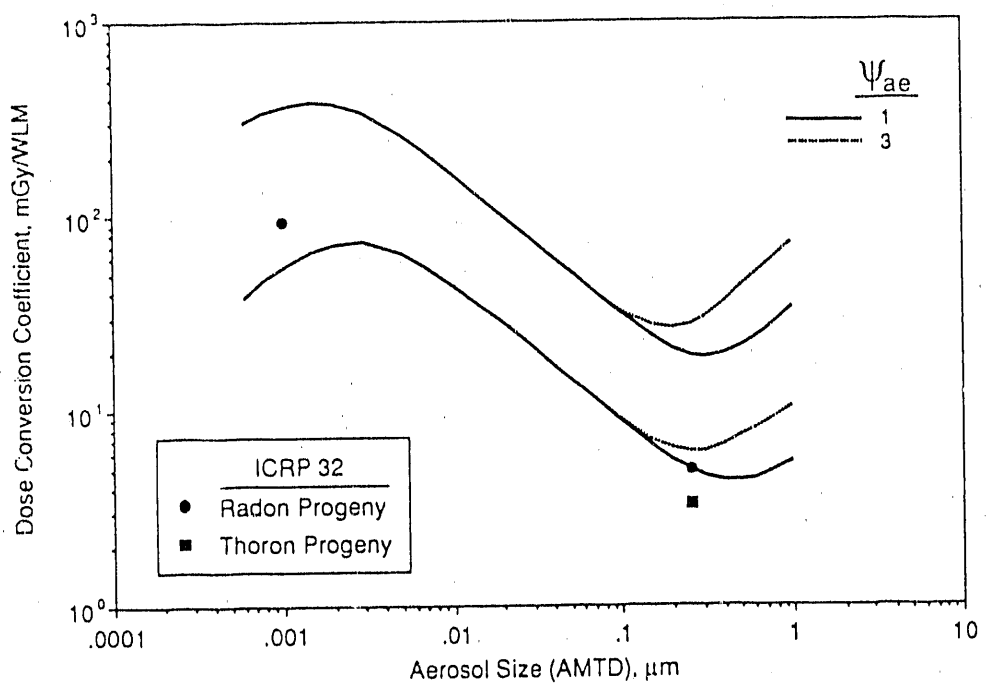

FIGURE 14. Comparison of Weighted Dose Equivalent per WLM Exposure to Radon or Thoron Progeny Calculated Using the Task Group Model with Values Assumed in ICRP 32.

at an aerosol size of 0.25 μm indicate the conversion coefficients recommended in ICRP 32 for the so-called "attached" fractions of potential α-energy. The symbol shown only for radon progeny at an aerosol size of 0.001 μm indicates the conversion coefficient recommended in ICRP 32 for the "unattached" fraction of potential α-energy. It is seen from Figure 14 that the proposed task group model gives dose conversion coefficients for radon progeny at equivalent aerosol sizes that are three to five-fold higher than ICRP 32, but values for thoron progeny that are only 30% to 80% higher.

The marked increase in the dose conversion coefficient given by the task group model over ICRP 32 arises from several factors:

- the weighting factor assigned to bronchial dose is 0.096, with an additional weight of 0.018 applied to bronchiolar dose, rather than 0.06 applied to the average dose calculated for the bronchial-bronchiolar region as a whole in ICRP 32
- the calculated dose is weighted to that received by secretory cells rather than only the deep-lying basal cells considered in ICRP 32
- higher doses are calculated for the bronchi as a separate region rather than the composite bronchial and bronchiolar region considered in ICRP Publication 32
- the deposition model takes into account recent experimental data that indicate enhanced deposition of sub-micron-sized particles (Cohen et al. 1990).

However, since the adjustment factor Ψ_{ae} was introduced in the deposition model as a moderately conservative measure, the task group will recommend that this is not applied in the calculation of doses from exposure to radon and thoron progeny. It can be shown that the correspondingly "conservative" value of the dose conversion coefficient given for radon progeny if Ψ_{ae} is increased to 3 implies a higher risk of bronchial cancer than that currently indicated by epidemiological studies of underground miners (Jacobi 1989). The task group will recommend the dose conversion coefficients for exposure of manual workers to radon and thoron progeny in dusty environments given in Table 6. The table also shows the assumed values of unattached fraction of potential α-energy and activity median thermodynamic diameter (AMTD) of the attached radon and thoron progeny aerosols on which they are based (NRC 1990).

In this table, the values of AMTD represent the median size attained by progeny aerosols within the respiratory tract. It is assumed that these aerosols are partly hygroscopic and grow rapidly to double their size in ambient air. D_u and D_a refer to the partial dose conversion coefficients for unattached and attached progeny, respectively. It is seen from Table 6 that the weighted net dose equivalent per unit exposure to thoron progeny potential α-energy (and thus the risk per unit exposure) is predicted by the task group model to be only about one-sixth of that for exposure to radon progeny.

TABLE 6. Assumed Aerosol Characteristics for Radon and Thoron Progeny in Dusty Occupational Environments and the Corresponding Dose Conversion Coefficients Given by the Task Group Model for Exposure to Potential α-Energy

		Radon Progeny	Thoron Progeny
Unattached Fraction:	f_p	2%	0.5%
	AMTD	0.0011 μm	0.0011 μm
	D_u	370 mSv/WLM	57 mSv/WLM
Attached Fraction:	AMTD	0.5 μm	0.5 μm
	D_a	21 mSv/WLM	4.2 mSv/WLM
Net Conversion Coefficient:		30 mSv/WLM	4.5 mSv/WLM

Tissues at Risk

Figure 15 shows the fractions of the weighted dose equivalent contributed by the different regions of the respiratory tract and other body organs that receive the highest doses from exposure to thoron progeny. It is seen that bronchial dose predominates over the entire range of aerosol size (from unattached progeny through to a large attached aerosol of 1-μm AMTD), followed by the weighted dose received by the bronchioles. The kidneys receive the next highest weighted dose. Kidney dose arises principally from uptake of the Bi-212 produced by decay of Pb-212 while this is temporarily retained in circulating blood. The half-time of 10 hours that is assumed to represent absorption of lead and bismuth from the lung effectively prevents the absorption of Bi-212 directly from the lung. In combination with the metabolic models for Pb-212 and bismuth given in ICRP 30 (ICRP 1979b), the task group

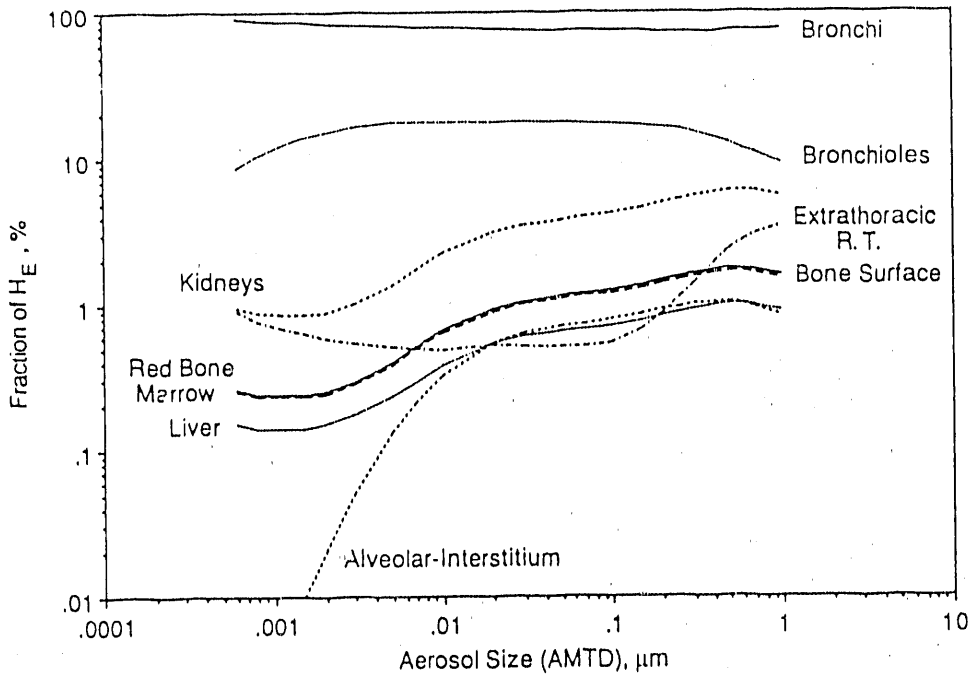

FIGURE 15. Tissue Contributions tc Weighted Dose Equivalent from Exposure to Thoron Progeny

model predicts that weighted doses received by red bone marrow, bone surfaces and the liver make insignificant contributions to the overall risk.

CONCLUSIONS

In summary, we conclude from this study that:

- The proposed model gives marginally higher values of weighted dose equivalent per unit intake of monazite sand for aerosol sizes in the respirable range between 1-μm and 10-μm AMAD than does ICRP 30.
- For larger-sized aerosols, the new model is less restrictive than ICRP 30.
- The DAC is about four-fold higher for a 15-μm AMAD aerosol than it is for 1-μm AMAD.
- The relaxation of the DAC with increasing aerosol size is substantially less for habitual mouth-breathers.
- The tissues at risk are predominantly the bronchial and bronchiolar epithelium, with relatively small risk of bone cancer and leukemia.
- The lifetime dose received from chronic exposure is not substantially overestimated by the 50-year committed dose.

- Urinary excretion of radiothorium cannot be used to monitor exposures to monazite sand.

- Measurement of thoron in breath is expected to detect chronic exposures at the rate of the current annual limit within a few months of starting the exposure.

- For exposure to radon progeny, the weighted dose equivalent given by the proposed model is about three-fold higher than that in ICRP 32, at about 30 mSv/WLM.

- The weighted dose equivalent given for exposure to thoron progeny is six-fold lower than that for radon progeny, but this is marginally higher than in ICRP 32.

- The risk to the kidneys and other organs of the body is insignificant compared with that for bronchial and bronchiolar epithelium.

- If the proposed model is adopted by the ICRP in conjunction with a lower primary limit on dose, it is likely to require more restrictive radiological protection practices in the mineral sands industry.

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REFERENCES

Bair, W. J. 1991. "Overview of the ICRP Respiratory Tract Model." Presented at the 3rd International Workshop on Respiratory Tract Dosimetry, July 1-3, Albuquerque, New Mexico. Radiat. Prot. Dosim. (in press).

Bailey, M. R. 1989. "Application of Human Volunteer Studies in Setting Exposure Limits." Radiat. Prot. Dosim. 26:249-257.

Bailey, M. R., A. Birchall, R. G. Cuddihy, A. C. James, and M. Roy. 1991. "Respiratory Tract Clearance Model for Dosimetry and Bioassay of Inhaled Radionuclides." Presented at the 3rd International Workshop on Respiratory Tract Dosimetry, July 1-3, Albuquerque, New Mexico. Radiat. Prot. Dosim. (in press).

Birchall, A. 1991. "A Computer Program for the Evaluation of Lung Dose." Presented at the 3rd International Workshop on Respiratory Tract Dosimetry, July 1-3, Albuquerque, New Mexico. Radiat. Prot. Dosim. (in press).

Booker, D. V., A. C. Chamberlain, D. Newton, and A. N. B. Stott. 1969. "Uptake of Radioactive Lead Following Inhalation and Injection." Br. J. Radiol. 42:457-466.

Chan, T. L., and M. Lippmann. 1980. "Experimental Measurements and Empirical Modeling of the Regional Deposition of Inhaled Particles in Humans." Am. Ind. Hyg. Assoc. J. 41:399-409.

Churg, A., and J. L. Wright. 1988. "Mineral Particles in Airway Walls in the Lung of Long-Term Chrysotile Miners." Ann. Occup. Hyg. 32(Suppl 1):173-180.

Cohen, B. S., R. G. Sussman, and M. Lippmann. 1990. "Ultrafine Particle Deposition in a Human Tracheobronchial Cast. Aerosol Sci. Technol. (in press).

Cuddihy, R. G. 1984. "Mathematical Models for Predicting Clearance of Inhaled Radioactive Substances." Pp. 167-179 in Lung Modelling for Inhalation of Radioactive Materials. H. Smith and G. Gerber, eds. Report EUR 9384, Luxembourg: Commission of the European Communities.

Cuddihy, R. G., and H. C. Yeh. 1988. "Respiratory Tract Clearance of Particles and Substances Dissociated from Particles." Pp. 169-188 in Inhalation Toxicology: The Design and Interpretation of Inhalation Studies and Their Use in Risk Assessment. U. Mohr, ed. Springer-Verlag, New York, New York.

Egan, M. J., W. Nixon, N. I. Robinson, A. C. James, and R. F. Phalen. 1989. "Inhaled Aerosol Transport and Deposition Calculations for the ICRP Task Group." J. Aerosol Sci. 20:1301-1304.

Hewson, G. S. 1989. "A Review of the Uncertainties in Internal Radiation Dose Assessment for Inhaled Thorium." Radiat. Prot. in Australia 7:35-44.

Hewson, G. S., and B. M. Hartley. 1990. "Radiation Research Priorities in the Mineral Sands Industry." Radiat. Prot. Dosim. in press.

Hursh, J. B., and T. T. Mercer. 1970. "Measurement of Pb-212 Loss Rate from Human Lungs." J. Appl. Physiol. 28:268-274.

International Commission on Radiological Protection (ICRP). 1977. Recommendations of the International Commission on Radiological Protection. ICRP Publication 26, Pergamon Press, Oxford.

International Commission on Radiological Protection (ICRP). 1979a. Limits for Intakes of Radionuclides by Workers. ICRP Publication 30 (Part 1), Pergamon Press, Oxford.

International Commission on Radiological Protection (ICRP). 1979b. Limits for Intakes of Radionuclides by Workers. ICRP Publication 30 (Part 2), Pergamon Press, Oxford.

International Commission on Radiological Protection (ICRP). 1981. Limits for Inhalation of Radon Daughters by Workers. ICRP Publication 32, Pergamon Press, Oxford.

International Commission on Radiological Protection (ICRP). 1986. Radiation Protection of Workers in Mines. ICRP Publication 47, Pergamon Press, Oxford.

International Commission on Radiological Protection (ICRP). 1988. Individual Monitoring for Intakes of Radionuclides by Workers: Design and Interpretation. ICRP Publication 54, Pergamon Press, Oxford.

Jacobi, W. 1989. "Radiation Exposure from Natural Sources: the Radon Problem." Presented at the Regional IRPA Conference, Visby/Gotland, Sweden, September 11-15, 1989 (proceedings in press).

James, A. C., W. Stahlhofen, G. Rudolf, M. J. Egan, W. Nixon, and J. K. Briant. 1991a. "The Respiratory Tract Deposition Model Proposed by the ICRP Task Group." Presented at the 3rd International Workshop on Respiratory Tract Dosimetry, July 1-3, Albuquerque, New Mexico. Radiat. Prot. Dosim. (in press).

James, A. C., P. Gehr, R. Masse, R. G. Cuddihy, F. T. Cross, A. Birchall, J. S. Durham, and J. K. Briant. 1991b. "Dosimetry Model for Bronchial and Extrathoracic Tissues of the Respiratory Tract." Presented at the 3rd International Workshop on Respiratory Tract Dosimetry, July 1-3, Albuquerque, New Mexico. Radiat. Prot. Dosim. (in press).

Johnson, J. R., and B. F. Peterman. 1984. "A Model to Describe Exhalation Following an Inhalation Exposure to Thorium Aerosols and its Relationship to Lung Dose for occupationally Exposed Persons." Pp. 193-197 in Lung Modelling for Inhalation of Radioactive Materials. H. Smith and G. Gerber, eds. Report EUR 9384, Luxembourg: Commission of the European Communities.

Johnson, J. R. 1985. "A Review of the Dosimetry from Inhalation of Long Lived Alpha Activity in Ore Dust." Pp. 495-502 in Occupational Radiation Safety in Mining. H. Stocker, ed. Canadian Nuclear Association, Toronto.

Kathren, R. L., D. J. Strom, J. F. McInroy, and R. E. Bistline. 1990. "Distribution of Plutonium and Americium in the Lungs and Lymph Nodes of United States Transuranium Registry Registrants and Relationship to Smoking Status." Health Phys. 58(Suppl 1):S48.

Lippmann, M. 1977. "Regional Deposition of Particles in the Human Respiratory Tract." Pp. 213-232 in Handbook of Physiology - Reaction to Environmental Agents. D. H. K. Lee, ed. American Physiology Society, Bethesda, Maryland.

Miller, F. J., T. B. Martonen, M. G. Ménarche, R. C. Graham, D. M. Spektor, and M. Lippmann. 1988. "Influence of Breathing Mode and Activity Level on Regional Deposition of Inhaled Particles and Implications for Regulatory Standards." Ann. occup. Hyg. 32(Suppl 1):3-10.

National Research Council (NRC). 1990. Report by a Scientific Panel on Dosimetric Assumptions Affecting the Application of BEIR IV Risk Estimates. National Academy Press, Washington, D.C.

Nuclear Energy Agency (NEA). 1983. Dosimetry Aspects of Exposure to Radon and Thoron Daughter Products. OECD Nuclear Energy Agency, Paris.

Patrick, G. 1989. "Requirements for Local Dosimetry and Risk Evaluation in Inhomogeneously Irradiated Lung." Pp. 269-277 in Low Dose Irradiation: Biological Bases of Risk Assessment. K. F. Baverstock and J. W. Stather, eds. Taylor and Francis, London.

Roy, M., and C. Courtay. 1991. "Daily Activities and Breathing Parameters for Use in Respiratory Tract Dosimetry." Radiat. Prot. Dosim. in press.

Snipes, M. B., B. B. Boecker, and R. O. McClellan. 1983. "Retention of Monodisperse or Polydisperse Aluminosilicate Particles Inhaled by Dogs, Rats, and Mice." Toxicol. and Appl. Pharmacol. 69:345-362.

Stahlhofen, W., J. Gebhart, and J. Heyder. 1980. "Experimental Determination of the Regional Deposition of Aerosol Particles in the Human Respiratory Tract." Am. Ind. Hyg. Assoc. J. 41:385-398.

Stahlhofen, W., J. Gebhart, J. Heyder, and G. Scheuch. 1983. "New Regional Deposition Data for the Human Respiratory Tract." J. Aerosol Sci. 14:186-188.

Stahlhofen, W. 1984. "Human Data on Deposition." Pp. 39-59 in Lung Modelling for Inhalation of Radioactive Materials. H. Smith and G. Gerber, eds. Report EUR 9384, Luxembourg: Commission of the European Communities.

Stahlhofen, W. 1989. "Human Lung Clearance Following Bolus Inhalation of Radioaerosols." Pp. 153-166 in Extrapolation of Dosimetric Relationships for Inhaled Particles and Gases. Academic Press, New York, New York.

Stahlhofen, W., G. Rudolf, and A. C. James. 1989. "Intercomparison of Experimental Regional Aerosol Deposition Data." J. Aerosol Med. 2:285-308.

Vincent, J. H., D. Mark, B. G. Miller, L. Armbruster, and T. L. Ogden. 1990. "Aerosol Inhalability at Higher Wind Speeds." J. Aerosol Sci. 21:577-586.

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