IMPLICATIONS OF THE DOSIMETRIC MODEL FOR THE RESPIRATORY SYSTEM ON LIMITS FOR INTAKES OF RADIONUCLIDES BY WORKERS (ICRP PUBLICATION 30)

DOUGLAS K. CRAIG,* JOSEPH D. BRAIN,† RICHARD G. CUDDIHY,‡ GEORGE KANAPILLY,§ ROBERT F. PHALEN,§ DAVID L. SWIFT,§ and BRUCE O. STUART‖

* Litton Bionetics, Inc., Kensington, Maryland, U.S.A.
† Harvard University, Boston, Massachusetts, U.S.A.
‡ Lovelace Inhalation Toxicology Research Institute, Albuquerque, New Mexico, U.S.A.
§ University of California, Irvine, California, U.S.A.
‖ Johns Hopkins University, Baltimore, Maryland, U.S.A.

Abstract—While evaluating respiratory tract kinetic models, we have encountered a number of problems with the recently published ICRP Dosimetric Model for the Respiratory System. Some of those to be discussed are the following.

1. The assumption that the nasopharyngeal (N-P) region can be ignored, despite considerable evidence of significant retention in the N-P region and of pathological effects.
2. Treatment of the tracheobronchial tree (T-B), the pulmonary (P) region and the pulmonary lymph nodes (PLN) as one combined organ for calculating dose.
3. The lack of deposition estimates for very small particles (<0.1 μm dia.) and the use of a probit scale for percentage deposition.
4. Use of the aerodynamic equivalent diameter of particles beyond the range of applicability of the model and the desirability of defining an appropriate 'equivalent deposition diameter' suitable for the whole range of particle sizes of interest.
5. Treatment of components of inhaled mixtures as if they are independent in regard to both deposition and clearance characteristics.
6. Limitations of the currently used rigid solubility classifications (D, W or Y) of inorganic compounds in estimating retention and disposition of deposited particles.

INTRODUCTION

The U.S. National Council on Radiation Protection and Measurements (NCRP) Scientific Committee on Internal Emitter Hazards established a number of task groups to evaluate and make recommendations on various aspects of this important problem. The mission of one of these concerns Respiratory Tract Kinetic Models. Some of the problems that this task group raised with the recently published ICRP Dosimetric Model for the Respiratory System (ICRP PUBLICATION 30, 1979) are discussed here, but it should be emphasized that this paper does not represent an official NCRP position.

In the Introduction, ICRP PUBLICATION 30 (1979) states that one of the reasons for revising ICRP PUBLICATION 2 (1960) is that there have been some misconceptions about its intent and some misuse of its recommendations. The same statement could be made about the report of the ICRP TASK GROUP ON LUNG DYNAMICS (1966) (TGLM).
Even though ICRP Publication 30 (1979) contains numerous caveats restricting the applicability of its recommendations to the estimation of radiation dose for occupationally exposed adults, it is the tendency of agencies and organizations to take models published by prestigious organizations like the ICRP and apply them to population groups and situations for which they were not intended and for which they may be inappropriate. The most recently updated dosimetric model is similar to the 1966 TGLM recommendations, but, while it incorporates some recent information on the respiratory tract, it still appears to have several shortcomings, some of which could be serious if the model is misused.

A number of models have been developed over the last half-century to describe the deposition of inhaled particles in the various regions of the respiratory tract, their retention and subsequent translocation to other body organs, and eventual excretion from the body. Factors considered in these models (which have been adequately reviewed by the 1966 TGLM) include the physiology of the respiratory tract and respiration, the physico-chemical characteristics of the inhaled particles, and interactions between deposited particles and the body systems. Even though it was never explicitly adopted by the ICRP, the TGLM presented comprehensive deposition and retention models which have been widely used since their 1966 publication. Some of the parameters were later updated and officially adopted (ICRP Publication 19, 1972).

ICRP Publication 30 (1979) as it relates to the new 'Dosimetric Model for the Respiratory System' is summarized in the next section, following which some of the perceived shortcomings of the model are presented and discussed as to their implications.

Summary of a New Model

ICRP Publication 2 (1960) recommended values of the maximum permissible concentrations in air and water (MPC) and of maximum permissible body burden (MPBB) for a number of radionuclides. These were based upon the concept of limiting the dose equivalent from inhaled or ingested radionuclides for any stated period to ICRP recommended values for occupational exposure. Misconceptions about the intent, and misuse of, these recommendations, together with the accumulation of new and more reliable biological and physical data, and changes in the basic recommendations of the Commission (ICRP Publication 26, 1977) made preparation of a new report necessary. The new recommendations are designed to avoid radiation-induced effects of early onset and later to minimize the possibility of radiation-induced cancer and hereditary disease, using the hypothesis that the risks of developing fatal cancer in exposed people or of genetic effects in their offspring are linearly related to dose equivalent, that there is no threshold dose-equivalent below which no effects would occur and that effects are independent of dose-equivalent rate and dose-equivalent fractionation.

The concepts of MPC and MPBB are not used in the new report, being replaced by the Annual Limit of Intake (ALI) for ingestion or inhalation, and the Derived Air Concentration (DAC) of each radioactive material. The primary standard is the Committed Dose equivalent, H_50, defined as the total dose equivalent in any tissue over the 50 yr after the intake of the radionuclide into the body. Values of H_50, ALI and DAC for radionuclides are intended only for interpreting the occupational exposure of
adults and the $ALI$ values are based only on radiation, not chemical, toxicity. The assumptions made in deriving the values given should not be regarded as matters of established fact.

Chapter 2 of ICRP PUBLICATION 30 (1979) presents the basic limits for the control of internal dose, while Chapter 3 considers secondary and derived limits for the control of internal dose in more detail. Standards are based on the anatomical and physiological values given in ICRP PUBLICATION 23 (1975). In Chapter 3 $ALI$ is defined as the greatest value of $I$ which satisfies both the inequalities

$$ I \sum_{T} W_T (H_{50,T} \text{ per unit intake}) \leq 0.05 \text{ Sv} $$

where $W_T$ is the weighting factor for tissue ($T$) given by the ICRP and $H_{50,T}$ (in Sv) is the total committed dose equivalent in tissue ($T$) resulting from intakes of radioactive materials from all sources during the year in question, and

$$ I (H_{50,T} \text{ per unit intake}) \leq 0.5 \text{ Sv} $$

where $I$ is the annual intake of the specified radionuclide either by ingestion or by inhalation, in Bq (one disintegration s$^{-1}$). $H_{50,T}$ per unit intake has the units Sv Bq$^{-1}$.

Appropriate adjustments must be made when mixtures of radionuclides and multiple routes of exposure are involved. Derived Air Concentration is the concentration of any radionuclide in air (Bq m$^{-3}$) which, if breathed by Reference Man for a working year of 2000 h (50 weeks at 40 h per week) under conditions of light activity, would result in the $ALI$ by inhalation, i.e.

$$ DAC = \frac{ALI}{(2000 \text{ h} \times 60 \text{ min/h} \times 0.02 \text{ m}^3 \text{ min}^{-1})} = \frac{ALI}{2.4 \times 10^3 \text{ Bq m}^{-3}}. $$

The rate of intake is restricted only in the case of occupational exposure of women of reproductive capacity and pregnant women. It is emphasized that the $ALI$ is the overriding limit.

Chapter 4 describes the methods used to calculate the committed dose equivalent ($H_{50}$) per unit of activity taken into the body and $ALI$. The dose equivalent $H$ is the product of $D$, $Q$ and $N$ at the point of interest, where $D$ is the absorbed dose (in greys, Gy), $Q$ is the quality factor for the particular radiation and $N$ is the product of any other modifying factors such as dose rate, fractionation, etc. and is assumed to be 1. The value of $Q$ is taken as 1 for beta particles, electrons and all electromagnetic radiation, 10 for fission neutrons and protons, and 20 for alpha particles, recoil nuclei and fission fragments.

The compartmental model used for the respiratory system is described in Chapter 5, which is the focus of the present paper. After a radionuclide has been inhaled, it will be translocated to the body fluids at a rate determined by the rate constants for the different compartments in the respiratory system. Loss of the radionuclide from any compartment is taken to be governed by first order kinetics, as is clearance from the transfer compartment (body fluids) with a half-life of 0.25 day (Fig. 1). Each site of deposition is assumed to consist of one or more compartments from which transfer is to excretion pathways with no feedback to the transfer compartment. For the most part, it is assumed that decay products produced in the body stay with and behave
metabolically like the inhaled parent radionuclide, but the ICRP recommends that information to the contrary should be used to calculate revised ALI values if available.

The Dosimetric Model for the Respiratory System relates specifically to the inhalation of radioactive particles which will deliver varying doses to different regions of the respiratory system, depending upon the distribution of the inhaled particles with respect to their size. Dose to the N-P region has been neglected, since the ICRP believes it to be small compared with that received by other regions for most particle sizes. It also decided that irradiation of the lung is likely to be more limiting than the very much greater dose delivered to pulmonary lymph nodes by translocated, insoluble radioactive particles. Because the distribution of dose to cells in the lung can be very inhomogeneous and because so little is known about the precise location of the cells at risk, the ICRP decided that for the purposes of radiological protection of adults, it would be satisfactory to consider the T-B region, P region and PLN as one composite organ of mass 1000 g.

The Deposition and Retention Model is essentially that of the TGLM (1966), with changes in parameters where appropriate new information has been developed. The most important factor affecting deposition is particle size. The activity median aerodynamic diameter of radioactive aerosols (AMAD in micrometers, μm) having geometric standard deviations (GSD) of less than 4.5 is plotted on the ordinate for AMAD values between 0.2 and 10 μm, against percent deposition with provisional estimates up to 20 μm and down to 0.1 μm, on the abscissa, using logarithmic probability paper. The parameters \( D_{N,P} \), \( D_{T,B} \), and \( D_p \) describe the fractions of inhaled particulate material initially deposited in the N-P, the T-B and the P regions, respectively. The model is not intended for aerosols having AMAD values of less than 0.1 μm. Figure 5.1 of ICRP PUBLICATION 30 (1979) presents this deposition model (Fig. 2).
Three classes of retention are defined to take into account the physico-chemical characteristics of the deposited material, since these determine the rate of clearance from the lung. Class D refers to material having retention half-times in the pulmonary region of less than 10 days, class W material retention half-times from 10 to 100 days (i.e. weeks) and class Y (yr) material retention half-times greater than 100 days. Each of the three respiratory tract regions are subdivided into compartments to reflect the fact that different clearance pathways are operative in each, having their characteristic clearance half-times ($T$) for the fraction ($F$) of material being cleared by that route. ICRP PUBLICATION 30 (1979) Fig. 5.2 is reproduced below (Fig. 3). The arrows between the N-P compartment b, and the T-B compartment d, and the GI tract in Fig. 5.2 of ICRP PUBLICATION 30 were pointing in the wrong direction, since mucociliary transport translocates material from these regions to the GI tract. The other principal transport processes are absorption into body fluids and transport via the pulmonary lymphatic system (L). The ICRP considers the indefinite retention compartment of L appropriate only for relatively insoluble materials.

The clearance of material deposited in the respiratory system is assumed to be governed by first order kinetics in every compartment, and a series of interlinked first order differential equations which describe the clearance of deposited material from the lung is presented. Details are then given as to how to calculate the committed dose equivalent, $H_{50}$, in the lung.

All calculations of committed dose equivalent are for an aerosol with an AMAD of 1 $\mu$m and, if AMAD is unknown, it is recommended that the values of $H_{50}$, ALI and DAC given in the dosimetric data be used. If AMAD is known, then $H_{50}$ for that median diameter may be calculated using Fig. 2 if the fraction $f_{N-P}$, $f_{T-B}$ and $f_p$ of the committed dose equivalent in the reference tissue resulting from deposition in the N-P, T-B and P regions, respectively, is known.
Discussion of the new ICRP model

The ICRP Dosimetric Model for the Respiratory System is in part only a slight modification of the 1966 TGLM. However, in its application to dose calculations, the new model departs from the Task Group model in a number of ways. These and some other limitations of the new model will be presented and discussed here.

Neglecting nasopharyngeal dose

There is abundant experimental evidence indicating that significant fractions of inhaled particles (Hounam and Morgan, 1977) and gases (Brain, 1970) can be deposited in the nose and pharynx. Important quantities of large particles (> 5 μm dia.) and very small particles (<0.01 μm) are deposited in the N-P region.

Evidence from studies of occupational disease suggest that particles deposited in the nasal passages can be retained long enough to produce biological responses, some transitory and reversible (allergies and irritation from gases, etc.), others irreversible (perforations of the nasal septum in workers following prolonged exposures to chromates, and other inorganic compounds) (Williams, 1974).

More important, neoplasms have also been associated with particle retention in the nose. Although nasal cancers are rare in the general population, their incidence increases dramatically among various occupational groups, such as furniture manufacturers (Andersen et al., 1977), leather industry workers (Acheson, 1976), asbestos workers (Stell and McGill, 1973), and radium dial painters. There is also evidence that the N-P region cannot be neglected for unattached radon and thoron daughter products. Cancers of the nasal tissues have been seen in thorotrast-treated patients, while squamous carcinomas in the nose and pharynx have been described in hamsters, rats and dogs exposed to radon daughters (Stuart, 1979) and other inhaled radionuclides (Benjamin et al., 1979).

Treatment of lung as a composite organ

The 1966 TGLM provided a system for calculating regional respiratory tract...
Limits for radionuclide intake (ICRP Publication 30)

Retention of aerosols. The new ICRP model treats the lung for radiological protection purposes as a composite tissue comprising the trachea and bronchi (T-B region), the pulmonary region (P), and its associated regional lymphoid tissues (PLN), and having a mass of 1000 g (1 kg). This procedure masks the very high dose delivered to the pulmonary lymph nodes by calculating a smear dose to the whole lung. However, even for relatively soluble actinides like americium dioxide, for which PLN retention is transitory and never exceeds 1% of the initial lung burden (ILB) in dogs, the dose to PLN can be considerable (0.5 Sv = 50 rad per nCi ILB over 1000 days) (Craig et al., 1979). Even though pulmonary lymphatic tissue appears to be fairly insensitive to radiation, there is no certainty that it is not implicated in effects that have been observed elsewhere in the lung.

A second objection to treating the lung as one composite tissue is that lung cancers in people that have resulted from inhaled radioactivity have been mainly due to irradiation of the bronchial epithelium, e.g. in underground uranium miners exposed to radon and radon daughter products. Calculations based upon parameters given in Figs 2 and 3, and ICRP Publication 23 (1975) for organ weights, mucous layer thickness and flow rate, airway diameters and lengths, etc. (Harley and Pasternack, 1979) show that most of the doses to the pulmonary region are about 30 times those to the bronchial epithelium for people exposed to long-lived actinide elements in the environment (i.e. submicron particle sizes), depending on the alpha energies and the assumed depth of the sensitive epithelial cells. For alpha-emitting radionuclides with radioactive half-times less than about 15 days, the bronchial epithelial dose exceeds that to the P region. This is also true for larger particles, radioactive vapours and gases, and for oral breathing, which results in large depositions of inhaled particles in the T-B region. For particles greater than 10 \( \mu m \) AMAD, deposition in the T-B region may be nearly complete.

It would appear that treatment of the T-B, P and PLN regions of the lung as one composite organ of mass 1 kg and calculating a smear dose for this organ is highly undesirable, as it masks what may be the biologically significant doses delivered to the different regions of the lung.

Small particle deposition estimates and use of probit scales

Stöber et al. (1969) have stated that the most powerful parameter for describing the dynamic behaviour of a particle in the airborne state is the aerodynamic diameter. This is defined as the diameter of a sphere of unit density which, in a mechanical force field, assumes the same steady-state velocity as the actual particle under consideration. Mathematically,

\[
d_{ae} = \frac{d_p \sqrt{C_{ae}}}{\sqrt{\rho_{ae}}} = \frac{d_p \sqrt{C_p}}{\sqrt{\rho_p}}
\]

or, since the density \( \rho_{ae} \) is unity

\[
d_{ae} = d_p \sqrt{C_{ae}} = d_p \sqrt{C_p}
\]

where the subscripts refer to aerodynamic equivalent (ae) or the actual particle (p), \( d \) is diameter, \( \rho \) is density and \( C \) the slip correction factor.

It has been shown that aerosol deposition is closely correlated to the median aerodynamic diameter of the aerosol size distribution as long as the geometric standard deviation (GSD) is less than about 4.5 (Mercer, 1973).
A few calculated values for a GSD value of 2.5 and a GSD range from 1.2 to 3.0 have been indicated on Fig. 2. These come from the original TGLM (1966) who plotted deposition curves on log-probability paper because of the sigmoid appearance of the N-P and P region curves when the logarithm of AMAD was plotted against percentage deposition on linear paper. They showed that the deposition curves for mean tidal volume (1450 cm³) and respiration rate (15 per min) could adequately represent a substantial range of respiratory states. This implied that the minute volume would not alter the percentage deposition very much under ordinary circumstances, though it would affect the total amount of dust inhaled.

Unlike the TGLM, which covered an AMAD range from 0.1 to 100 μm, in their calculations and graphs, the new ICRP model is restricted to the range from 0.2 to 10 μm AMAD, with only provisional estimates up to 20 μm and down to 0.1 μm AMAD. They state explicitly that the model in not intended for aerosols having AMAD values of less than 0.1 μm. Even between 2 and 10 μm there are substantial differences between the TGLM and the new ICRP model values as plotted for the P and N-P regions.

The principal limitation of the model is the lack of deposition estimates for AMAD values of less than 0.1 μm and the use of a probit scale on the percentage deposition axis. There is a need for deposition values for particles smaller than 0.1 μm AMAD, because these do occur in situations of interest from a radiation dosimetry point of view (e.g. radon daughter products, freshly generated condensation aerosols, rapidly dispersed metal fumes).

The TGLM maintained that plotting the deposition curves for the various regions on log-probability paper simplified the graphic relationship. In our view, it does exactly the reverse. It is very difficult to interpolate mid-range deposition values on a probit scale and it gives unjustifiable accuracy for very small or very large deposition efficiencies, and it obscures the true shapes of the regional deposition curves. The independent variable, particle size, should be plotted on a logarithmic scale abscissa, and the dependent variable, percentage deposition, on a linear ordinate scale with approximate deposition ranges as was done in the original TGLM (1966) report.

Range of applicability of aerodynamic diameters

The TGLM (1966) concluded that deposition in the respiratory tract could be adequately estimated using a single parameter of the aerosol distribution, the median aerodynamic diameter. For convenience, they assumed that the log-normal distribution was applicable to all dusts. This is adequate for single-component aerosols, but not for complicated distributions like the trimodal of some environmental aerosols (Whitby, 1978).

When diffusion is the dominant transport mode (less than a few tenths of a μm real particle diameter), density is of no significance for deposition (Fig. 4). The aerodynamic equivalent diameter $d_{ae}$ is defined in terms of the settling velocity of unit density spheres, and a 0.2 μm dia. sphere of density 10 g cm⁻³ has a $d_{ae}$ of about 0.8 μm. Figure 4 shows that the size for which diffusion becomes predominant is not unique but depends upon the density of the particles. Thus, the settling velocity of typical aerosols becomes less than the diffusional displacement somewhere between 0.2 and 0.5 μm dia. and the aerodynamic equivalent diameter is no longer really the appropriate parameter for describing the deposition characteristics of inhaled aerosols of smaller diameter.
These considerations suggest the need for the definition of a new parameter, the equivalent deposition diameter, to describe the deposition of inhaled particles. A simple but clumsy approach is to define this as follows:

\[ d_{ed} C_{ed}^{1/2} = d_{p} \rho^{1/2} C_{p}^{1/2} \]

where for

\[ d_{p} \rho^{1/2} \geq 0.5, \quad n = 1 \]

and for

\[ d_{p} \rho^{1/2} < 0.5, \quad n = 0 \]

and

\[ d_{ed} = \text{the equivalent deposition diameter of particles.} \]

In reality the transition from being density dependent to being density independent is gradual, but a simple step change at an appropriate aerodynamic equivalent diameter serves to illustrate the principle. Note that the criterion for the transition is the aerodynamic, not the microscopic, diameter (right-hand side of equation is \( d_{ae} \) when \( n = 1 \)).

If aerosol sampling is carried out using samplers whose characteristics depend upon the diffusional displacement of the particles, e.g. diffusion batteries, then equivalent deposition diameter should be directly measurable.

**Treatment of components of inhaled mixtures**

The new ICRP model treats components of inhaled mixtures as being independent in their deposition and clearance characteristics. This will only be the case if there is no interaction between the nuclides. The question of interactions between radioactive gases, vapours and particles, which can significantly alter the site of deposition and the quantity of radionuclide deposited, is not addressed in the ICRP model.
Available evidence from experimental animal studies on the biological behaviour of the components of mixtures is somewhat conflicting. For example, no preferential dissolution of $^{241}$Am from inhaled $^{239}$PuO$_2$ in dog lung was observed (BAIR and PERKINS, 1968), and the $^{241}$Am dissolution from $^{239}$PuO$_2$ is much slower than the observed $^{241}$AmO$_2$ dissolution in dog lungs (e.g. CRAIG et al., 1979). However, faster in vivo dissolution of U than Th was suggested from inhalation studies with pitchblende (STUART and BEASLEY, 1967) or carnottite (STUART and JONES, 1968). These and other similar data (JOHNSON et al., 1970; KANAPILLY and GOH, 1973; KANAPILLY et al., 1973; THOMAS, 1971; and Moss, 1976) suggest that, when the particle matrix is relatively soluble, the dissolution rates of the individual components could be substantially different from each other. The intrinsic solubility of the major components will be one of the most important factors affecting the dissolution, and, therefore, clearance from the lung and translocation to other tissues or excretion, of mixed matrix aerosols. Thus, both deposition and clearance may be substantially different for a particular material when it is a component of a mixture of inhaled compounds.

### Solubility classifications of inorganic compounds

The D, W and Y classifications for the clearance of inorganic compounds from the lung have been extensively used for the estimation of radioactive material hazards despite the ICRP caveats that the classifications should be ignored whenever there is enough information about a compound to be specific. Translocation or dissolution of particles deposited in the respiratory tract reflects the combined effects of the physico-chemical nature of the particles and the physiological conditions existing in the lung, few of which play any part in classification of materials.

Recent studies (STAHLHOVEN et al., 1979) have shown that particles deposited in the pulmonary region of the lung do not clear as rapidly as indicated by the clearance parameters of Fig. 3. In lung retention measurements using four different aerosol particle sizes inhaled by people, small particles ($<2.3 \mu m$ AMAD) did not show the specified 40% early clearance of lung deposits. Larger particles did show a rapid clearance, but this was material deposited in the T-B region. Thus, a 40% underestimation of the dose to the lung and a large underestimation of absorbed radioactivity may occur.

By way of illustration, assume that the lung deposit remains in the pulmonary region until mechanically cleared to the GI tract or absorbed into the systemic circulation, that all material is available to both clearance pathways, and that a single rate constant applies to each clearance pathway. The lung dose in $\mu$Ci days/$\mu$Ci ILB (or Bq.s/Bq ILB) is a convex function of the absorption half-times (Fig. 5(A)). The new ICRP model gives a 20% higher estimate of lung dose for class Y material but a 33% lower estimate for class W material. Larger differences are seen in the predicted fractions of deposited radioactivity absorbed into the systemic circulation (Fig. 5(B)). The new ICRP model predicts that ultimately about 20% of the pulmonary deposit will be absorbed for both class W and Y materials (Fig. 3), but 90% of the class W material should be absorbed, if all material retained in the lung is available for absorption. This is consistent with observations in dogs that inhaled insoluble and soluble radioactive aerosols. This simpler lung clearance model has been used to fit the internal organ cerium radioactivity distribution patterns (NCRP REPORT No. 60, 1979). Both people
and dogs appear to have a long-term fractional pulmonary clearance rate of only about 0.001 per day.

The above considerations seem to indicate the necessity of either changes to the new ICRP clearance model parameters or extreme caution in their application to materials for which no data are available. The clearance pathway showing 40% of the initial pulmonary burden being cleared to the gastrointestinal tract with a 1 day half-time should be eliminated, while the estimates of the amounts of radioactivity absorbed into the systemic circulation, and therefore available for deposition in bone and liver, seem to need to be increased.

Options for improved classification on the in vivo dissolution of radionuclides include establishment of up to five radionuclide classes based upon chemical form, particle size, specific activity, etc. Such a classification should distinguish between 1 and 0.01 μm PuO₂ particles, between ²³⁸PuO₂ and ²³⁹PuO₂ (both now class Y materials), ²³⁹Pu in UO₂ and in pure ²³⁹PuO₂, etc. A second option is the use of time-dependent
dissolution rates along with mechanical clearance functions. These could be used in the assessment of lung and systemic organ burdens based on bioassay data. Also, the influence of therapeutic procedures on removal half-times could easily be taken into account.

DISCUSSION AND CONCLUSIONS

In a review of available experimental data, Mercer (1975) concluded that for most individuals TGLM (1966) would overestimate P deposition, underestimate T-B deposition and provide an adequate estimate of N-P deposition. A number of other limitations of the new ICRP Model exist which will be briefly presented here.

Nasal vs mouth breathing. The new ICRP model is applicable only to nasal breathing, even though the ICRP assumed dose to the N-P region to be negligible. The TGLM (1966) presented curves (p. 180) showing that deposition in the P region is much higher for oral than for nasal breathing for all particle sizes greater than 1 μm, four to five times for larger sizes. Mouth breathing is common under conditions of heavy work and for people who have impaired nasal conductance.

Hygroscopic growth of particles. The effect of hygroscopic growth of particles on regional deposition in the respiratory tract of certain particle sizes may be more pronounced than predicted by TGLM (1966) (Ferron, 1977). Even inert materials may grow rapidly under supersaturation conditions caused by the temperature differential between aerosol particles and the respiratory environment. The hygroscopic growth of particles in the diffusion size range may alter their deposition pattern substantially (Sinclair et al., 1974).

Normal vs abnormal or diseased lung. Many abnormal states appear to enhance T-B deposition at the expense of P deposition. On the other hand, there is some evidence that cigarette smoking may hinder clearance of particles from the P region. Many workers smoke cigarettes, but their lungs could not be considered normal.

Electrical charge effects. The experimental evidence indicates that particles in nature will probably not have levels of charge that will significantly alter the deposition efficiencies. However, this is not a universally held view (John, 1977) and equilibrium charge levels may be greatly exceeded by freshly-generated aerosols (Mercer, 1973). In instances of exposure to such aerosols, deposition in the respiratory tract may be enhanced by as much as 30% (Melandri et al., 1977).

Biological variability. Since the new ICRP model is intended specifically for workers, it confines itself to use of the physiological and morphological parameters for Reference Man (ICRP Publication 23, 1975). The population at large includes the very young and the very old, who can be expected to display different deposition efficiencies and clearance characteristics. These need to be considered in arriving at ALI and DAC values for the public. However, even amongst healthy workers, the variability in deposition fractions and clearance rates of radioactive materials are mean values subject to a three-fold variation from one individual to another (Cuddihy et al., 1979).
Acute vs chronic exposures. The new ICRP model assumes biological effects to be independent of dose-equivalent rate and dose-equivalent fractionation, but clearance rates may be substantially affected by the chronic deposition of radionuclides in the lung. Also, removal half-times may be high enough to impair clearance mechanisms.

In conclusion, the authors feel that the new ICRP Dosimetric Model for the Respiratory System can be refined, qualitatively and quantitatively, to account for most of the factors discussed above.

REFERENCES

ACHESON, E. D. (1976) Nasal cancer in the furniture and boot and shoe manufacturing industries. *Prev. Med.* 5, 295–315.

ANDERSEN, H. C., ANDERSEN, I. and SOLGAARD, J. (1977) Nasal cancers, symptoms and upper airway function in woodworkers. *Br. J. Ind. Med.* 34, 201–207.

BAIR, W. J. and PERKINS, R. W. (1968) Plutonium–Americium ratio in dogs after inhalation of 239PuO2 and 239Pu(NO3)4. Battelle-Pacific Northwest Laboratory Report 174, 3.12–3.15.

BENJAMIN, S. A., BÖCKER, B. B., CUDDIHY, R. G. and McCLELLAN, R. O. (1979) Nasal carcinomas in beagle dogs after inhalation of relatively soluble forms of beta-emitting radionuclides. *J. nat. Cancer Inst.* 63, 133–139.

Brain, J. D. (1970) The uptake of inhaled gases by the nose. *Ann. Otol. Rhinol. Lac.* 79, 529–539.

Craig, D. K., PARK, J. F., POWERS, G. J. and CATT, D. L. (1979) Variability in target organ deposition among individuals exposed to toxic substances. *Toxic, appl. Pharmac.* 49, 179–187.

FERRON, G. A. (1977) The size of soluble aerosol particles as a function of the humidity of the air. Application to the human respiratory tract. *J. Aerosol Sci.* 8, 251–267.

HARLEY, N. H. and PASTERNAK, B. S. (1979) Potential carcinogenic effects of actinides in the environment. *Hlth Phys.* 37, 291–300.

HOUNAM, R. F. and MORGAN, A. (1977) Particle deposition. In *Respiratory Defense Mechanisms* (Edited by Brain, J. D., PROCTOR, D. F. and REID, L. M.) pp. 125–156. Marcel Dekker, New York.

ICRP PUBLICATION 2 (1960) Recommendations of the International Commission on Radiological Protection Report of Committee II on Permissible Dose for Internal Radiation (1959). Pergamon Press, Oxford, U.K.

ICRP PUBLICATION 19 (1972) The Metabolism of Compounds of Plutonium and Other Actinides. Pergamon Press, Oxford, U.K.

ICRP PUBLICATION 23 (1975) Reference Man: Anatomical, Physiological and Metabolic Characteristics. Pergamon Press, Oxford, U.K.

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

ICRP PUBLICATION 30, Part 1 (1979) Limits for radionuclides by workers. *Ann. ICRP* 2.

ICRP TASK GROUP ON LUNG DYNAMICS (1966) Deposition and retention models for internal dosimetry of the human respiratory tract. *Hlth Phys.* 12, 173–207.

JOHN, W. (1977) Particle charge effects. Presentation at Conference on Aerosol Techniques in Health Effects Studies, 12–13 December 1977, Berkeley, California. Sponsored by the California Air Resources Board.

Johnson, L. J., WAITERS, R. L., LAGERQUIST, C. R. and HAMMOND, S. E. (1970) Relative distribution of plutonium and americium following experimental PuO2 implants. *Hlth Phys.* 19, 743–749.

KANAPILLY, G. M. and GOH, C. H. T. (1973) Some factors affecting the in vitro rates of dissolution of respirable particles of relatively low solubility. *Hlth Phys.* 25, 225–237.

Kanapilly, G. M., RAABE, O. G., GOH, C. H. T. and CHIMENTI, R. A. (1973) Measurement of in vitro dissolution of aerosol particles for comparison to in vivo dissolution in the lower respiratory tract after inhalation. *Hlth Phys.* 24, 497–507.

MELANDRI, C., PRODI, V., TARRONI, G., FARMOGANI, M., DE ZIACOMO, T., BOMPANI, G. F. and MAESTRI, G. (1977) On the deposition of unipolarly charged particles in the human respiratory tract. In *Inhaled Particles IV* (Edited by WALTON, W. H.) pp. 193–201. Pergamon Press, Oxford, U.K.

MERCER, T. T. (1973) *Aerosol Technology in Hazard Evaluation*, pp. 41–45. Academic Press, New York.

MERCER, T. T. (1975) The deposition model of the Task Group on Lung Dynamics: a comparison with recent experimental data. *Hlth Phys.* 29, 673–680.

Moss, O. R. (1976) Dissolution of uranium and vanadium from aerodynamically size-separated ore particles in a simulated lung fluid. UR-3490.1005, University of Rochester, New York.
DISCUSSION

O. G. Raabe: Are there any examples of the nasopharyngeal region being the critical organ for occupational exposure to radioactive aerosols?

Dr Craig: The full text of the paper has a number of references to effects observed in the nasopharyngeal region of thorotrast patients and radium dial painters, and from inhalation of other compounds. In experiments with dogs and rats, both at the Lovelace Inhalation Toxicology Research Institute and at Battelle Pacific Northwest Laboratory, there have been nasal carcinomas observed from inhalation of radioactive particles. There are, however, no examples that I know of in humans who have worked in the nuclear industry.

The presumption is that, providing the particle size distribution is of the right order of magnitude, one could get the same sort of effects that have been seen in other industries, for example, the furniture industry. I do not think it is something that should be neglected and the chances are very strong that a model like this is going to be widely adopted by people who have nothing to do with radioactivity. This has been the case in the past, where the original ICRP Task Group On Lung Dynamics lung model was adopted and has been used by all sorts of industries.

G. Major: Would the authors confirm the impression that I have received, namely, that ICRP should go back to the drawing board and reconsider their model?

Dr Craig: Yes.