Dosimetry modeling of inhaled particles
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Abstract

A one dimensional model for describing the transport and fate of inhaled particles and gases in the human respiratory tract has been developed. The architecture of the human lung is described using the Weibel’s model with regularized dichotomy. The Aerosol General Dynamic Equation is solved numerically during inhalation using a discrete-nodal point method for describing the particle size distribution. In the model the mechanisms of nucleation, condensation, coagulation, convection and deposition of gases and particles and a module for considering gas phase reactions have been included. Boundary layer effects are incorporated in the model by introducing appropriate dimensionless numbers (Nusslet, Sherwood) in the estimation of the thickness of the corresponding boundary layer. The model predicts the evolution of the size distribution and composition of inhaled particles and their deposition characteristics for each generation of the human airways. An application to study the deposition profile of inhaled particles under different experimental studies has been performed. The results of the model are in qualitative agreement with the Tracheobronchial and Alveolar deposition data.
1 Introduction

The relationship between exposure to particulates and resulting health effects has been examined by various researchers ([9], [2], [8]). Data from epidemiology studies conducted to date demonstrate an association between ambient particulate concentrations and increased morbidity and mortality, while data from toxicology studies have begun to provide potential biological explanations for this observed association.

The development of a mechanistic model to examine the human exposure due to inhalation of particulate matter is an important step for understanding the exposure-dose-response relationship [2]. Exposure to particulate matter is a complex procedure which includes the mechanisms of chemical disposition, toxicant-target interactions, and tissue response. In addition, there is variability in the regional deposition due to human gender, age, health condition and particle physical and chemical characteristics ([2], [4]).

A number of mathematical models of lung deposition have been developed the last years to predict particle deposition in the human airways (e.g. [7], [6], [11], [2]).

The present model is based on a mechanistic approach to evaluate particle dynamics in the human respiratory tract. It is considering the dynamics, including deposition, growth and transport of particle and gases using at the same time a boundary theory analysis for the fluid flow.

2 Dosimetry model of inhaled particles

To model the behaviour of the gaseous and particulate matter in the respiratory system, the aerosol dynamics during inhalation have been modeled. The aerosol general dynamic equation (GDE) is solved numerically by combining the solution methods introduced by Im et al. (1985) ([3]) and Jokiniemi et al. (1994) ([5]). Particles are divided into n size classes each of which having different chemical composition. Basic physical interactions, i.e. nucleation, vapor condensation, coagulation and deposition are considered.

In the model the gaseous species balance equation and the aerosol GDE are solved simultaneously as a function of location. It is assumed that the flow is one dimensional and boundary layer effects
Figure 1: Comparison of modeling results with experimental data and the predictions of the ICRP66 model for Tracheobronchial deposition by Lippmann (1977) and Chan and Lippmann (1980). The theoretical predictions are using tidal volume of 1000 cm$^3$ and flow rate of 1000 cm$^3$/sec.

are incorporated by introducing appropriate dimensionless numbers (Nusselt, Sherwood) in the estimation of the corresponding boundary layer. Besides, it is assumed that vapor concentrations are uniform over the axial volume step ($\Delta V \equiv \Delta x A$). The step time $\Delta x$ is determined by the characteristic time for nucleation, coagulation, condensation and deposition. This 1-D Semi-Lagrangian solution marches along the flow direction and solves the aerosol number size distribution, chemical species size distribution, deposition, gaseous species concentration, etc. as a function of location ([5]).
2.1 Solution of the GDE equation

In the present aerosol model we simulate the transport of aerosols inside the human respiratory track, where the architecture of the human lung is described according to Weibel’s model with regulatory dichotomy ([10]). The simulations start from the Trachea region (0th Generation) for different breathing air flows. Input data for the model are the breathing pattern, the initial particle size distribution and the temperature/saturation profile inside the respiratory tract. The size and chemical composition change in the particle spectrum is due to the mechanisms of chemical reactions, homogeneous nucleation, vapor condensation, coagulation and deposition. The whole process is described by the GDE. In the following calculations steady-state conditions are assumed. Thus we solve the aerosol GDE in one-dimensional form along the flow direction:

\[
\frac{dn_k}{dx} = \frac{1}{u} J_k \delta(k - k^*) + \left( \frac{dn_k}{dx} \right)_{\text{coag}} + \left( \frac{dn_k}{dx} \right)_{\text{grow}} - \frac{v_d A_d}{u} n_k
\]  

(1)

where \( n_k \) is the particle number concentration for a size class \( k \) with particle radius \( r_k \); the terms at the right side of the equation correspond respectively, to: 1) particle formation due to binary homogeneous nucleation mechanism (* indicates nucleating droplet); 2) the coagulation mechanism; 3) growth by condensation and chemical reactions; 4) particle removal due to deposition; \( v_d \) is the particle deposition velocity, \( A_d \) is the settling area (equal to the bottom of the considering cell in the simulations) in every volume step, \( u \) is the fluid velocity, \( J_k \) is the nucleation rate and \( \delta \) is a delta function.

3 Modeling of the architecture of the human lung

In modeling the architecture of the human respiratory tree of humans we adopt the Weibel’s description of bronchial branches as regular and dichotomous ([10]), where the branching of a tube results to two smaller tubes with the same diameter. This picture is an simplification of the human bronchial tree, where there is irregular dichotomous branching, and not well defined and invariant over time geometric characteristics of the different bronchial branches. In the present approach a simple model is presented which aims to describe
the main physical and chemical processes in the human lung. More detailed studies concerning with the variability of the human respiratory tract will be addressed in a future work. The modeling domain consists of a number of 24 generations (from Trachea to the Alveoli) based on the Weibel's description ([10]). This regular branched morphological model of the lungs forms the basis of most lung ventilation models [2]. An angle at each bifurcation point can be introduced and deposition at the bifurcation connections is modeled. The model considers a lognormal size distribution as input, as well as, gaseous phase flow rates of different species.

4 Comparison with the experimental measurements of Chan and Lippmann (1980)

Monodispersed $Fe_2O_3$ particles with aerodynamic mass median diameter larger than $2\mu m$ and density of $2.56 g/cm^3$ were used for determining the deposition in vivo and hollow cast studies [1]. The tidal volume was approximately equal to $1000 cm^3$ and the vivo studies considered twenty six healthy nonsmokers. In the calculations we choose the results in the 18th generation to represent the Alveolar region, due to limitations in the modeling approach. The reason is that the very small volumes of the last generations in the model cause an very substantial increase in the diffusion of the particles. Since, the mechanistic models included in the present model are not determined at the extreme conditions of very small volumes and almost zero fluid velocity we expect that the present physical formulation is not valid in the last generations of the human respiratory tract.

In Figures 1 and 2 we compare the predictions of the model calculations using a tidal volume of $1000 cm^3$ and inhalation flow rate of $1000 cm^3/sec$ with the experimental data. The modeling predictions seem to follow the deposition trend in the Tracheobronchial and Alveolar regions. The theoretical predictions for the different conditions in Figures 1 and 2 are based on the following estimates: (typical conditions): tidal volume of $1000 cm^3$ and flow rate of $1000 cm^3/sec$; bifurcation angle of $45^o$; particle density: $2.56 gr/cm^3$; (maximum conditions): tidal volume of $1000 cm^3$ and flow rate of $1000 cm^3/sec$; bifurcation angle of $65^o$; particle density: $2.56 gr/cm^3$; (minimum conditions): tidal volume of $1000 cm^3$ and flow rate of $1000 cm^3/sec$; bifurcation angle of $25^o$; particle density: $2.56 gr/cm^3$. Therefore,
variability in the bifurcation angle is a crucial parameter in the calculations of the particle deposition in the human respiratory system.

5 Summary, results and conclusions

A new respiratory mechanistic tract dosimetry model for describing fine particle dynamics in the human respiratory tract is developed. An important advantage of the model because of its mechanistic nature is that it has the feature to examine variability of internal human exposure to particulate matter due to different airway morphologies and transient effects.

The results of the mechanistic respiratory tract model are in qualitative and quantitative agreement with available Tracheobronchial and Alveolar deposition data. The utilization of the present model can provide detailed information for aerosol deposition profile, size and composition, for the different generations of human respiratory tract.

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Figure 2: Comparison of modeling results with experimental data and the predictions of the ICRP66 model for Alveolar deposition by Lippmann (1977) and Chan and Lippmann (1980). The theoretical predictions are using tidal volume of 1000 cm$^3$ and flow rate of 1000 cm$^3$/sec.

References


