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THE DEPOSITION OF ASBESTOS PARTICLES IN THE HUMAN RESPIRATORY TRACT

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The deposition of asbestos particles in the human respiratory tract was studied theoretically by treating them as ellipsoids of revolution, in a modified version of the method previously used by the author to study the deposition of spherical particles. The results show that as the eccentricity of the ellipsoids is increased at constant equatorial diameter, deposition is reduced in the diffusion-controlled particle-size region below about 0.2 μ. In the particle-size region where sedimentation and impaction are the controlling mechanisms, an increase in the eccentricity of the ellipsoidal particle at constant equatorial diameter is equivalent to an appropriate increase in particle density, and the deposition curves are displaced to the left.

INTRODUCTION

Microscopic asbestos fibres in the atmosphere represent a very real potential hazard to individuals occupationally exposed to such conditions. The assessment of the relative toxicity of a particular atmosphere containing asbestos includes a determination of the chemical and physical properties of the dust, including its particle-size and particle-shape distribution, and an estimate of what proportion of this dust may be expected to be deposited in the lower respiratory tract, where the dust is potentially dangerous. This paper is concerned with the latter aspect of the problem, which does not appear to have received adequate attention to date.

The deposition of spherical particles in the respiratory tract is fairly well understood, and it has been the subject of many experimental and theoretical investigations. Since it is virtually impossible to measure the deposition of dust of controlled properties in the lower respiratory tract in humans, the deposition of spherical particles in this region has been studied theoretically by a number of investigators (Findeisen,[1] Landahl,[2,3] and Beeckmans[4,5]). These same studies also yielded curves for total deposition, as functions of particle size, particle density, and breathing pattern, which were in relatively good agreement with experimental data. This agreement naturally strengthens the validity of the calculated alveolar deposition curves. No such studies have been made in the case of elongated particles such as fibres, and it is the objective of this paper to provide an assessment of the deposition behaviour of such particles in the respiratory tract, with particular reference to asbestos dust.

CHARACTERISTICS OF ASBESTOS DUST

Since the bulk of the world's production of asbestos is of the variety known as chrysotile, this form will be taken as the model in what follows. Chrysotile occurs in the form of needles with diameters between 150 Å and 1000 Å (0.015–0.1 μ) having a density of approximately 2.6 g/cc. However, when dispersed in the atmosphere chrysotile fibres are frequently agglomerated into fibre bundles up to 0.5 μ in diameter, and with lengths up to 60 times the diameter (Walter[6]). Other forms of asbestos occur as fibres having diameters up to 0.5 μ.

DEPOSITION EQUATIONS

The deposition of fibrous particles was calculated by assuming that they behave approximately as prolate ellipsoids of revolution. A prolate ellipsoid of revolution is obtained by rotating an ellipse about its major axis. The mobility of such a particle is given by Fuchs[7] as follows:

$$B = C/2\pi\mu a(2\chi_1 + \chi_2), \qquad (1)$$

where B is the mobility, defined as the ratio of the terminal velocity of a particle to the applied force, C is a slip correction factor, a is the semi-minor axis of the ellipsoid, μ is the fluid viscosity, and χ_1 and χ_2 are shape factors given by

$$\chi_1 = 2.667(\beta^2 - 1)/\left\{\frac{2\beta^2 - 3}{\sqrt{\beta^2 - 1}}\ln(\beta + \sqrt{\beta^2 - 1}) + \beta\right\}$$

$$\chi_2 = 1.333(\beta^2 - 1/\left\{\frac{(\beta^2 - 1)}{\sqrt{\beta^2 - 1}}\ln(\beta + \sqrt{\beta^2 - 1}) - \beta\right\}$$

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β is the eccentricity, defined as the ratio of the major axis to the minor axis.

The mobility equations for the ellipsoid was then used to modify the deposition equations for spheres previously used by Beeckmans[4] to calculate aerosol deposition in the respiratory tract, and the calculations were performed by computer as described in that publication.

For deposition by diffusion, the equation for spheres may be written (Beeckmans[4]) as

$$D_a' = 1 - \exp(-7.31\ D\tau/2R_a^2), \qquad (3)$$

where D_a' is the probability that a particle is deposited during inspiration in region a, D is the particle diffusion coefficient, τ_a equals the time of passage through region a, and R_a is the radius of region a. Equation (3) was used to calculate the deposition probability of an ellipsoidal particle in region a of the respiratory tract by using the expression $D = kTB$ for D, where k is the Boltzmann constant, T is the absolute temperature, and B is the mobility as calculated by means of Eq. (1). (Each region of the respiratory tract was approximated by a set of parallel cylinders, as outlined by Beeckmans.[4])

For deposition by sedimentation, the corresponding expression previously used by Beeckmans[4] for spheres may be stated as

$$S_a' = 1 - \exp(-2\mu\tau_a/\pi R_a), \qquad (4)$$

where S_a' is the probability that a particle will be deposited by sedimentation in region a during inspiration, and μ is the sedimentation velocity of the particle. The sedimentation velocity of an ellipsoid may be obtained from its mobility as follows. The mass of the ellipsoidal particle equals $4\pi\beta a^3\rho/3$, where ρ equals the density of the particle. This mass times the gravitational constant g equals the gravitational force on the particle. Hence, from the definition of β,

$$\mu = 4\pi\beta a^3\rho/3 \qquad (5)$$

Equations (1), (2), (4), and (5) together allow the calculation of S_a'. The expression previously used by Beeckmans[4] to calculate deposition by inertial impaction at the branching of the airways may be written as

$$I_a' = (L/R_a)/(1 + (L/R_a)), \qquad (6)$$

where L equals the stopping distance of the particle travelling with an initial velocity $v_{a-1}\sin\theta$, v_{a-1} being the velocity of the particle in region $(a-1)$, θ the angle between the axes of airways $(a-1)$ and a. The stopping distance of a sphere or of an ellipsoid is given by

$$L = VMB/3, \qquad (7)$$

where V is the initial velocity of the particle, and M equals its mass. Eqs. (1), (2), (6), and (7), together with the expression for the mass of the ellipsoid, are sufficient to allow calculation of I_a'. The angle θ was assumed to be equal to 30° in all calculations.

The slip correction factor C was calculated by means of an equation for spheres derived by Davies.[8] It was applied to ellipsoidal particles, with the equatorial diameter of the ellipsoid substituted for the diameter of the sphere in Davies' equation, in default of a correct expression for slip in ellipsoidal particles in the literature. This procedure seemed preferable to the alternative of not correcting for slip, but it probably resulted in a slight overestimate of the mobility of particles in the lower size ranges.

RESULTS

The program was executed for a 1000 cm³ tidal volume, and a 3-sec breathing cycle, with 0.3 sec pauses at the end of inspiration and at the end of expiration. The functional residual capacity of the

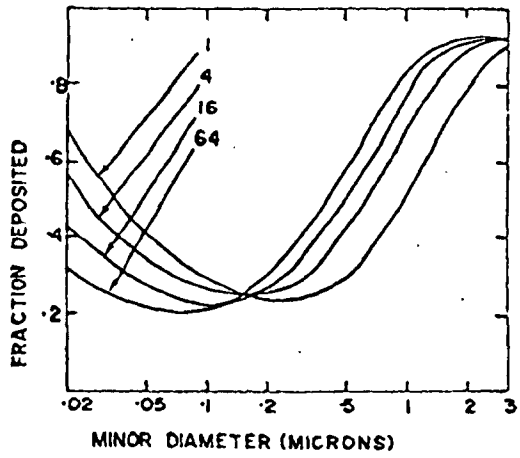

FIGURE 1 Fraction of inhaled particles deposited in all regions of the respiratory tract, vs equatorial diameter of the particle, for various values of β.

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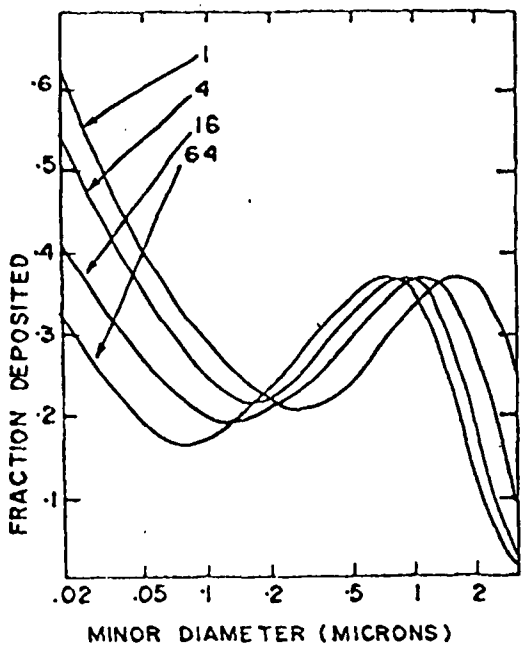

FIGURE 2 Fraction of inhaled particles deposited in the lower (alveolated) region of the respiratory tract, vs equatorial diameter of the particle, for various values of β.

lung was taken as 2000 cm³. The total and alveolar deposition curves for β values of 1, 4, 16, and 64 are shown in Figures 1 and 2. The abscissa represents the equatorial diameter of the ellipsoidal particles, in microns.

These results are perhaps most easily understood by considering the effect of increasing β on the particle mobility. It is easily shown, using equations (1) and (2), that

$$\lim_{\beta \to \infty} B = \frac{3C\ln(\beta)}{20\pi\mu^2\beta} \qquad (8)$$

Thus as β is increased indefinitely, B tends to zero. This accounts for the reduced deposition of the elongated ellipsoids in the particle size range 0.02 μ to approximately 0.2 μ, because diffusion is the predominant mechanism for deposition in this region, and the diffusion coefficient of a particle is proportional to its mobility. Thus the exponential factor in Eq. (3) will tend to zero as β increases indefinitely, and consequently D_1' will also tend to zero.

In the particle-size region above 0.2 μ sedimentation and impaction are more important mechanisms than diffusion. The exponential factor in the sedimentation equation (Eq. 4) is proportional to the sedimentation velocity of the particle, which in turn is proportional to βB. This product becomes proportional to ln β as β increases indefinitely. Consequently increasing β increases the sedimentation velocity, and increases the rate of deposition due to sedimentation. A similar argument for impaction shows that the stopping distance becomes proportional to ln β as β increases indefinitely, and consequently the factor I_1' approaches unity. Now the equations for impaction and sedimentation both contain the factor $\rho\beta B$; consequently an increase in β at fixed ρ can be simulated as far as these two mechanisms are concerned by an appropriate increase in ρ at fixed β. In other words, the deposition behaviour of ellipsoids of a given density and of a given equatorial diameter is equal to that of spheres of equal diamater, but having a greater density such that ($\rho\beta B$) is equal for both classes of particles. This is true only in the particle size region where diffusion is insignificant. Landahl[2, 3] and Beeckmans[4, 5] had shown that as the density of spherical particles is increased, the total and alveolar deposition curves are shifted towards the left, and the same can be seen in the results presented here as β is increased.

Finally, it is pertinent to ask if interception might not play a role in deposition for very elongated particles. Interception might be expected to be of significance when the polar diameter of the particle approaches the dimensions of the smaller airways. The respiratory tract model used in this study is due to Weibel,[9] who calculated a value of 0.041 cm for the diameter of the alveolar sacs, at a functional residual capacity of 4.8 liters. In the present study, the functional residual capacity was 2 liters, and the tidal volume was 1 liter. Consequently the mean lung volume during the breathing cycle was 2.5 liters, and the corresponding alveolar diameter was $0.041 \times \sqrt[3]{(2.5/4.8)}$ cm, or 349 μ. The longest particle for which calculations are reported here had a β value of 64 and an equatorial diameter of 3μ. Consequently its length was 192 μ. Moreover, as can be seen from the figures, this type of particle is virtually completely deposited in the upper respiratory tract, and consequently interception in the lower respiratory tract, if it were to occur, would not be of significance.

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