Environmental Health Perspectives
Vol. 79, pp. 249–258, 1989

Mass Transfer Rates of Polycyclic Aromatic Hydrocarbons Between Micron-Size Particles and Their Environment—Theoretical Estimates

by P. Gerde* and P. Scholander*

This paper presents a mathematical model of how rapidly polycyclic aromatic hydrocarbons (PAHs) adsorb onto initially clean micron-size particles in the ambient air and how fast these substances are likely to be desorbed from the particles after deposition on the surface lining layer of the lung. Results show that, on the one hand, the very low gas-phase concentrations of PAHs in the ambient air should result in a comparatively slow transfer of such compounds to micron-size particles, a process that may last from minutes to hours. On the other hand, the comparatively high solubilities of PAHs in the lining layer of the lung should promote an almost instantaneous release of PAHs onto nonporous particles, and a release within a matter of minutes of most PAHs reversibly adsorbed onto the interior surfaces of porous particles. Two important conclusions can be drawn from this. First, the PAHs in tobacco smoke do not have time enough to interact in the gas phase with other airborne particles before these agents are inhaled into the smoker's lungs. Therefore, adsorption in the gas phase of PAHs onto asbestos fibers can hardly be a characteristic parameter in the mechanism behind the synergistic effect between tobacco smoking and asbestos exposure for the induction of bronchial cancer. Second, the release rate of reversibly adsorbed PAHs from their carrier particles in the lung seems to be so fast that this cannot be a parameter of importance in directly influencing the residence times of such substances in the lung.

Introduction

Particle association is an important characteristic of polycyclic aromatic hydrocarbons (PAHs) both in relation to their behavior in the ambient air and in relation to their suspected role as initiators of lung cancer in man. Before inhalation in the ambient air, this association or adsorption of PAHs can be characterized as a solid-gas equilibrium, and after the deposition of particles and PAHs in the lung it can be described as a solid-liquid equilibrium. Crude estimates for these equilibria can be obtained from the literature. In the ambient air, a dominant fraction of the heavier PAHs is bound to particles (*1,2*), and, when these particles have been deposited in the lung, almost all adsorbed hydrocarbons will be released to the surrounding media (*3–5*). In contrast to these equilibria, very little is known about the rate of those mass transfer processes that lead to these equilibria. This should be of vital importance to the understanding of the subsequent biological action of these substances.

Most PAHs are adsorbed or condensed onto airborne particles at the high concentrations and elevated temperatures present in smoke stacks or exhaust pipes leading from combustion processes in which these agents are generated (*2*). For one important class of particles, however, this adsorption of PAHs has to occur under typical ambient air conditions. This class of particles is mineral fibers, on which particular interest has been focused due to the strong synergism observed between tobacco smoking and asbestos exposure for the induction of bronchial cancer (*6*). One proposed explanation for this synergistic effect is that carcinogenic compounds in the smoke, e.g., PAHs, are adsorbed onto the surfaces of the asbestos fibers, which in turn may lead to an increased retention of these carcinogens in the lung (*7*). Due to a very unfavorable equilibrium of PAHs on particles in a liquid such as the surface lining layer of the lung (*8*), this adsorption most likely has to occur before inhalation in the ambient air. The time required to saturate a fiber with PAHs in the ambient air determines how specific this effect can be. A very rapid process, perhaps lasting a second or so, may

*Department of Chemical Engineering, Royal Institute of Technology, S-100 44 Stockholm, Sweden.

give smokers considerably higher doses of adsorbed PAHs than nonsmokers, if such fibers are inhaled together with the smoke. If a span of time of minutes or hours is required, this should give smokers as well as nonsmokers roughly the same fiberborne doses of PAHs.

After deposition in the lung, two virtually counteractive mechanisms have been suggested through which particle association may influence the biological action of PAHs. On one hand, particle association has been shown to increase the carcinogenic action of PAHs in animal experiments (*9–11*). This has been explained by a slow release of these substances from their carrier particles, causing increased retention in the lung (*12*). On the other hand, it has also been suggested that a slow release of PAHs from their carrier particles might contribute to a mechanism protective of the lung. This mechanism is the rapid redistribution of PAHs from the lung to the gastrointestinal tract, apparently through mucociliary action during the first 30 min after the inhalation of these substances as aerosols in animals (*13*). Again, the true release rate of PAHs from their carrier particles is of decisive importance.

Measurements have been made of the release rates of PAHs from various bulk samples of particulates in biological fluids or models. Half-lives of PAHs of generally less than 30 min have been measured for particles like asbestos, silica, and talc (*14*). Others have demonstrated that half of the original amount of benzo[a]pyrene (BaP) adsorbed on chrysotile asbestos is released within roughly 10 min (*8*). Assuming in these cases an instantaneous surface reaction, these release rates can be regarded as being determined by a transport process acting against two resistances connected in series. The first is the particle resistance which, if wetting is good, stems from the diffusion of the adsorbed PAHs within the pores of their carrier particles. Solid particles without pores thus have very low or no particle resistance at all. The second is film resistance, which stems from the diffusion of the released substance through a more or less stagnant film of liquid surrounding the particle. The distinction between these two resistances is of great importance, because particle resistance is determined to a great extent by the properties of the particles, whereas film resistance is dependent mainly upon the properties and movements of the surrounding medium. This suggests that particle resistance is responsible for only a fraction of the time in the above release rate experiments.

The purpose of this study is to estimate the rate of adsorption of reversibly adsorbed PAHs onto solitary micron-size particles in the ambient air by means of mathematical modeling and to estimate the rate of desorption of PAHs from such particles in water, which is the principal solvent these particles encounter if inhaled and deposited in the lung. A special effort is made to use plausible dimensions and hydrocarbon concentrations.

Modeling

A vast body of literature exists concerning modeling of transport phenomena. The mathematics of fundamental transport phenomena can be found, for example, in Carslaw and Jaeger (*15*), Crank (*16*), and Bird et al. (*17*). A general treatment of transport phenomena in biological systems is provided by Lightfoot (*18*). As regards the particular question of inhalation of PAH-contaminated particles, there is a phase when physical transport phenomena are likely to dominate and could be modeled using the fundamental equations described in the literature. This is the phase from the moment when PAHs and particles are emitted into the air until the released hydrocarbons are first absorbed by cellular membranes in the lung. A better knowledge of the duration of these processes, which involve mechanisms such as molecular diffusion, convection, adsorption onto solids, and partition between various phases, can be of great value for an understanding of the subsequent biological action of these hydrocarbon carcinogens. This is particularly true since mechanisms involving very low concentrations and a micro-size scale are extremely difficult to discover experimentally without any *a priori* knowledge of what should be expected. The very complex interactions between a multitude of PAHs and particles in the environment have to be greatly simplified in order to be treated with a fairly simple model. Still, it would be of great value to have at least a rough idea of the importance of these mostly unknown phenomena. A successful simulation will at the best lie close to the center of the wide distributions of important time intervals likely to occur in reality. Of course, whatever comes out of these models must be tested in appropriate biological systems.

PAHs are formed in combustion processes through reactions in the gas phase, generating molecules with a great variation in size (*2*). The larger the molecule, the lower its vapor pressure (*19*). As soon as the combustion gases cool, an adsorption or condensation of the aromatic hydrocarbons occurs onto particles formed in the same process (*2*). The lower the vapor pressure, the greater this adsorption. At ambient air temperatures, PAHs with six ring closures or more will exist almost completely in the particle phase, whereas hydrocarbons with fewer than three ring closures will exist predominantly in the gas phase. Compounds with three to five ring closures thus will exist in a fairly equally balanced gas-solid equilibrium between gas and particles (*1*). Some well-known carcinogens such as BaP and dibenz[a,h]anthracene belong to the latter group, and this molecular size is therefore chosen for the model. The other compounds also occurring within these limits must be represented by one model PAH.

The nature and the strength of the binding between PAHs and particles varies greatly, not only between different particle types but also between the various binding sites available on the same particle (*2*). A

fraction of these binding sites on fly ash have energies that will result in a chemisorption of PAHs, whereas molecules adsorbed in excess of this amount will gradually be more reversibly bound to the surface. Also, a stronger adsorption of PAHs has been demonstrated for the carbonaceous particle fraction in fly ash as compared with the mineral and magnetic particle fraction (*20*). This great variation in the binding energies has also lead to very different estimates of the rate of the sorption reaction at the particle surfaces. In some studies this reaction has been found to be very rapid (*2*), and it has been calculated to occur within seconds at ambient air temperatures as well (*21*). However, in case of carbonaceous particles the surface reaction can be very slow, and for some fractions of PAHs this may take on the order of several days (*22*). Apparently, a fast surface reaction will make the diffusional mass transfer a rate-determining step, whereas a slow surface reaction will in itself control the rate of release of the PAHs. Since most biological experiments and release rate experiments have been performed under conditions where a very fast surface reaction of PAHs on the particles must be expected, in the present model this reaction is assumed to occur instantly, so only the kinetics of diffusional mass transfer of PAHs to and from the reacting surfaces are considered.

A general porous particle of micron-size will be treated. This particle has a homogeneous surface divided into the outer surface, directly exposed to the surroundings, and the surface of the pores in the interior of the particle. The outer surface is modeled as that of a smooth sphere, and the surface of the pores is modeled as that of straight cylindrical dead-end pores of a chosen length and diameter. The model pores are chosen fine enough so that there are essentially two mass transfer processes separated in time, with a rapid mass transfer to or from the outer surface preceding the much slower mass exchange within the particle interiors. The settling speed of micron-size particles in either the liquid phase or the gas phase should not influence the rate of mass transfer to these particles and this will therefore be the same as the mass transfer in a stagnant medium. The model PAH is assumed to have linear adsorption isotherms on the surfaces of the particles, which are estimated from the literature and from our own experimental results to be published in parallel with this article. These isotherms thus corresponds to the more reversibly adsorbed fractions of PAHs. Also, only ordinary diffusion of PAHs in the pores is considered surface diffusion being neglected.

First, the initially clean model particle is emitted into air with a certain concentration of the model PAH. The rate of adsorption of the hydrocarbon onto the outer surface and the surfaces of the pores is then modeled. Secondly, when this particle has reached equilibrium in the gas phase, its inhalation and deposition on the lining layer of the lung are simulated as the immersion of the particle into an infinite stagnant medium of pure water. The desorption of the model PAH from first the outer surfaces and then the porous interior is treated. All surfaces of the particle are assumed to be instantaneously wetted by the water. The PAH is regarded as having been released as soon as the dissolved substance reaches the outside of the particle curvature. The equations in this model are fundamental or can be easily derived from fundamental equations described in the literature. The accuracy of the solutions under the stated preconditions will be far better than the errors introduced through model simplifications and parameter estimations.

Model of the Adsorption of PAHs in the Gas Phase

Outer Surface of a Sphere

Consider a solid sphere with a diameter d_S introduced into a stagnant gas phase containing a model PAH of concentration C^G_∞ (g/m^3). This hydrocarbon has a linear adsorption isotherm on the outer surface of the sphere described by

$$C^S = K^G \, C^G_0 \qquad [1]$$

C^S = surface concentration of PAH adsorbed onto the sphere (g/m^2)
C^G_0 = gas phase concentration at the surface of the sphere (g/m^3)
K^G = linear adsorption isotherm coefficient (m)

If the initial nonsteady-state build-up of the concentration profile around the sphere is neglected, then an expression for the relative surface concentration of the PAH on the sphere as a function of time can be easily derived. The equation for steady-state diffusion to a sphere of radius R is given by Crank (*16*).

$$\frac{d}{dr}\left\{ r^2 \frac{dC^G}{dr} \right\} = 0 \qquad [2]$$

Integrating twice with respect to r gives the concentration profile around the sphere of radius R (Fig. 1). Differentiating this with respect to r gives the concentration gradient at the surface of the sphere, which in turn by multiplication with the diffusivity D^G (m^2/sec) of the model PAH gives the mass flux N (g/m^2,sec) to the surface of the sphere.

$$N = D^G \left. \frac{dC^G}{dr} \right|_{r=R} = \frac{D^G}{R} \left(C^G_\infty - C^G_0 \right) \qquad [3]$$

The concentration of the PAH in the gas phase at the surface of the sphere and the surface concentration on the sphere are related through the adsorption isotherm (Eq. 1). Combining Equations 1 and 3 and integrating over time now gives the relative surface concentration on the sphere as a function of time

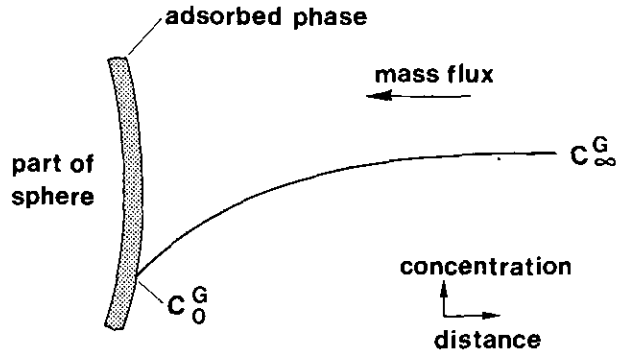

FIGURE 1. A schematic concentration profile around the sphere during adsorption of the model PAH in the gas phase.

$$\frac{C^S(t)}{C^S(t=\infty)} = 1 - \exp\left\{\frac{-2D^G t}{K^G d_s}\right\} \qquad [4]$$

A Pore

Consider a straight cylindrical pore, with a diameter d_p and length L, in contact with a gas phase containing an adsorbing substance at a concentration C^G_∞ (Fig. 2). If this compound has a linear adsorption isotherm on the walls of the pore, the adsorption onto the walls as a function of time can be derived from the solution for the diffusion of a substance into a plane sheet. With notations changed, the relative amount of the adsorbing substance in the pore as a function of time can be obtained from Crank (*16*)

$$\frac{Q(t)}{Q(t=\infty)} = 1 - \frac{8}{\pi^2}\sum_{n=0}^{\infty}\frac{1}{(2n+1)^2}\exp\left\{-D^G_{app}(2n+1)^2\pi^2 t/L^2\right\} \qquad [5]$$

The diffusivity of the substance has been modified for the adsorption onto the walls of the pores according to (*23*)

$$D^G_{app} = \frac{d_p D^G}{4 K^G} \qquad [6]$$

Simulation of Adsorption in the Gas Phase

Two model particles with different adsorption capacities will be considered, one a porous spherical particle typical of the ambient air and one a porous fiber simulating chrysotile asbestos. The adsorption coefficient chosen for the spherical particle is $K^G = 10^4$(m); this choice is explained at greater length in the Appendix. The adsorption coefficient chosen for the fiber particle is $K^G = 250$ (m), which is based on our own experimental data (*25*). The 40 times higher adsorptivity of the average spherical particle compared with the chosen mineral fiber is justified by the fact that carbonaceous particles or higher adsorptivity make up a considerable fraction of the ambient air particulates. The diffusivity chosen for the model PAH in air is $D^G = 5 \times 10^{-6}$ (m^2/sec), derived from the diffusivity of naphthalene, which is 5×10^{-6} (m^2/sec) at 0°C (*26*).

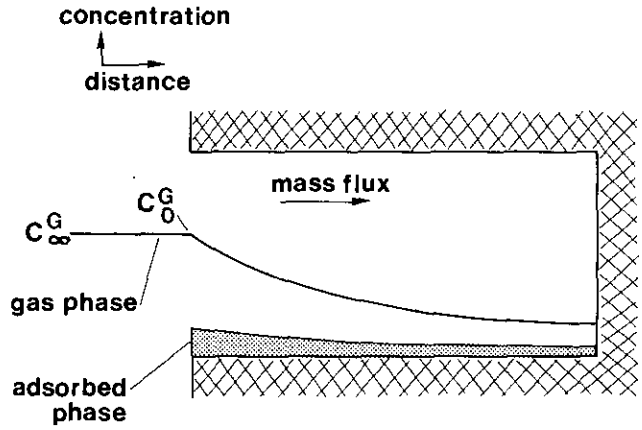

FIGURE 2. Schematic concentration profiles for the model PAH during adsorption to a straight cylindrical pore.

The rate of adsorption onto the outer surfaces of the particles is considered first. The spherical particle in the ambient air is given a diameter of $d_s = 1$ μm, and the model fiber has also a diameter of 1 μm and a length of 10 μm. For simplicity, each model particle will be characterized only by diameter of its outer curvature and by the size of its pores. This means that the outer surface of the fiber will also be assumed to act as a sphere with the same diameter as that of the fiber. This should simulate the rate of mass transfer to the fiber ends with their more or less spherical geometry quite well, but the rate of mass transfer to the cylindrical midsection of the fiber will be overestimated. However, the accuracy required in this case should not suffer appreciably if time taken for the midsection to reach a certain degree of equilibrium is 10 times longer than the time taken for the end sections, a limit that is probably not exceeded. Insertion into Equation 4 gives that the outer surface of the spherical model particle will reach 90% of its equilibrium concentration within about 40 min and the model fiber within about 1 min (Fig. 3), a difference that is due to the higher adsorption capacity of the former particle.

The rate of adsorption onto the porous interiors of the particles is now simulated. The pore diameter chosen for both particles is 100 Å, which is a fairly good estimate of the size of both a pore in chrysotile asbestos (*27*) and of a typical pore in a spherical ambient air particle (*28*). The spherical particle with a diameter of 1 μm will simply be assigned pores of unit length 0.5 μm. The chrysotile fiber is built up of bundles of finer hollow fibrils where the pores are axially aligned from

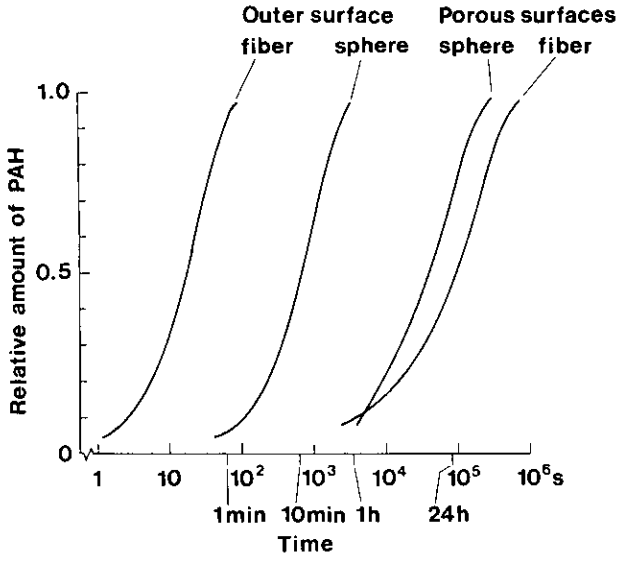

FIGURE 3. The adsorption of the model PAH in the gas phase onto the two types of surfaces of the model particles as a function of time. The two curves to the left represent mass transfer to the outer surfaces of the particles and the two curves to the right represent mass transfer to the porous interiors of the same particles.

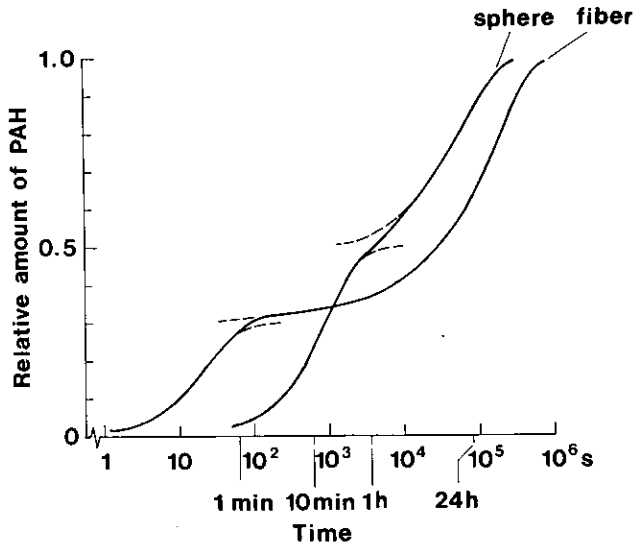

FIGURE 4. The total adsorption of the model PAH in the gas phase onto the model particles as a function of time, where mass transfer to the two types of surfaces of the particles has been weighed together at certain ratios. The dotted lines indicate the transition from mass transfer to the outer surfaces of the particles to the subsequent mass transfer to the porous interiors of these particles. The outer surface thus constitutes 30% of the total surface area in case of the model fiber and 50% in case of the spherical model particle.

one end of the fiber to the other. The open-end pores of the model fiber 10 μm in length can thus be treated as dead-end pores 5 μm in length. Insertion into Equation 5 reveals that the porous interiors of the particles will reach 90% of the equilibrium concentration within about 50 hr for the spherical particle and about 120 hr for the model fiber (Fig. 3). Here the uncertainty is greater because the neglected surface diffusion in the pores may increase this rate of mass transfer by a factor of about 10, thus shortening the given time intervals accordingly.

The two simulated mass transfer processes with such different rates can be weighed together at any chosen ratio for outer surface area to porous surface area. The ratio chosen for the spherical particle is 1 to 1, thus determined by the number of pores per particle, in this case 200. The cylindrical model fiber has 230 pores of the specified dimensions, which gives a ratio of outer surface area to porous surface area of 0.3 to 0.7, a ratio that is to be regarded only as a rough estimate of the true relation between outer surface area and porous surface area for a fiber of chrysotile asbestos. The lumped curves of mass transfer rates to the two types of surfaces for both model particles are shown in Figure 4.

Model of Desorption of PAH in the Aqueous Phase

Outer Surface of a Sphere

The complexity of the phenomena to be modeled increases considerably when the particles are deposited on the lining layer of the lung. This simple model has to be regarded as an upper estimate of the time required for PAHs to be desorbed from micron-size particles. In aqueous solution, and certainly in the lipid-aqueous lining layer of the lung, the outer surfaces of the particles will be desorbed during the initial transient phase when the steady-state concentration profile of the sphere is established. This results in a complex model, which has to be solved numerically. A much simpler solution, entailing a considerable overestimation of the time required for desorption, is to use the analytical solution for a steady-state concentration profile around the sphere. The amount of PAH required to establish this concentration profile is then neglected.

Many particle types, including asbestos fibers (*25*), have a decreasing adsorption capacity for PAHs with increasing humidity of the ambient air. Initially, deposition in water should therefore give a saturated solution of PAHs at the particle surface C^L_{sat}. The model particles will also have a saturated solution at the surface as long as the amount of PAHs previously adsorbed in gas phase exceeds the adsorption capacity of the saturated water solution, or $C^S(t) > K^L C^L_{sat}$. for $0 < t < t_{iso}$. Until the linear adsorption isotherm of the saturated solution is reached, mass transfer will give a relative surface concentration on the outer surface of the sphere as a function of time according to

$$\frac{C^S(t)}{C^S(t=0)} = 1 - \frac{2\,D^L\,C^L_{sat}\,t}{d_s\,C^S(t=0)}, \quad \text{for} \quad 0 < t < t_{iso} \qquad [7]$$

The time at which the adsorption isotherm is reached

can be calculated from the Equation 7 with insertion of $C^S(t) = K^L C^L_{sat}$, which gives

$$t_{iso} = \frac{d_s}{2D^L}\left\{\frac{C^S(t=0)}{C^L_{sat}} - K^L\right\} \quad [8]$$

For $t > t_{iso}$, the surface concentration is given by the isotherm, and the relative surface concentration is obtained from Equation 4 with changed notations

$$\frac{C^S(t)}{C^S(t=0)} = \frac{K^L C^L_{sat}}{C^S(t=0)} \exp\left\{\frac{-2D^L(t - t_{iso})}{K^L d_s}\right\}, \quad [9]$$

$$\text{for} \quad t > t_{iso}$$

A Pore

The pore is also assumed to have more of the PAH adsorbed before deposition in water than will be adsorbed in a saturated water solution. The release of the PAH from such a pore can be described in three steps (Fig. 5). First, the pore is filled with water, which immediately becomes saturated with PAH, giving a surface concentration in the pore of

$$C^S = C^S_i - d_p C^L_{sat}/4 \quad [10]$$

where $C^S(t = 0)$ has been written C^S_i. Then the desorption will propagate as a moving boundary into this pore with a linear drop in the concentration of PAH from saturation at this boundary to zero concentration at the pore mouth. When the bottom of the pore is reached by the moving boundary, there will be a nonsteady-state desorption of the pore starting with this linear concentration profile.

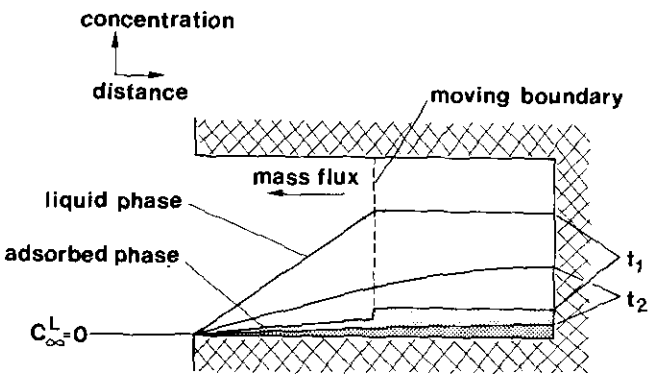

FIGURE 5. A schematic description of the concentration profiles of the model PAH at the desorption from a straight cylindrical pore. The curves marked t_1 show the concentration profiles during the moving boundary part of the problem and the curves marked t_2 show the concentration profiles during the final transient desorption of the pore.

The moving boundary part of the problem can be solved as follows. The total amount of PAH in the pore when the moving boundary has reached a distance x down into the pore is given by

$$Q(x) = \pi\left\{\frac{d_p^2 C^L_{sat} x}{8} + \frac{d_p K^L C^L_{sat} x}{2} + d_p C^S_i (L - x)\right\} \quad [11]$$

Differentiating this with respect to x and combining it with the expression for the mass transfer out of the pore:

$$\frac{dQ}{dt} = -\frac{N(x)\pi d_p^2}{4} = \frac{-\pi d_p^2 D^L C^L_{sat}}{4x} \quad [12]$$

gives a new equation, which after integration with respect to t describes the position of the moving boundary as a function of time:

$$x = \sqrt{\frac{4 d_p D^L C^L_{sat} t}{8 C^S_i - d_p C^L_{sat} - 4 K^L C^L_{sat}}} \quad [13]$$

Combining Equations 11 and 13 provides an analytical solution of $Q(t)$, valid for $0 < x < L$,

$$\frac{Q(t)}{Q(t=0)} = 1 - \left\{\frac{1}{L} - \frac{d_p C^L_{sat}}{8 L C^S_i} - \frac{K^L C^L_{sat}}{2 L C^L_i}\right\} \times \sqrt{\frac{4 d_p D^L C^L_{sat} t}{8 C^S_i - d_p C^L_{sat} - 4 K^L C^L_{sat}}} \quad [14]$$

This equation is valid for $0 < t < T$, where T is the time when the moving boundary has reached the bottom of the pore or

$$T = \frac{L^2(8 C^S_i - d_p C^L_{sat} - 4 K^L C^L_{sat})}{4 d_p D^L C^L_{sat}} \quad [15]$$

The starting point of the third and transient part of the desorption process is when the moving boundary reaches the bottom of the pore. Then there is a linear concentration profile both in the liquid and on the surface of the pore ranging from zero at the pore mouth to a saturated liquid at the bottom of the pore. The solution to this diffusional problem can be found in the solution for the corresponding heat transfer problem, which is described in Carslaw and Jaeger (*15*).

The concentration in the liquid at a position x down into the pore after a lapsed time t is then given by

$$C^L(x) = \frac{8\,C^L_{sat}}{\pi^2} \sum_{n=0}^{\infty} \frac{1}{(2n+1)^2} \exp$$

$$\left\{ -D^L_{app} (2n+1)^2 \pi^2 (t - T) / L^2 \right\}$$

$$\times \cos\left(\frac{(2n+1)\,\pi\,x}{L} \right) \quad \text{for } t > T. \qquad [16]$$

D_{app} is the apparent diffusivity in the liquid given by Equation 6. The total amount of the PAH in the pore at a given moment can be obtained by integrating both the liquid concentration profile calculated from Equation 16 and the corresponding surface concentration profile obtained from the adsorption isotherm over the pore length. This procedure is repeated with increasing time.

Simulation of Desorption in the Aqueous Phase

The two model particles with PAH previously adsorbed in the gas phase are now desorbed in the aqueous phase. As in the previous section, the mass transfer processes to the two types of surfaces will first be treated separately and then weighed together using the same ratios as before. The chosen diffusivity of the model PAH is $D^L = 5 \times 10^{-10}$ (m^2/sec), which is derived from a diffusivity of 2-naphthol in pure water of 7×10^{-10} (m^2/sec) at 29°C (*29*). The solubility chosen for the model PAH in pure water is the same as that of BaP adjusted to 37°C or 5 μg/L (*30*). The composition of the real lining layer is of course much more complicated, and the lipid-aqueous heterogenic structure is particularly important due to its content of surfactant lipids. The coefficient for the linear adsorption isotherm when the model PAH is adsorbed onto the fiber is taken from actual measurements (to be published) of the adsorption of BaP onto chrysotile asbestos in water and is chosen to be 2.5×10^{-5} (m). Since no data are available from the literature on K^L for the spherical particle, this is given a value 40 times higher than that of the fiber here as well, or 1×10^{-3} (m).

Insertion into Equations 7 and 9 shows that the model PAH adsorbed on the outer surfaces of the particles should be desorbed within about 0.1 sec in the case the model fiber and within about 4 sec in the case the spherical particle with its higher adsorption capacity (Fig. 6). However, these figures probably greatly underestimate the true release rates of readily elutable PAHs in the lung for reasons given in the next section. Insertion into Equations 14 and 16 shows that the fraction of PAHs adsorbed within the pores of the particles will be desorbed within a few minutes in the case of the spherical particle and within about 10 min in the case of the model fiber (Fig. 6). Here the

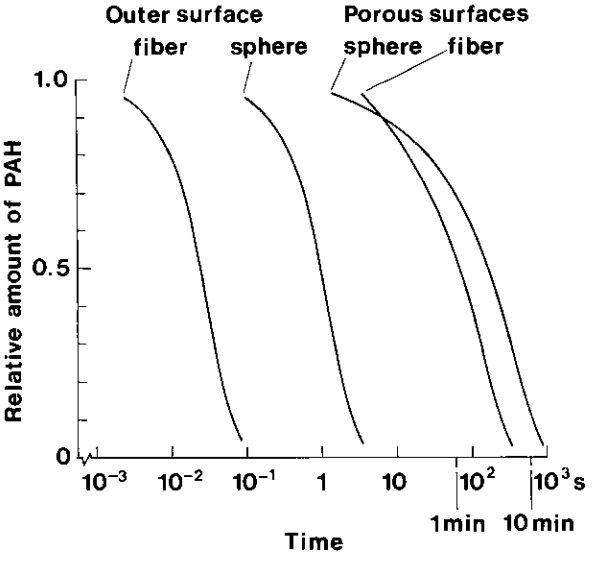

FIGURE 6. The corresponding desorption of the model PAH from the two types of surfaces of the model particles in water as a function of time.

influence of lipids in the liquid filling the pores on the release rate of PAHs is more difficult to assess, but the results obtained should indicate the order of magnitude. If these curves are weighed together in the same ratios of outer surface area to porous surface area as before, this gives the release of the model PAH from the particles (Fig. 7).

Discussion and Conclusions

In general, the rate of desorption of PAHs from particles in water is much more rapid than the rate at which these hydrocarbons are adsorbed in the ambient air. The reason for this is the extremely low

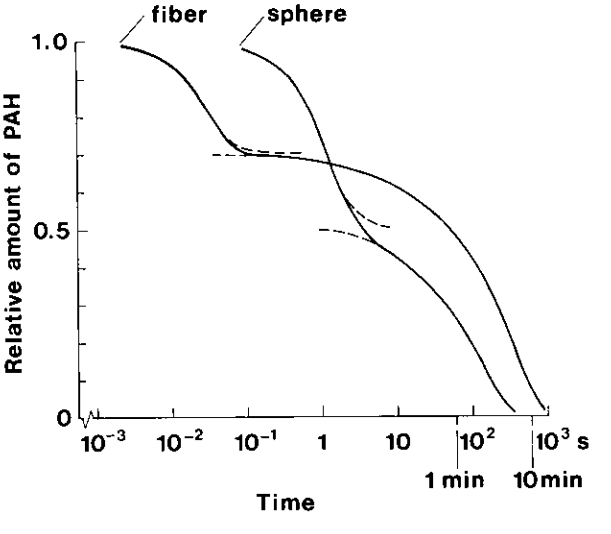

FIGURE 7. The total desorption of the model PAH from the model particles in water as a function of time.

gas-phase concentrations of PAHs compared even with their very low water solubilities, a condition that leads to a driving force for the desorption in the calculated examples that is roughly 5 million times that of the adsorption.

The order of magnitude of the simulated rates of adsorption in the gas phase indicates that the amount of PAHs adsorbed on micron-size particles cannot be appreciably altered during inhalation of these particles together with the smoke from a cigarette. Whether inhaled by a smoker or not, the amount of PAHs carried into the lung by an asbestos fiber should thus be determined by the concentration of PAHs in the air in contact with this fiber during the hours before inhalation. Any interaction between inhaled asbestos fibers and PAHs in the smoke from the smokers own tobacco should therefore take place within the lung, probably in the surface lining layer of this organ. At typical ambient air conditions, it seems reasonable to assume that diffusional mass transfer is a rate-determining step in comparison with the kinetics of the surface reaction in the transfer of larger size PAHs to airborne particles. The calculated mass transfer rates of PAHs onto a typical ambient air particle are not contradicted by equilibrium conditions suggested between PAHs and particles in the ambient air (*1*).

Once deposited in the lung, the general picture is a rapid desorption of reversibly adsorbed PAHs on both model particles. Still, a concentration of only a few promille of surfactant lipids in the lining layer is likely to raise the average solubility of PAHs several orders of magnitude compared with the simulated pure water, which in turn increases the release rate from outer surfaces of particles considerably. The behavior of the desorbed plume of PAHs is then dependent upon the lipid distribution within the liquid surrounding the particle. If these lipids are distributed as spherical liposomes in the lining layer, then PAHs will be rapidly desorbed along steep concentration gradients close to the particle during a transient phase that most likely will last during the whole desorption process. The properties of a diffusant in such a highly heterogeneous barrier have been described by Higuchi and Higuchi (*31*). If the lipids are instead concentrated in laminar layers in the lining layer (*32*), then the desorbed PAHs are likely to be rapidly dispersed within these layers. Taken together, these data indicate that if the outer surfaces of the particles are wetted instantaneously upon deposition in the lung, then PAHs reversibly adsorbed onto these should be released within a matter of milliseconds rather than seconds. This should apply for the essentially nonporous, submicron fraction of particles in fly ash as well as for nonporous mineral fibers such as man-made mineral fibers and the amphibolic asbestos types. These particles should release their adsorbed PAHs in a concentrated pulse at the moment of deposition in the lung. Porous particles should likewise give a concentrated pulse of PAHs upon desorption from their outer surfaces, but this is followed by a slower phase of pore diffusion, giving an asymptotic tail on the release curve for PAHs that may last for perhaps some minutes. Thus, the dominating picture is a surprisingly rapid release of PAHs even from porous particles when this is governed by diffusional mass transfer.

The other mechanism that may retain PAHs within the particles, a slow surface reaction, can, as mentioned before, in some cases extend the tail on the release curve up to several days (*22*). Adding the two mechanisms gives a two-stage release process of PAHs from a general carrier particle consisting of one intense initial pulse of reversibly adsorbed PAHs at the moment of deposition in the lung followed by a slowly declining leakage of PAHs of very low intensity. This discussion will be focused on the hypothetical consequences of the rapid initial phase, which has not been addressed in the literature before.

The very rapid release of PAHs from nonporous particles seems to limit the hypothesis of a carrier function attributed to mineral fibers for PAHs into the lung tissue (*33*) to porous fibers such as chrysotile asbestos. Solid fibers such as man-made mineral fibers and probably also the amphibolic asbestos types should release their adsorbed PAHs so rapidly upon deposition in the lung that probably little remains even after a direct passage of the surface lining layer. The porous chrysotile fiber, however, should be able to carry its load of PAHs adsorbed onto its interior surfaces across the lining layer and into the epithelium below. Although the amounts of PAHs adsorbed by mineral fibers in the ambient air should be less than the amounts adsorbed by other particle types, their elongated shape may provide a means for introducing this load directly into the epithelial cells, through the documented ability of thin fibers to penetrate cellular membranes (*34*). It should be noted that due to the slow adsorption of PAHs in the ambient air, this effect should be quite the same for smokers as for nonsmokers. Furthermore, this effect has to occur at the instant of deposition of the fiber in the lung, or shortly thereafter. After this phase a direct interaction between mineral fibers and PAHs in the lung is not very plausible, which is also suggested in other papers (*8*).

The calculated release rates of PAHs from micron-size particles are thus considerably faster than those obtained in the experiments cited earlier, where bulk samples of the particles were used. This delay may well be caused by an agglomeration of particles into larger aggregates and by a slow mixing of the vessel used, adding a considerable film resistance to the particle resistance of real interest.

If the release rates of PAHs from micron-size particles in the lung are faster than previously thought, this should have a bearing on our understanding of the mechanisms behind the uptake of these hydrocarbons by the lung. One context where this is of particular interest is in the relation between the release rate of PAHs from their carrier particles in the bronchial

lining layer and the rate of expulsion from the lung of both particles and PAHs with the mucous blanket through ciliary action. A rather extensive clearance of PAHs through mucociliary action during the first half hour or so after their inhalation as aerosols in animals has been attributed to the slow release of the hydrocarbons from their carrier particles (*13*). A comparison with the calculated rates of release shows, however, that this delay probably occurs, if not within the carrier particles, then within the bronchial lining layer itself. It is then interesting to note that there may be a physicochemical mechanism for this slow penetration of lipophilic substances such as PAHs through the bronchial lining layer (*35*). This is due to the highly heterogeneous lipid-aqueous structure of the lining layer, caused by its content of surfactant lipids (*36*). This lining layer constitutes a very heterogeneous barrier toward the diffusion of lipophilic substances with a very high lipid solubility and a very low aqueous solubility. If the rate-determining step in the overall transfer of PAHs from carrier particles to the bronchial epithelium is shifted from the carrier particles to the lining layer itself, then this thin layer is of a great interest in a search for mechanisms that may increase the uptake of aromatic hydrocarbons by the lung. Any change in the lipid-aqueous distribution of the lining layer should be particularly crucial. The creation of a lipid connection through the lining layer could serve as a short circuit for lipophilic substances across this otherwise highly impermeable barrier. One obvious candidate for causing such a phenomenon is the asbestos fiber, with its documented ability not to adsorb PAHs in the lung but to adsorb phospholipids from lung surfactant (*37*). This has lead us to formulate a new hypothesis for the mechanism behind the strong synergism observed between asbestos exposure and tobacco smoking for the induction of bronchial cancer (*38*).

This is of course a rather crude model of the mass transfer of PAHs between micron-size particles and their environment, and the results obtained mirror the uncertainty of both the input data and the model simplifications made. The safety margin for the conclusions drawn from the comparatively slow adsorption of PAHs in the ambient air should be sufficient, but the release of PAHs from particles in the lung is much more complex. One crucial point is then the true environmental distribution of particle-associated PAHs between the two categories where either the rate of the surface reaction is limiting for the release rate of PAHs or the diffusional mass transfer to and from the particle surfaces. Being relevant only for the latter case, the presented model should then, however, indicate the right order of magnitude. Future research thus has to determine the relative magnitude of these two phenomena, preferably through release rate experiments using single, micron-size particles. But more important, the biological impact of these two fractions of PAHs to humans has to be assessed. A clear indication that a rapid initial phase in the release of aromatic hydrocarbons really exists is shown both in our own experiments with mineral fibers (*25*), and with more important carrier particles of PAHs. Subcutaneous fat fluoresces strongly in ultraviolet light within minutes after the administration of tobacco tar on the skin of mice (*39*). A prolonged exposure to PAHs leading to a critical transformation of pulmonary epithelial cells in man is perhaps not the result of a slow release of larger doses of PAHs administered at a few occasions as often is the case in animal experiments. Transformation might well be caused by frequently repeated, high-intensity doses of PAHs released shortly after the deposition of such carrier particles in the lung.

Appendix

Estimation of the Adsorption Coefficient of the Spherical Particle

Typical urban air has a total content of both free and particle-associated BaP of 0.6 to 3.5 ng/m^3 (*24*). If about 3% of this amount is estimated to be free in the gas phase (*1*), this gives a concentration in the gas phase of BaP of, say, 0.05 ng/m^3. If it is further assumed that the concentration of BaP constitutes 5% of the total concentration of the model PAH, which represents the multitude of components present in reality, then the free gas-phase concentration of the model PAH will be $C^G = 1$ ng/m^3.

A typical concentration of particle-bound BaP in urban air is 5 to 50 μg/g (*24*). Assume that this constitutes 5% of the total concentration of the model PAH. Then the particle-bound concentration of the model PAH will be 10^{-4} g/g. If the specific surface area of a typical urban air particle is set at 10 m^2/g, this gives a surface concentration of the adsorbed model PAH on the particles of $C^S = 10^{-5}$ g/m^2 and the adsorption coefficient can thus be estimated to be $K^G = C^S/C^G = 10^{-5}/10^{-9} = 10^4$(m).

Notations

C^G	gas-phase concentration of the model PAH (g/m^3)
C^L	liquid concentration of the model PAH (g/m^3)
C^S	surface concentration of the adsorbed model PAH (g/m^2)
C_i^s	surface concentration before deposition in water (g/m^2)
d	diameter (m)
D	diffusivity of the model PAH (m^2/s)
K	coefficient of the linear adsorption isotherm (m)
N	mass flux of the model PAH (g/m^2,sec)
L	length of pore (m)
Q	total amount of PAH in a pore (g)
r	distance from center of sphere (m)
R	radius of the particles (m)
t	time (sec)
T	time when the moving boundary has reached the bottom of a pore (sec)
x	distance into a pore (m)

Subscripts

app	apparent
iso	reach of isotherm
p	pore
s	sphere
sat	saturation
0	at the surface of the sphere
∞	infinite distance from the surface of the sphere

Superscripts

G	gas phase
L	liquid phase
S	solid surface

This study was supported by the Swedish Work Environment Fund through Grant No. 81-0929.

REFERENCES

1. Yamasaki, H., Kuwata, K., and Miyamoto, H. Effects of ambient air temperature on aspects of airborne polycyclic aromatic hydrocarbons. Environ. Sci. Technol. 16: 189–194 (1982).
2. Eiceman, G. A., and Vandiver, V. J. Adsorption of polycyclic aromatic hydrocarbons on fly ash from a municipal incinerator and a coal-fired power plant. Atmos. Environ. 17: 461–465 (1983).
3. Kotin, P., and Falk, H. L. The role and action of environmental agents in the pathogenesis of lung cancer. Cancer 12: 147–163 (1959).
4. Brockhaus, A., Tomingas, R., Dehnen, W., Pott, F., and Beck, E. G. Das verhalten kanzerogener Kohlenwasserstoffe in der Lunge. Prax. Pneumol. 25: 519–526 (1971).
5. Mossman, B. T., Eastman, A., Landesman, J. M., and Bresnick, E. Effects of crocidolite and chrysotile asbestos on cellular uptake and metabolism of benzo(a)pyrene in hamster tracheal epithelial cells. Environ. Health Perspect. 51: 331–335 (1983).
6. Selikoff, I. J., Hammond, E. C., and Churg, J. Asbestos exposure, smoking and neoplasia. J. Am. Med. Assoc. 204: 106–112 (1968).
7. Harington, J. S. Chemical factors (including trace elements) as etiological mechanisms. In: Biological Effects of Asbestos (P. Bogovski, J. C. Gilson, V. Timbrell, and J. C. Wagner, Eds.), IARC Scientific Publications No. 8, 1973, pp. 304–311.
8. Chang, M. J. W., Singh, N. P., Turturro, A., and Hart, R. W. Interaction of benzo(a)pyrene and chrysotile. In: International Symposium on Polynuclear Aromatic Hydrocarbons 6. Columbus, OH, 1981, pp. 167–176.
9. Shabad, L. M. Experimental cancer of the lung. J. Natl. Cancer Inst. 28: 1305–1317 (1962).
10. Saffiotti, U., Cefis, F., and Kolb, L. H. A method for the experimental induction of bronchogenic carcinoma. Cancer Res. 28: 104–124 (1968).
11. Stenbäck, F. Morphogenesis of experimental lung tumors in hamsters: The effects of the carrier dust. In: Experimental Lung Cancer (E. Karbe and J. F. Park, Eds.), Springer Verlag, New York, 1974, pp. 161–172.
12. Henry, M. C., Port, C. D., and Kaufman, D. G. Importance of physical properties of benzo(a)pyrene-ferric oxide mixtures in lung tumor induction. Cancer Res. 35: 207–217 (1975).
13. Sun, J. D., Wolff, R. K., and Kanapilly, G. M. Deposition, retention and biological fate of inhaled benzo(a)pyrene adsorbed onto ultrafine particles and as a pure aerosol. Toxicol. Appl. Pharmacol. 65: 231–244 (1982).
14. Lakowicz, J. R., Bevan, D. R., and Riemer, S. C. Transport of a carcinogen, benzo(a)pyrene, from particulates to lipid bilayers. A model for the fate of particle-adsorbed polynuclear aromatic hydrocarbons which are retained in the lungs. Biochim. Biophys. Acta. 629: 243–258 (1980).
15. Carslaw, H. S., and Jaeger, J. C. Conduction of Heat in Solids. Clarendon Press, Oxford, 1959.
16. Crank, J. The Mathematics of Diffusion. Clarendon Press, Oxford, 1975.
17. Bird, R. B., Stewart, W. E., and Lightfoot, E. N. Transport Phenomena. John Wiley and Sons, New York, 1960.
18. Lightfoot, E. N. Transport Phenomena and Living Systems. John Wiley and Sons, New York, 1974.
19. Murray, J. J., Pottie, R. F., and Pupp, C. The vapor pressures and enthalpies of sublimation of five polycyclic aromatic hydrocarbons. Can. J. Chem. 52: 557–563 (1974).
20. Griest, W. H., and Tomkins, B. A. Influence of carbonaceous particles on the interaction of coal combustion stack ash with organic matter. Environ. Sci. Technol. 20: 291–295 (1986).
21. Natusch, D. F. S., and Taylor, D. R. Environmental Effects of Western Coal Combustion: Part IV—Chemical and Physical Characteristics of Coal Fly Ash. EPA-600/3-80-094, Environmental Protection Agency, Duluth, MN, 1980, pp. 91–114.
22. Rivin, D., and Atkins, J. H. Sorption of bioactive materials on carbon. Carbon 25: 135–145 (1987).
23. Weber, T. W., and Chakravorti, R. K. Pore and solid diffusion models for fixed-bed adsorbers. Am. Inst. Chem. Eng. J. 20: 228–238 (1974).
24. Katz, M., Sakuma, T., and Ho, A. Chromatographic and spectral analysis of polynuclear aromatic hydrocarbons-quantitative distribution in air of Ontario cities. Environ. Sci. Technol. 12: 909–914 (1978).
25. Gerde, P., and Scholander, P. Adsorption of polycyclic aromatic hydrocarbons onto asbestos and man-made mineral fibers in the gas phase—Influence of the gas humidity. Presented at the Conference on Mineral Fibers in the Non-Occupational Environment, IARC, Lyon, September 8–10, 1987.
26. Perry, R. H., Green, D. W., and Maloney, J. O. Perry's Chemical Engineers' Handbook. McGraw-Hill, New York, 1984.
27. Yada, K. Study of microstructure of chrysotile asbestos by high resolution electron microscopy. Acta Cryst. A27: 659–664 (1971).
28. Yokley, R. A., Garrison, A. A., Wehry, E. L., and Mamantov, G. Photochemical transformation of pyrene and benzo(a)pyrene vapor-deposited on eight coal stack ashes. Environ. Sci. Technol. 20: 86–90 (1986).
29. Nigam, K. K., Singh, R. K., Upadhyay, S. N., Mishra, P., Pandey, G. N., and Shukla, S. D. Short contact dissolution in falling film. Indian J. Technol. 16: 45–49 (1978).
30. Mackay, D., and Shiu, W. Y. Aqueous solubility of polynuclear aromatic hydrocarbons. J. Chem. Eng. Data 22: 399–402 (1977).
31. Higuchi, W. I., and Higuchi, T. Theoretical analysis of diffusional movement through heterogeneous barriers. J. Am. Pharm. Assoc. 49: 598–606 (1960).
32. Ueda, S., Kawamura, K., Ishii, N., Matsumoto, S., Hayashi, K., Okayasu, M., Saito, M., and Sakurai, I. Ultrastructural studies on surface lining layer (SLL) of the lungs: Part III. J. Jpn. Med. Soc. Biol. Interface 15: 67–88 (1984).
33. Mossman, B. T., and Craighead, J. E. Mechanisms of asbestos carcinogenesis. Environ. Res. 25: 269–280 (1981).
34. Woodworth, C. D., Mossman, B. T., and Craighead, J. E. Squamous metaplasia of the respiratory tract—possible pathogenic role in asbestos-associated bronchogenic carcinoma. Lab. Invest. 48: 578–584 (1983).
35. Gerde, P., and Scholander, P. A mathematical model of the penetration of polycyclic aromatic hydrocarbons through the bronchial lining layer. Environ. Res. 44: 321–334 (1987).
36. Lopez-Vidriero, M. T., and Reid, L. Respiratory tract fluid—Chemical and physical properties of airway mucus. Eur. J. Respir. Dis. 61(suppl 110): 21–26 (1980).
37. Light, W. G., and Wei, E. T. Surface charge and hemolytic activity of asbestos. Environ. Res. 13: 135–145 (1977).
38. Gerde, P., and Scholander, P. A hypothesis concerning asbestos carcinogenicity: The migration of lipophilic carcinogens in adsorbed lipid bilayers. Ann. Occup. Hyg. 31: 395–400 (1987).
39. Ermala, P., and Holsti, L. R. Distribution and absorption of tobacco tar in the organs of the respiratory tract. Cancer 8: 673–678 (1955).