A FOOTNOTE ON THE ROLE OF DIFFUSIOPHORESIS
IN LUNG DEPOSITION OF AEROSOLS.1

by

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ABSTRACT

The importance of diffusiophoresis in influencing the deposition of aerosol particles in human and animal lungs has been speculated upon for some time. In this note, we present calculations for the magnitude of this effect based on typical flow behavior in human lungs. Two extreme cases of breathing rate are included, corresponding to sedentary situations and to vigorous exercise. In the upper portions of the respiratory system, the diffusiophoresis velocity caused by diffusion of water vapor into inhaled air should dominate the motion of particles smaller than $\sim 1\mu m$, as compared with Brownian motion. Where diffusion of oxygen and carbon dioxide to and from the surfaces of the lower lung occurs, diffusiophoresis is much weaker but can become significant compared with Brownian diffusion for particles in the $0.1 - 1.0\mu m$ size range, particularly for conditions of heavy exercise.
A. INTRODUCTION

Lung irritation can develop, and diseases can be promoted by the presence of aerosols in the respiratory system. Therefore, it is important to understand the details of physical processes leading to deposition of these small particles on the surfaces of the lungs and the air ducts leading to them. Generally, the deposition of aerosols on any wall can take place by impaction, interception, sedimentation or Brownian diffusion. The first three mechanisms tend to dominate the removal of larger particles of $1\mu$ radius or larger. The inertial forces acting on these larger particles will cause a departure of particle motion from the flow of the suspending gas, which gives rise to capture on a surface. In the second mechanism, the finite size of particles will increase their depositional probability once their trajectories come within a particle radius of a surface. Gravity contributes to deposition of particles in the third mechanism by adding a vertical component of force acting on small particles in a gas stream. The last mechanism usually influences mainly the behavior of small particles of $0.5 - 1\mu$ radius or less. In this size range, particles experience a thermal agitation which is sufficiently large for a significant diffusional motion compared with other effects if there exists a gradient in aerosol concentration near a surface.

In addition to the "classical" processes of deposition listed above, particles may reach surfaces by the influence of certain phoretic forces, which produce drift velocities normal to the surface. One
A known example of such a process is the motion of particles resulting from a thermal force arising in a temperature gradient. Potentially more important to aerosol deposition in the lungs, however, is the diffusiophoretic force. In this phenomenon, small particles can develop a drift velocity in a non-uniform gas with gradients in concentration of one or more gaseous species. The diffusional transport of water vapor, oxygen and carbon dioxide to and from air inhaled into the lungs must involve concentration gradients of these gases. Therefore, diffusiophoresis may have an important supplemental influence on the depositional processes of aerosols in the lungs. Despite the potential significance of this process in contributing to the behavior of aerosols in the lung, few attempts to estimate its importance have appeared in the literature. It is not mentioned at all, for example, in Hatch and Gross' recent monograph (6). And, without reference to any calculations, Davies has dismissed it as unimportant in his introductory remarks in Inhaled Particles and Vapours (3). Only the briefest comment is made about its role in pulmonary processes by Derjaguin and Dukhin (4).

At a conference on depositional processes in the lungs held in January 1968 at the California Institute of Technology, the question of the relevance of diffusiophoresis was brought up again. Unfortunately, none of the participants were aware of any detailed calculations of the importance of the effect in the lungs although it was generally believed to be only of very minor importance in the depositional process.
B. THE THEORY OF DIFFUSIOPHORESIS

Before attempting to estimate the importance of diffusiophoresis to aerosols in the pulmonary environment, let us first briefly review certain aspects of the theory for this phenomenon.

Several years ago, Facy\(^{(5)}\) observed that a dust free space persisted over an evaporating liquid surface. The inhibition of dust fall towards the surface was attributed to a drift of particles away from the surface resulting from a current of vapor molecules leaving the liquid. The theory for explaining this observation and similar ones has been reviewed recently by Waldmann and Schmitt\(^{(7)}\). They have remarked that the diffusiophoretic force is proportional to the gradient in concentration of gaseous species in a non-uniform gas. The proportionality factor depends on the gas dynamic regime in which the particle exists. This regime may be specified by the particle Knudsen number, defined as the ratio of the mean free path of the suspending gas, \(\lambda\), to the particle radius, \(R\). If \(\lambda/R \gtrsim 10\), the particles exist in the free molecule regime, while if \(\lambda/R \lesssim 0.1\), the particles are characterized in the slip flow regime. In between, one defines a transitional regime of Knudsen number. A theory for the transition regime has been proposed recently\(^{(2)}\), but we shall confine out attention principally to the extremes of \(\lambda/R \lesssim 0.1\) and \(\lambda/R \gtrsim 10\).
Free Molecule Theory. For the free molecule case, the force exerted by an isothermal, multicomponent gas on a small spherical particle is (7):

\[ F = -\frac{32}{3} R^2 \sum_i \left(1 + \frac{\pi}{8} a_i \right) p_i \left(\vec{v}_p - \vec{v}_i\right) / c_i, \quad (1) \]

where \( a_i \) are the momentum accommodation coefficients for different gas species with partial pressure \( p_i \). \( (\vec{v}_p - \vec{v}_i) \) is the difference between the particle velocity and the mass velocity of diffusing species \( i \). And \( c_i \) is the average thermal speed of species \( i \), given by

\[ c_i = \left( \frac{8 K_T}{\pi m_i} \right)^{1/2}, \quad (2) \]

where \( K \) is Boltzmann's constant, \( T \) is absolute temperature, and \( m_i \) is the molecular mass of species \( i \).

Of particular interest are two special cases of a diffusing binary gas mixture, where (a) species 1 diffuses through a stagnant species 2 and (b) equimolar counter diffusion. Then in a steady state for case (a) \( \vec{v}_2 = 0 \), and

\[ \vec{v}_1 = -\frac{\chi_1}{\chi_2} \cdot \frac{1}{\lambda_2} \cdot D \nabla \chi_1, \quad (3) \]

Here

\[ \frac{\chi_2}{\chi_1} = \frac{\left(1 + \frac{\pi a_1}{\bar{\theta}}\right) \sqrt{m_1}}{\left(1 + \frac{\pi a_2}{\bar{\theta}}\right) \chi_1 \sqrt{m_1} + \left(1 + \frac{\pi a_2}{\bar{\theta}}\right) \chi_2 \sqrt{m_2}}, \quad (4) \]
\(x_1\) refers to the mole fraction of 1 in the gas mixture, and \(D\) is the (binary) mass diffusivity of the gas species. In case (b), the average molecular velocity in the gas is zero, and for \(a_1 = a_2 = 1.0\),

\[
\vec{v}_p = -\frac{\sqrt{m_1} - \sqrt{m_2}}{x_1 \sqrt{m_1} + x_2 \sqrt{m_2}} D \nabla x_1,
\]

From Eq. (3) and (5) we note that the particle moves with a drift proportional to the gradient in concentration of a diffusing species, in the same direction as the average mass velocity of the gas mixture, equal to:

\[
\vec{v} = \sum_i \frac{m_i}{\sum m_i} \frac{x_i \vec{v}_i}{x_1 \sqrt{m_1} + x_2 \sqrt{m_2}}
\]

**Slip Flow.** If the spherical particle is relatively large, it will still experience a force in the presence of a concentration gradient, but the proportionality factor will differ from that estimated in the free molecule regime. In the case of a particle suspended in a resting carrier gas (species 2) through which a vapor diffuses (species 1), Waldmann and Schmitt\(^{(7)}\) give:

\[
\vec{v}_p = -\left(1 + \sigma_2 x_2 \right) \frac{D}{x_1} \nabla x_1,\]

\(^{(7)}\) Waldmann and Schmitt.
where the subscript \( \sigma \) denotes the gradient in mole fraction of 1 at a large distance from the particle. The slip correction factor has the form

\[
\tau_{l} = \frac{A}{m_{1} + m_{2}} \cdot \frac{m_{1} - m_{2}}{d_{1} + d_{2}} + B \cdot \frac{d_{1} - d_{2}}{d_{1} + d_{2}}
\]

(8)

where \( A \) and \( B \) are empirical constants, equal to 0.95 and -1.05 respectively, and \( d_{1} \) is the molecular diameter of species 1.

For equimolar counter diffusion, the velocity of the large particle is:

\[
\vec{v} = -\tau_{l} D \nabla x_{1} \omega .
\]

(9)

The results for the transitional regime in Knudsen number, \( 0.25 \leq \text{Kn} \leq 10 \), must be bounded by the calculations at the limit represented by Eqs. (3) to (9). Thus Eqs. (3) to (9) provide a basis for the subsequent calculations of the magnitude of the diffusiophoretic force in the pulmonary environment.

C. APPLICATION TO AEROSOL BEHAVIOR IN THE LUNGS

When air is inhaled into the lungs, it becomes saturated with water vapor rather quickly. Evidence\(^{1}\) indicates that the air is saturated by the time it has passed through the nasal and airway passages into the lower lung. The fresh air penetrates the regions of the terminal bronchi in the lower lungs, where transfer occurs
by a slow circulation or gaseous diffusion into the respiratory bronchi and the alveolar ducts. In this zone, oxygen diffuses to the walls of the alveolar sacs where it is absorbed. At the same time, carbon dioxide diffuses outwards from these surfaces to be removed later during exhalation.

As air is breathed, suspended particles are transported down from the nose into the airway passages and into the lower lung. By impaction, interception and sedimentation, particles larger than about a micron in radius are trapped in the upper portions of the respiratory system while particles smaller than \( \sim 0.5 \mu \) in radius are believed to penetrate as far as the alveolar sacs before deposition by Brownian diffusion to the alveolar walls. Brownian diffusion, of course, will also be involved to an extent in the (aerosol) concentration boundary layers formed along the walls of the upper respiratory system as air flows in and out of the lungs.

As far as the influence of diffusiophoresis is concerned, two distinctly different regimes of behavior can be identified. In the first, diffusional transfer of particles towards the walls of the nasal and airway passages must be accompanied by a diffusiophoretic drift away from these surfaces with the transfer of water vapor to the incoming air. The second regime involves diffusiophoresis induced by carbon dioxide transfer away from the surfaces of the alveoli into saturated air, and oxygen transfer towards the surfaces of the lower lung.
In the following calculations, we shall assume that the total gas pressure is one atmosphere, or \( A \approx 6 \times 10^{-6} \) cm, so that the two extremes of Knudsen number will deal with particles in the limiting sizes of about 1 \( \mu \) (slip flow) and 0.01 \( \mu \) (free molecule flow). Based on data typical of gas transport in the lungs \(^{(1, 6)}\), we shall make estimates of the diffusiophoretic velocity for the extremes of sedentary breathing, and virorous exercise. Comparison of these calculations with typical velocities for Brownian diffusion will give an illustration of the relevance of diffusiophoresis to aerosol deposition in the lungs. Although calculations for particles in the transition regime (\( R \approx 0.1 \mu \)) cannot be made accurately at this time, values are included with extreme cases based on slip flow theory for comparison. For the purposes of this study, the characteristic velocity of Brownian diffusion \( v_B \) will be estimated by the ratio

\[
v_B \approx \frac{D_p}{L}
\]

where \( D_p \) is the Brownian diffusivity of the particle and \( L \) is a characteristic diffusion length such as a tube radius or a thickness of the concentration boundary layer for particles.

**Conditions in Upper Lung System.** Evidence from physiological studies of respiration in the lung system indicates that the inhaled air stream becomes saturated with water vapor by the time it reaches the alveolar ducts. This requires a rather substantial
transfer of water vapor to the entering air from the nose to the lower bronchi so that one expects that the diffusiophoretic force may be rather large in at least a portion of this region.

As air travels down to the alveoli through the upper respiratory system, particles of \( \leq 1 \mu \) radius or less will diffuse towards the passage walls through a concentration boundary formed as particles are absorbed on the walls. The characteristic thickness of the boundary layer for particle concentration will be very thin, the order of 0.1 mm. Compensating for the diffusional motion of particles towards the moist walls will be a diffusiophoretic drift away towards the core flow of air. To estimate the magnitude of this draft, we assume that the concentration of water vapor in the alveolar air corresponds to its value at saturation for a body temperature of 98.6\(^\circ\)F. It is further assumed that the inhaled air is saturated with water vapor without mixing with moist air in the alveoli.

Taking the characteristic volume flow of air to be the tidal volume during a normal breathing cycle, it is possible to estimate the net flux of water vapor from the walls of the upper lung system to the inhaled air stream. The tidal volume is taken from Hatch and Gross\(^\text{(6)}\) to be about 0.5 liter of air with breathing cycle of 20 times/minute. Their tabulated data indicates also that water vapor will be transferred over a surface area of about 500 cm\(^2\) if we include as the transfer zone all regions from the trachea to
the bronchiole respiratorii. We further note that the difference between sedentary conditions and heavy exercise, corresponding to a work rate of 1600 kg-m/min, is an order of magnitude increase in gas flow\(^{(6)}\).

With the above assumptions, and the further assumption that water vapor diffusion into air corresponds to case (a), diffusion of species 1 through stagnant species 2, the vapor flux and the diffusiophoretic velocity can be estimated. Values for two different ranges of particle size are listed in Table 1 along with a characteristic diffusion velocity given by Eq. (9).

For sedentary breathing, we find for the region of the upper lungs that the diffusiophoretic velocity directed away from the walls of the upper pulmonary system should be somewhat less than the diffusion speed for very small particles, but may exceed the diffusion velocity for particles in the 0.1 - 1\(\mu\) size range in dry climates. Only in rather wet climates where the relative humidity of the atmosphere exceeds \(\sim 90\%\) will diffusiophoresis by water become unimportant for the larger particles in the diffusional regime of deposition. Under conditions of heavy exercise, the assumption of complete saturation of inhaled air, the diffusiophoretic effect becomes important for inhibiting deposition in the upper pulmonary passages for particles of all sizes below \(\sim 1\mu\) radius.

This conclusion is significant in that it suggests that nature provides a substantial barrier to particle deposition in the
upper portions of the respiratory system for all but the most humid climates. This is quite consistent with the well known observation(1) that particles smaller than \( \sim 1 \mu \text{m} \) radius are deposited primarily in the lower lungs.

The importance of diffusiophoresis in the zone of water vapor transport also may be of interest because it may restrict to a certain extent the applicability of many recent calculations for transport of small particles by convectional diffusion for removal in the upper part of the respiratory system. For the initial inflow of air through the nasal passages down to the trachea, the diffusiophoretic effect should be most intense because the gradient in water vapor is likely to be strongest here. In this zone, diffusiophoresis should most strongly inhibit diffusional deposition. Further down in the bronchi, the incoming air will become more and more moist. Therefore, deposition resulting from Brownian diffusion should continually increase in importance the lower in the respiratory system the air penetrates.

**Particle Motion in the Lower Lungs.** Since the incoming air becomes saturated by the time it reaches the alveolar ducts and sacs, diffusiophoresis in the lower lungs can only take place through the action of the diffusion of oxygen to the alveolar walls and of carbon dioxide away from these surfaces. In this regime, estimation of the magnitude of the diffusiophoretic effect is more difficult because of the uncertainties in the mechanism of gas mixing, and because of the fact that transport occurs
with the involvement of more than one gaseous component. If the bulk mixing of gases is disregarded, one can visualize two extremes in this regime for purposes of calculation. In the first, one can consider that carbon dioxide is diffusing through pure oxygen in such a way that there is roughly equimolar counter-diffusion (case b). In the other extreme, one can neglect collisions between oxygen and carbon dioxide molecules and consider the two species as diffusing "independently" through a stagnant carrier gas. In this latter situation, the diffusiophoretic velocity will be given roughly by the vector sum of the two velocities calculated from Eq. (7) and (7) for oxygen and carbon dioxide diffusing through stagnant nitrogen.

To make estimates based on the two extremes, we have adopted Hatch and Gross' values for oxygen consumption and carbon dioxide production for calculation of the molal fluxes of these gases. Calculations were carried out for the situations for sedentary breathing, and for heavy exercise. Data for the alveolar concentration of $O_2$ and $CO_2$ were taken from Best and Taylor. The area of the alveolar transfer surface is $1.6 \times 10^5 \text{ cm}^2$, and the characteristic diffusional length is taken as the radius of the alveolar duct, about 0.01 cm. The calculated values of $\left| \nabla \mathbf{v} \right| = v_p$ for the extreme sizes of particles are listed with other pertinent parameters in Tables 2 and 3.

We find from the computations that the diffusiophoretic effect is likely to play only a minor role in the behavior of aerosols in the lower lungs for sedentary conditions. In the case of
particles with 0.1\(\mu\) radii or less, diffusiophoresis evidently can only be about 10 - 15\% of the diffusion speed even for conditions of heavy exercise. The influence of deposition will be larger for the larger particles with 1\(\mu\) radius perhaps amounting to only 20\% of the diffusional speed in this case (Table 3). Under conditions of heavy exercise, however, the diffusiophoretic velocity can become appreciable for particles of about 1\(\mu\) size, with a magnitude the order of diffusional speed.

An interesting feature of this problem as revealed in the tabulated data is the strong dependence of the direction for the drift velocity on the assumed model for the gas diffusion. For conditions of equimolar counter diffusion of CO\(_2\) through pure O\(_2\), the theory predicts that the diffusiophoretic velocity will be directed away from the alveolar surfaces. However, for the case of "independent" diffusion of CO\(_2\) and O\(_2\) through N\(_2\), the observations reported in Hatch and Gross\(^{(6)}\), Table 2.1, indicate an unbalanced molar flux of oxygen and carbon dioxide exists (see also Table 3). Despite the cross-over in magnitudes of O\(_2\) and CO\(_2\) production and consumption, oxygen diffusion will dominate diffusiophoresis, and the drift velocity will be directed towards the alveolar walls with this latter assumption (Table 3). It is likely that this latter situation more closely describes the gas diffusion conditions in the lower lung. Therefore, with heavy exercise, one would expect some enhancement of particle deposition over sedentary
conditions. To our knowledge, this effect has not been verified experimentally. However, Hatch and Gross do show curves of increase in deposition with breathing rate, which may be related to diffusiophoresis.

To make the calculations, we assumed a surface area in the lower lungs for an average "normal" individual. If the effective surface area of the alveolar is reduced, for example, by a disease like emphysema, the diffusiophoretic effect could become much more significant. Assuming that it is correct to conclude the diffusiophoresis in the alveolar enhances diffusional deposition of aerosols, an emphysema patient who smokes or lives in a polluted, urban area could suffer more serious lung damage by aerosol interaction with tissue than a normal, healthy individual.

Because the diffusiophoretic effect can become appreciable under some conditions of ventilation of the lower lungs, it might be useful to conduct some laboratory experiments in glass vessels to check which of the extreme assumptions, equimolar counterdiffusion, or parallel diffusion in a stagnant gas is more applicable to the pulmonary system.

**Influence of Inert Gases.** In the case of parallel diffusion of CO$_2$ and O$_2$ in a stagnant carrier gas, one wonders if there can be a significant modification in the predicted diffusiophoretic speed if nitrogen were replaced by gases of different molecular weight and diameter of species 1 and 2, the net effect of changing
\( \sigma_{12} \) for \( O_2 \) and \( CO_2 \) tend to cancel each other out, so that the use of extremely different carrier gases does not appear to be a useful practical method for modifying the diffusiophoretic effect.

D. CONCLUSIONS

Based on the computations outlined above, it appears that the diffusiophoretic effect is most significant in retarding diffusional deposition of \( 0.1\mu \) and larger particles in the upper portions of the respiratory system. In this region, diffusiophoresis involves the diffusion of water vapor into the inhaled air. In the lower parts of the lung, diffusiophoresis depends on the diffusion of carbon dioxide and oxygen from and to the alveolar surfaces. Here the direction of the diffusiophoretic velocity is uncertain, but its magnitude appears to become appreciable compared with Brownian diffusion only for \( 0.1\mu \) or larger particles inhaled during periods of heavy exercise. It is likely that diffusiophoresis proceeds towards the alveolar walls because of the stronger influence of oxygen diffusion on the particles.

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REFERENCES


Estimated magnitude of diffusiophoretic velocity in the upper respiratory system compared with a characteristic velocity for Brownian diffusion. Assumed surface area of nasal passages to bronchi 500 cm². Equilibrium partial pressure of water at 98.6°F is 49.16 mm Hg. Inhaled air bone dry.

<table>
<thead>
<tr>
<th>Particle Radius ((\mu))</th>
<th>Molar Flux (cm-sec⁻¹)</th>
<th>Particle Diffusion Velocity (cm-sec⁻¹)</th>
<th>Diffusiophoretic Velocity (cm-sec⁻¹)</th>
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<tr>
<td></td>
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<td>Sedentary</td>
<td>Exercise</td>
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<td>1.4 \times 10⁻²</td>
<td>1.3 \times 10⁻⁵</td>
<td>1.3 \times 10⁻³ 1.3 \times 10⁻²</td>
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<td>1.3 \times 10⁻²</td>
<td>1.1 \times 10⁻³ 1.1 \times 10⁻²</td>
</tr>
</tbody>
</table>
TABLE 2

The diffusiophoretic effect in breathing pure oxygen:

Comparison between diffusiophoretic velocity and diffusion velocity
for equimolal counterdiffusion of carbon dioxide and oxygen.

Equilibrium partial pressures in alveolar air: 40 mm Hg (CO\textsubscript{2}), and
100 mm Hg (O\textsubscript{2}). Carbon dioxide production from Hatch and Gross, Table
2.1. Alveolar area: 1.6 x 10\textsuperscript{5} cm\textsuperscript{2}. Exercise rate: 1660 kg-m-min\textsuperscript{-1}.

<table>
<thead>
<tr>
<th>Particle Radius (\mu)</th>
<th>Carbon Dioxide Production (cc-min\textsuperscript{-1})</th>
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<th>CO\textsubscript{2} Molal Velocity (cm-sec\textsuperscript{-1})</th>
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<tr>
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<td>3598</td>
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</table>
TABLE 3

Comparison between diffusionphoretic velocity and diffusion velocity for the case of \( \text{O}_2 \) and \( \text{CO}_2 \) diffusing independently through stagnant \( \text{N}_2 \).

Equilibrium partial pressures in alveoli: 40 mm Hg (\( \text{CO}_2 \)), 100 mm Hg (\( \text{O}_2 \)).

Carbon dioxide production and oxygen consumption from Hatch and Gross, Table 2.1. Alveolar area: \( 1.6 \times 10^5 \) cm\(^2\). Exercise rate: 1660 kg m\(^{-1}\) min\(^{-1}\).

<table>
<thead>
<tr>
<th>Particle Radius (( \mu ))</th>
<th>Carbon Dioxide Production (cc-min(^{-1}))</th>
<th>Oxygen Consumption (cc-min(^{-1}))</th>
<th>Velocity (cm-sec(^{-1}))</th>
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