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Urban Aerosol Toxicity: The Influence of Particle Size

Particle size, adsorption, and respiratory deposition profiles combine to determine aerosol toxicity.

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It is now generally accepted that particle size is a major factor in determining the toxic effects of airborne particulate matter (*1, 2*). For example, pulmonary flow resistance resulting from inhalation of zinc ammonium sulfate aerosols increases as the particle diameter decreases (*3*) in the range 1.4 to 0.29 micrometers. Similarly, coarse manganese dioxide dust can be tolerated for many years without adverse effects whereas fine fume, in which 80 percent of the particles are less than 0.2 μm in diameter, produces pneumonitis (*4*).

Recent studies have shown that a number of potentially toxic trace species, including lead, cadmium, antimony, selenium, nickel, vanadium, zinc, cobalt, bromine, manganese, sulfate, and benzo[*a*]pyrene, predominate in small, lung depositing, particles in most urban aerosols (*5–9*) and in the smallest particles emitted from high-temperature combustion sources (*10, 11*). Furthermore, it has been shown that the tissues extract trace metals much more efficiently from the small particles that become deposited in the lung than from the larger particles that are deposited higher up in the respiratory system (*12*). The toxic effects produced by respirable particles depend, of course, on the chemical species that they contain. These effects may be manifest either locally in the respiratory tract, as in the case of pneumonitis produced by fine smog particles, or systemically, as in the case of lead poisoning resulting from inhalation of lead-containing aerosols. In either case, however, small particles are generally more toxic than large.

Clearly, the effective toxicity of respirable particles will depend upon the nature of the toxic species present, on its size distribution in an aerosol, and on the efficiency with which it is extracted in the region of respiratory deposition. It is our intention in this article to illustrate the collective effects of these factors in determining the overall toxicity of ambient aerosols. In particular, we emphasize the importance of volatile species that are adsorbed onto particle surfaces.

Deposition and Extraction Characteristics of Aerosols

A number of theoretical and experimental studies have demonstrated that inhaled particles are deposited in different regions of the respiratory system depending on their effective aerodynamic size (*2*). There is some disagreement, however, about the exact dependence of deposition efficiency on particle size; in Fig. 1 we show a somewhat simplified summary of the available information. It should be noted that particle size is presented in terms of the diameter of an equivalent spherical particle of unit density. Although many particles of interest are indeed spherical (*13*), the wide range of densities encountered means that particle diameters plotted in Fig. 1 do not correspond to physical diameter as determined by a Coulter counter or an electron microscope.

Particles that are deposited in the nasopharyngeal and tracheobronchial regions of the respiratory tract do not remain there for long. Such particles are normally removed to the pharynx, often by cilial action, and swallowed within a matter of hours. Consequently, extraction of toxic species from these particles takes place predominantly in the stomach where their residence time is short. Exceptions to this rule occur in the case of particles whose constituents either are extremely soluble or paralyze cilial action (*14*). On the other hand, particles deposited in the pulmonary region may remain there for weeks or even years (*15*) in intimate contact with approximately 30 square meters of the microscopically thin alveolar membranes which separate the bloodstream from inhaled air (*1*).

On the basis of residence time alone one would expect the extraction of toxic species to take place much more efficiently in the lung than in the stomach. This is confirmed in practice since it has been estimated that 60 to 80 percent of the lead is extracted from particles deposited in the lung, whereas only 5 to 15 percent is extracted from those deposited in the stomach (*12*). Residence time is probably not the only factor controlling extraction efficiency, however, since even comparatively insoluble species are extracted at a surprising rate in the pulmonary region. For example, barium sulfate retained in the lung is extracted into the bloodstream in a matter of days (*16*) whereas ingested barium sulfate is not significantly absorbed (*17*). At least part of this phenomenon is probably due to particle size since the rate of extraction per unit mass depends on

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Table 1. Typical size parameters of urban aerosol components.

Species	Mass median diameter (μm)	Standard deviation	Reference	Comments
Iron	2.7	2.9	(*5*)	One-year average for six eastern U.S. cities
Lead	0.56	4.1	(*5*)	One-year average for six eastern U.S. cities
Zinc	1.03	2.06	(*5*)	One-year average for Chicago
Barium	1.95	3.54	(*5*)	Denver quarterly average
Noncarbonate carbon	0.6		(*21*)	Los Angeles photochemical smog, 90 percent of particles less than 0.6 μm
Benzo[α]-pyrene	0.15		(*7*)	Budapest
SO_4^{2-}	0.11 to 1.1	7.8	(*34*)	Range in Los Angeles and San Francisco, diameter varies with relative humidity

the surface-to-volume ratio of a particle. Chemical effects and membrane transport characteristics may also play a determining role.

Enrichment of Toxic Species in Respirable Aerosols

Many toxic trace elements present in airborne particles are derived from high-temperature processes such as occur in internal combustion engines, power plants, blast furnaces, metallurgical smelters, cement kilns, and municipal incinerators (*11*). It has now been established that many of these elements, such as lead, zinc, arsenic, cadmium, antimony, mercury, selenium, and sulfur are enriched in urban aerosols by as much as 100- to 1000-fold over natural crustal abundance (*18*). In addition, it has been shown that the elements lead, thallium, antimony, selenium, arsenic, nickel, chromium, zinc, and sulfur increase markedly in concentration with decreasing particle size in airborne fly ash leaving a coal fired power plant (*10, 11, 19*). (The elements bismuth, tin, cobalt, iron, manganese, vanadium, titanium, calcium, potassium, magnesium, aluminum, beryllium, carbon, and silicon showed no strong particle size dependence.)

The reasons why some, but not all, elements increase in concentration with decreasing particle size are not firmly established. However, it has been suggested (*10*) that an element (or one of its compounds) is volatilized in a high-temperature (1300° to 1600°C) combustion zone and is then adsorbed onto the large available surface area of entrained particles. Models based on this volatilization-adsorption concept predict that the average concentration per unit mass, $\overline{C}_X$, of a volatilizable species X should depend on particle diameter, D, according to an equation of the form

$$\overline{C}_X = \overline{C}_0 + \overline{C}_A D^{-1} \qquad (1)$$

Here $\overline{C}_0$ is the average concentration of X instrinsic to the particle and $\overline{C}_A$ is related to the average surface concentration added by adsorption. While this equation incorporates a number of simplifying assumptions, it does describe the observed dependence of element concentration on particle size within the errors associated with sampling and analysis. Furthermore, scanning electron microscopic analyses of individual fly ash particles etched with argon ions (*20*) confirm that nickel, chromium, and zinc are considerably more concentrated on particle surfaces than in their interior. (Other adsorbed elements were not sufficiently concentrated to be observed by this method.) This surface adsorption is an extremely important consideration from the standpoint of environmental health because it is the particle surface which is immediately in contact with extracting fluids and body tissues.

The dependence of trace element concentration on particle size described by Eq. 1 for particles derived from an individual emission source does not persist in the atmosphere, however. This is because the mixing of particles derived from a wide variety of emission sources, each with its own characteristic trace element distribution, produces considerable modification of individual source profiles. Particle size distributions have been established for a number of elements (*5, 8*) and organic species (*7, 21*) in ambient urban aerosols. These distributions vary somewhat in different urban areas but, when averaged over periods of a few months, most elements exhibit an approximately log-normal distribution with respect to particle diameter. This dependence can be described (*5*) by a normalized distribution of the form

$$\frac{dM}{d(\ln D)} = \frac{1}{(2\pi)^{1/2} \ln \sigma_g} \exp\left[\frac{-(\ln D/D_g)^2}{2\,(\ln \sigma_g)^2}\right] \qquad (2)$$

where D is particle diameter, σ_g is the geometric standard deviation of the size distribution, and M and D_g are, respectively, the mass and mass median diameter of the element or species in question. Some representative values of D_g and σ_g are presented in Table 1. It can be seen that the bulk of most toxic elements is concentrated in particles whose diameters are somewhat larger than the mass median diameter of the total aerosol ($\sim$ 0.5 μm). This is because most emission sources (with the notable exception of automobiles) contribute relatively large particles (0.5 to 10.0 μm) (*5, 22*) to the ambient aerosol.

At this point it is meaningful to consider the influence of surface adsorption on the mass median diameter of the adsorbed species. Thus, preferential adsorption onto small particles, as described by Eq. 1, will reduce the mass median diameter of the adsorbed species below that of the adsorbing substrate particles. A quantitative relationship describing the size distribution of the adsorbed species, X, can be obtained by combining Eqs. 1 and 2 to give

$$\frac{dM_X}{d(\ln D)} = \frac{1}{(2\pi)^{1/2} \ln \sigma_g} \left\{ \overline{C}_0 \exp\left[\frac{-(\ln D/D_g)^2}{2\,(\ln \sigma_g)^2}\right] + \overline{C}_A \exp\left[\frac{(\ln \sigma_g)^2}{2}\right] \exp\left[\frac{-(\ln D/D_g + \ln^2 \sigma_g)^2}{2(\ln \sigma_g)^2}\right] \right\} \qquad (3)$$

Here D_g and σ_g refer to the distribution of the adsorbing substrate particles and $\overline{C}_0$ and $\overline{C}_A$ are as defined for Eq. 1. Equation 3 does not provide a simple analytical expression for the mass median diameter, $D_g(X)$, of the adsorbed species, X, except when $C_0 = 0$ (that is, when the adsorbing particle contains none of the species X other than in a surface adsorbed layer). In this case it can readily be shown (*23*) that

$$\ln D_g(X) = \ln D_g - (\ln \sigma_g)^2 \qquad (4)$$

Since it has been demonstrated that many volatilizable elements are most concentrated (on a mass per mass basis) in the smallest particles, surface adsorption may account in part for the wide variation in mass median diameters of species present in urban aerosols.

Just as adsorption or condensation of species volatilized in a high-temperature process may coat source particulates with a surface layer of toxic trace elements, so also can adsorption or condensation of gaseous species from the atmosphere produce a high surface concentration on particles which are already airborne. In fact, deposition of hydrocarbons onto the surface atmospheric particles has been implicated in the Los Angeles aerosol (*24*). However, since deposition occurs after the atmospheric size distribution has achieved an approximately steady state, one might expect particulate hydrocarbon concentrations to obey Eq. 1. This is the case for two out of the three aerosol samples collected by Mueller *et al.* (*21*) in Greater Los Angeles.

Respiratory Deposition and Extraction of Toxic Species

Comparison of Fig. 1 with the data in Table 1 shows that the mass median diameters listed cover the range of sizes which most critically determine where in the respiratory tract a particle will be deposited. Consequently, quite small differences in mass median diameter may produce profound differences in the regions of predominant deposition of individual species.

The deposition function describing the dependence of deposited mass of any toxic species which is retained in each region of the lung is obtained by multiplying the particle size distribution function, Eq. 2, by the deposition efficiency, E, from Fig. 1. Thus,

$$\left[\frac{dM}{d(\log D)}\right]_{\text{deposited}} = 2.3E\left[\frac{dM}{d(\ln D)}\right]_{\text{airborne}} \qquad (5)$$

Plots of this function are presented in Fig. 2, a–c, for iron, lead, and benzo[*a*]pyrene present in the urban aerosol, respectively. The fractions of the inhaled aerosol deposited in each respiratory region are obtained by integrating the curves in Fig. 2, a–c, and are presented in Table 2. It should be noted that the abscissas in Fig. 1 and Fig. 2, a–c, are compatible because all the data are based on aerodynamic size.

It is clear from these results that for a typical ambient aerosol, iron is deposited mainly in the upper (nasopharyngeal and tracheobronchial) regions of the respiratory tract from where it is readily removed to the stomach. Somewhat more than half of the lead entering the respiratory tract is deposited in the pulmonary region; and the carcinogenic benzo[*a*]pyrene is deposited almost exclusively in the pulmonary region.

Table 2. The percentage of inhaled iron, lead, and benzo[*a*]pyrene deposited in each respiratory region (from Fig. 2).

Species	Respiratory tract region		
	Upper		Lower
	Naso-pharyngeal	Tracheo-bronchial	Pulmonary
Iron	48	7	22
Lead	17	6	32
Benzo[*a*]pyrene	5	7	39

While Fig. 2, a–c, describe the average deposition efficiency in the major regions of the respiratory tract, no account has been taken of localized variations in particle deposition within a single region. Indeed, it has been suggested (*25*) that particle deposition efficiency is greatly enhanced at bifurcations in the bronchial tubes and lower airways. For this reason such deposition "hot spots" may be especially susceptible to the presence of toxic species. In this regard it is noteworthy that certain lung diseases originate at bifurcations.

The importance of surface adsorption or condensation on the region of deposition in the respiratory tract can be illustrated as follows: If the element zinc, for example, were uniformly distributed throughout all iron-containing particles in an aerosol, it would have the same mass median diameter as iron, that is, approximately 2.7 μm; the respiratory deposition function of zinc would therefore be the same as that depicted in Fig. 2a for iron. However, adsorption or condensation of zinc onto the surface of these same particles according to Eq. 1 would result in a mass median diameter of approximately 1 μm for zinc; its respiratory deposition function would then be as shown in Fig. 2d. Comparison of (a) and (d) in Fig. 2 demonstrates the marked effect of surface adsorption on respiratory deposition characteristics. The effect is even more pronounced in the case of organic species which may adsorb onto particle surfaces from the atmosphere.

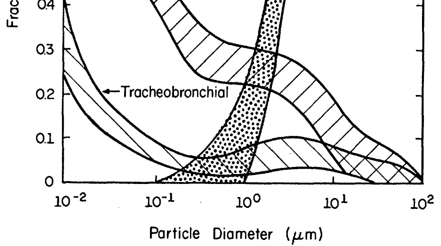

Fig. 1. Respiratory deposition efficiencies for inhaled particles.

Respiratory deposition profiles similar to those presented in Fig. 2 can be developed for any species whose atmospheric particle size distribution has been experimentally determined. In this regard it should be strongly emphasized that the assumption of a log-normal distribution or, indeed, of any explicit analytical size distribution function for individual elements, is by no means a necessary part of the foregoing arguments. In fact, recent information (*26*) suggests that the total mass of most urban aerosols does not conform to a log-normal distribution at all but is more closely approximated by a bimodal distribution. The present treatment has, however, been developed in terms of a log-normal distribution for the following reasons:

1) The great majority of data on the particle size distributions of individual elements in atmospheric aerosols have been obtained (*5*) by means of cascade impactors with insufficient resolution to give precise definition of bimodal distributions.

2) The available evidence suggests that many trace elements contribute mainly to the larger particle size mode of the total mass distribution whereas the smaller size mode consists largely of particles, such as sulfate particles, derived from gas-to-particle conversions. (Benzo[*a*]pyrene and other polynuclear aromatic hydrocarbons also contribute mainly to the smaller size mode.) Thus, there is reasonable circumstantial evidence to suggest that while the total aerosol mass may be distributed bimodally, most toxic species probably predominate in a single mode.

3) Even if toxic species are

distributed bimodally, each mode conforms quite closely to a log-normal distribution so that the assumption of such a distribution may offer a convenient, mathematically explicit way of treating the data.

The foregoing remarks notwithstanding, it must be emphasized that cascade impactors leave much to be desired in terms of their ability to establish aerodynamic particle size distributions. Errors are introduced by particle "bounce off," particle reentrainment, limited size definition, and overlap of particles of a given size onto more than one collection plate. This last factor can be especially important (*27*) where a chemical species has a marked dependence on particle size such as that described by Eq. 1. Furthermore, Lee *et al.* (*5*) themselves noted that their data approached log-normal statistics more closely after being averaged over a period of months during which time a wide variety of meteorological conditions had been encountered. It is entirely possible, therefore, that this averaging may mask the actual aerosol distribution.

If, however, a unimodal log-normal distribution is assumed for individual chemical species, the available data (*5–8, 11*) show that the pattern of deposition of magnesium and barium is similar to that of iron in Fig. 2a, the deposition profile of vanadium is similar to that of lead in Fig. 2b, and the deposition profile of noncarbonate carbon is similar to that of benzo[*a*]pyrene in Fig. 2c. Zinc, nickel, copper, manganese, cadmium, chromium, and tin have similar deposition profiles to the example of zinc adsorbed on iron shown in Fig. 2d.

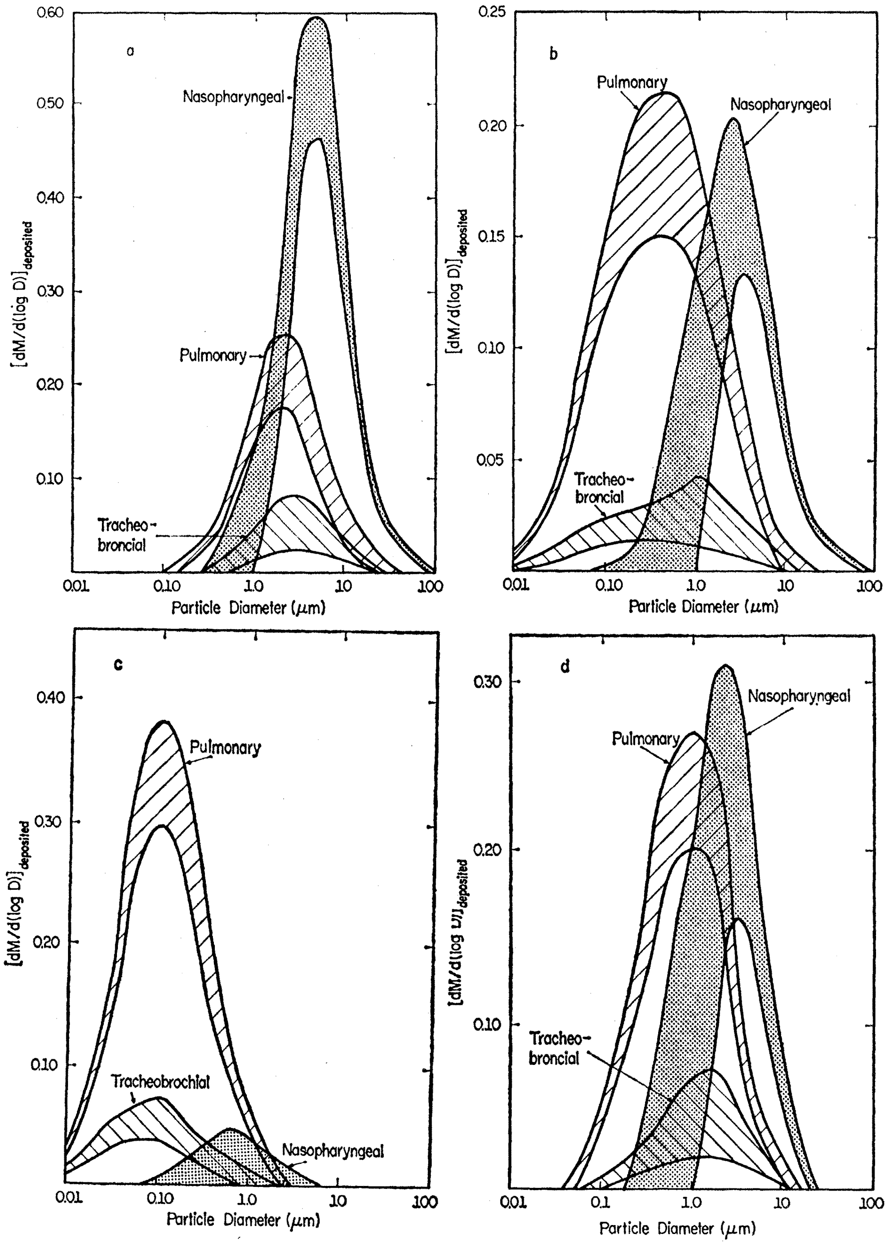

Fig. 2. Respiratory deposition profiles of (a) iron, (b) lead, and (c) benzo[*a*]pyrene in an urban aerosol. In (c), a typical value of $\sigma_g = 3.0$ was assumed since no experimental values were available. (d) Respiratory deposition of zinc adsorbed onto the iron-containing particles distributed as in (a).

Considerable significance is attached to the pulmonary deposition of benzo[*a*]pyrene and noncarbonate carbon. The available information suggests that organic species have a high probability of being adsorbed so that they predominate in particles that become deposited in the pulmonary region of the respiratory tract. Consequently, potentially carcinogenic species present as gases in source effluents or in the atmosphere can be expected to reach the innermost regions of the lung concentrated in particulates.

Absorption of toxic species into the bloodstream appears to be strongly weighted in favor of the mass fraction deposited in the pulmonary region, thereby further enhancing the toxicological significance of small particles. If we assume that 70 percent of the lead deposited in the pulmonary region is extracted and absorbed into the bloodstream, while only 10 percent of that deposited in the other regions is so absorbed, it is possible to determine the dependence of lead entry into the bloodstream on particle size.

For example, the data in Table 2 show that 32 percent of the total inhaled lead will be absorbed with 70 percent efficiency and 6 percent and 17 percent will be absorbed with 10 percent efficiency. This means that 22 percent of the total inhaled lead reaches the bloodstream by absorption through lung membranes while only 2.3 percent enters from the stomach. These figures indicate that an average adult inhaling 20 cubic meters of air per day containing 2 micrograms of lead per cubic meter of air (a typical urban aerosol loading) would adsorb 10 μg/day. By comparison, it is estimated that some 30 μg/day are absorbed from food and water (*14*). These results are in agreement with those of lead isotope labeling studies (*28*) which indicate that approximately 30 percent of the normal daily lead intake comes from inhaled aerosols. Insofar as many individuals are exposed to ambient aerosols containing much more than 2 μg/m^3 of lead, the respiratory intake of this metal cannot be considered trivial.

Care should, however, be taken in making estimates such as these, because hygroscopic particles have been shown to markedly increase their aerodynamic size when exposed to the high relative humidity ($\sim$ 96 percent) of the respiratory tract (*29, 30*). For example, 0.4-μm and 1.0-μm particles can increase to approximately 1.0 μm and 3.0 μm, respectively, within 0.1 second at 37°C and 96 percent relative humidity even when inhaled from air at 80 to 90 percent humidity (*29*). Under such conditions the deposition profiles depicted in Fig. 2 would be altered in that the particle mass being deposited in the pulmonary region would be reduced. Indeed, in one sense, this effect constitutes a natural pulmonary protection mechanism against hygroscopic particles. Unfortunately, little is known about the matrix composition of particles with which toxic species are preferentially associated, so that the importance of this mechanism is unclear. In the case of aerosols derived from inorganic gases (for example, sulfate aerosols) a significant increase in size is probable; however, industrial particulates, which consist mainly of the oxides of silicon, iron, aluminum, calcium, magnesium, and sodium, are expected to experience much smaller size increases. This is clearly an area where considerable research is required.

Although we have only considered toxic species present in particulate matter it is important to recognize that certain species, notably mercury, selenium dioxide, and arsenic trioxide, can also exist in the atmosphere as vapors. Indeed, thermodynamic data (*31*) indicate that at 25°C as much as 80 μg/m^3 of selenium as SeO_2 and 70 μg/m^3 of arsenic as As_2O_3 can exist as vapor before homogeneous condensation to the solid begins. By comparison, urban aerosols have typically been found to contain less than 10 nanograms of selenium and arsenic per cubic meter of air (*6, 32*). It is possible, therefore, that additional amounts of these elements may be present as vapors. Consistent with this suggestion, Pillay and Thomas (*33*) have reported that at least 50 percent of the selenium present in urban air passes through a filter designed to collect all particles greater than 0.1 μm in diameter. Comparable data are not available for arsenic. Irrespective of whether selenium and arsenic are inhaled as vapors or as very small particles, however, their toxicology will undoubtedly involve the pulmonary region of the lung.

Conclusion

The basic thesis developed herein is that particle size is an extremely important parameter to consider when assessing the potential toxicity of species present in urban aerosols, and that surface adsorption or condensation greatly promotes toxicity. In the majority of cases this means that particles that are less than 1 μm in size are of primary importance. Special significance is attached to the organic constitutents of aerosols. Many of these are known or potential carcinogens and most predominate in extremely small particles which become deposited almost exclusively in the lung.

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