

Aerosols – Dusts, Fumes and Mists

Instead of the definitions for total dust and fine dust which were used in the past, from 1996 the new internationally agreed conventions for measuring concentrations of particles (DIN EN 481, 1993) are to be used in the establishment of MAK values. Therefore the definitions of particle fractions and the methods of measuring them have been modified in line with this convention and also extended to apply to all aerosols, that is, not only to dusts but also to fumes and mist.

1 Definitions

1.1 General

Aerosols are multiphase systems of particles (solids or liquids) dispersed in air or in other gases.

Dusts consist of particles of solid matter which have been produced in mechanical processes or have been stirred up and dispersed in gases, in particular in air. The term “dust” is also commonly used colloquially for particles which have been precipitated from the gas phase and deposited on a surface. Here the term “settled dust” will be used for such particles.

Fibrous dusts consist of dust particles for which the ratio of length (l) to diameter (d) is larger than 3:1, the length is larger than 5 µm and the diameter less than 3 µm (see the *List of MAK and BAT Values 1997*, p. 112 and “Fibrous Dust” in Volume 8 of the present series). In these documents, however, the above-mentioned definition of aerosols is not taken into account. This definition describes fibrous dusts as elongated particles (called fibres) of the dimensions given above dispersed in gases, in particular in air. They can be formed during mechanical processing especially of fibrous material (e.g., acicular fibres), during the stirring up of deposited fibres, during erosion of materials containing fibres or during handling of such materials.

Fumes are dispersions of solid matter in gases, in particular in air, which have been formed in thermal or chemical processes. Thermal processes can yield fumes in two ways:

- by condensation from the vapour phase, sometimes in association with chemical reactions (examples: welding fumes, metal (oxide) fumes) or
- by incomplete combustion of organic material and release of impurities that it contains (examples: soot and flue ash).

Chemical processes can also yield fumes (example: the reaction of ammonia with hydrogen chloride). In many cases the primary particles in fumes have diffusion-equivalent

diameters $d_D < 0.5 \mu\text{m}$. Agglomeration—especially at higher fume concentrations—can produce particles with much larger diffusion-equivalent diameters. The agglomerates can be chain-like in structure. When two spheres of the same diameter (d_1) and density of 1 g/cm^3 aggregate, the particle produced has a diffusion-equivalent diameter of $d_D \approx 1.20 \cdot d_1$ (Horvath 1974, 1979).

Mists are dispersions of liquids in gases, in particular in air. They are formed during nebulization of liquids, during condensation from the vapour phase and during chemical processes (example: oil mist, hydrogen chloride in damp air). After sedimentation, the liquid particles of mists lose their particulate form, unlike undissolved solid particles which generally retain their form.

For isometric particles with diameters $d < 0.5 \mu\text{m}$, the deposition in the respiratory tract is caused by diffusion. The diffusion of aerosol particles is controlled by their Brownian motion. This constant random movement of the aerosol particles in a gas is caused by collision of these particles with the molecules of the gas and only takes place when the mass of the aerosol particle is very small. If the motion of the aerosol particles is caused entirely by this mechanism, they are designated as **ultrafine particles** (Bricard *et al.* 1975, Fissan 1986, Kanapilly *et al.* 1982). The particles have diameters $d < 100 \text{ nm}$. The molecular motion of such particles obeys the laws of thermodynamics, for which reason this particle size range is frequently called the thermodynamic range.

1.2 Particle diameters

When estimating the danger for health caused by aerosols, not only the specific toxic effects of the substance, the concentration and the exposure time must be taken into account but also the particle size and particle shape because of their effects on transport and deposition in the respiratory tract. This distinguishes aerosols clearly from gases and vapours. Uptake into the organism takes place mainly via the inhaled air. Transport and deposition of the particles in the respiratory tract are determined by the aerodynamic diameters and the diffusion-equivalent diameters (Davies 1982, Heyder *et al.* 1980, Lippmann and Albert 1969).

1.2.1 Aerodynamic diameter (d_{ae})

For particles with a geometric diameter larger than $0.5 \mu\text{m}$ the aerodynamic diameter (d_{ae}) must be used for characterization.

The aerodynamic diameter of a particle whatever its shape and density is defined as the diameter of a sphere with the density 1 (1.0 g/cm^3) which settles at the same velocity as the particle in calm or laminarly flowing air. This definition also applies for fibrous particles. The aerodynamic diameter of a fibre is characterized essentially by the diameter of the fibre and less by its length; for long fibres ($l \gg d$) the aerodynamic diameter is about three times the fibre diameter.

Laboratory instruments for the determination of the settling velocity and the aerodynamic diameter include centrifuges, sedimentation cells, impactors and aerodynamic particle sizers (APS).

A sphere with a diameter d and density ρ has a stationary settling velocity in air under gravity which is given by

$$v_s = \frac{d^2 \cdot \rho \cdot g \cdot f(d)}{18 \cdot \eta}$$

where

g is acceleration due to gravity

η is the viscosity of the air

ρ is the density of the particle substance

d is the geometric diameter of the sphere

$f(d)$ is the slip correction (see Section 1.2.2) (Fuchs 1964)

The aerodynamic diameter d_{ae} of a sphere with density ρ and geometric diameter d corresponds to the geometric diameter of a sphere with density $\rho_0 = 1 \text{ g/cm}^3$ and the same settling velocity v_s . Thus, for a sphere of any density ρ ,

$$d_{ae} = d \sqrt{\frac{\rho \cdot f(d)}{\rho_0 \cdot f(d_{ae})}}$$

For particles with geometric diameters greater than $1 \text{ }\mu\text{m}$, the quotient $f(d):f(d_{ae}) \approx 1$. Therefore

$$d_{ae} = d \sqrt{\frac{\rho}{\rho_0}}$$

1.2.2 Diffusion-equivalent diameter (d_D)

The diffusion-equivalent diameter (d_D) of an aerosol particle of any shape corresponds to the diameter of a sphere for which the Brownian motion is exactly like that of the particle in the same dispersion medium (at the workplace: air).

The diffusion-equivalent diameter (d_D) can be determined with a diffusion battery or a differential mobility analyser (DMA). For determination of the particle concentration, a Condensation Nucleus Counter (CNC) can be used. For particles with a diffusion-equivalent diameter $d_D > 0.1 \text{ }\mu\text{m}$, concentrations can be determined by measurement of scattered light with a nephelometer.

Diffusion of monodisperse particles in stationary or flowing media can be described with differential equations. The diffusion transport of particles in flowing media depends on the diffusion coefficients, the rate of flow of the medium, the time, and the number of particles. The diffusion coefficient D is a measure of the effectiveness of the Brownian motion of the particles and is given for spherical particles by the equation:

$$D = \frac{k \cdot T \cdot f(d)}{3\pi \cdot \eta \cdot d} = B \cdot k \cdot T$$

where

k is the Boltzmann constant

T is the absolute temperature

η is the viscosity of the medium

d is the geometric diameter of the particle

$f(d)$ is the slip correction

B is the mechanical mobility of the particle

The slip correction $f(d)$ compensates for deviations which arise when the diameter of the particle d is comparable with the mean free path length of motion in air. The expression $B = f(d)/(3\pi\eta d)$ is called the mechanical mobility of a particle.

Density does not play a role in motion by diffusion (see the above equation).

2 Inhalation, Deposition and Clearance of Aerosols in the Respiratory Tract

2.1 Inhalation

Data for the inhalability of airborne particles have been obtained from experiments with a model head in a wind tunnel (Armbruster *et al.* 1981, Armbruster and Breuer 1984, Ogden and Birkett 1977, Vincent and Armbruster 1981). The model heads were fitted with filters behind the mouth and nose openings and were made to “breathe” according to real breathing patterns with pumps (so-called “artificial lungs”). The experiments were carried out at wind speeds between 1 and 8 m/s, with various positions of the head relative to the wind direction and with breathing through either the mouth or the nose in various breathing patterns (e.g., breathing at rest, during physical activity, in awkward positions). A particle size range of 0.3 to 150 μm was studied.

Data from three research groups for average inhalability are shown in Figure 1 (Appendix). The values are mean values for wind speeds between 1 and 4 m/s, head position angles between 0° and 360° and three characteristic breathing patterns.

It is typical for the inhalability of particles that the mean value over the range of head positions for particles with $d_{ae} < 50 \mu\text{m}$ does not depend significantly on the wind speed, breathing pattern or on whether breathing is through the mouth or the nose. At higher wind speeds, however, the inhalability of particles with $d_{ae} > 50 \mu\text{m}$ increases with increasing wind speed (Figure 2, Appendix).

In Figure 3 (Appendix), the inhalability is shown as a function of the particle diameter and the angle of the head to the wind direction. A value of 100% for inhalability means

that all the particles in the ambient air can be inhaled into the respiratory tract. Because of their inertia, larger particles cannot follow the flow of air around the head. Thus when the wind direction is from the front (angle 0°) the inhalability can exceed 100 %.

2.2 Deposition in the respiratory tract

Particles which have entered the mouth or the nose can be carried with the inhaled air into all parts of the airways including the alveoli. At the end of each inhalation, the air in the airways stops flowing for an instant and then, apart from a residual volume, is moved in the opposite direction out of the respiratory tract during exhalation. The residual volume is comprised of the volumes contained in the relatively immobile parts of the airway system (mouth and nasal cavity, throat and tracheobronchial tree) and the volume of the alveolar region in maximal exhalation position.

During inhalation, particles are deposited in the upper regions of the airways mainly because of their inertia and because of diffusion, and in the alveoli by sedimentation and diffusion. In addition, mixing of the inhaled air with the residual volume results in particle exchange.

During exhalation, exchange of particles between the residual volume and the exhaled air also takes place so that particles from the inhaled air can remain in the lungs without being deposited immediately. On the other hand, particles inhaled in previous breaths can be exhaled later, the so-called wash-in/wash-out effect. This effect is particularly conspicuous when different concentrations are inhaled in consecutive breaths.

There are five significant mechanisms which can play a role in particle deposition in the respiratory tract. These are impaction, sedimentation, Brownian motion, interception (only with elongated particles, fibres) and electrostatic precipitation (Schlesinger 1989).

2.2.1 Total deposition (DE)

The dependence of total deposition (DE) of particles in the human respiratory tract on various respiratory parameters has been determined experimentally in the particle size range from $0.005 \mu\text{m}$ (d_p) to $20 \mu\text{m}$ (d_{ac}) (Anderson *et al.* 1988, Bennett and Smaldone 1987, Bennett *et al.* 1996, Blanchard and Willeke 1983, Brain and Valberg 1979, Davies 1982, Diu and Yu 1983, Heyder *et al.* 1973, 1986, Landahl 1950, Schiller-Scotland *et al.* 1988). In the particle size range around $0.5 \mu\text{m}$ (d_{ac}), the total deposition has a minimum of 0.1–0.2 and then rises with both decreasing and increasing particle diameters. The increase in total deposition at smaller particle diameters is a result of the increasing Brownian motion with decreasing particle size (Blanchard and Willeke 1984, Cheng *et al.* 1996). Deposition increases linearly with the retention time in the lungs (t) and the diffusion coefficient (D) (Anderson *et al.* 1990, Schiller-Scotland *et al.* 1986, Tu and Knutson 1984). At a particle diameter of 5 nm (d_p), total deposition values of $DE > 0.9$ are reached. Recent data from Swift and Strong (1996) indicate that for particles with diameter $d_p = 0.5 \text{ nm}$, total deposition $DE = 1$.

With increasing particle size, two other mechanisms for the deposition of particles in the lungs come into effect, sedimentation and impaction (Gebhart *et al.* 1984, Gentry *et al.* 1994, Scheuch and Stahlhofen 1992). Deposition by sedimentation is caused by the gravitational field of the earth and increases with increasing retention time in the lungs and with increasing particle size (d_{ac}). Another mechanism, impaction, also becomes more important for larger particle sizes; when the airstream changes direction abruptly, the larger particles cannot follow the air flow because of their inertia and are deposited on the walls of the airways. Deposition by this mechanism increases linearly with particle size and respiratory flow (Q) (Heyder *et al.* 1980, Köbrich *et al.* 1994). In this way, for particles with diameters greater or equal to $10 \mu\text{m}$ (d_{ac}), total deposition values $DE > 0.9$ are attained (Figure 4, Appendix). For particles with $d_{ac} > 0.5$, deposition is also affected by whether breathing is through the mouth or the nose. During breathing through the nose deposition is higher because of impaction and, depending on the respiration rate, can attain values of $DE > 0.9$ for particles with diameters $d_{ac} \geq 5 \mu\text{m}$ (Figures 4 and 5) (Egan and Nixon 1989, Heyder and Rudolf 1977, Hounam *et al.* 1971).

2.2.2 Regional deposition and clearance

In studies of regional deposition of insoluble particles in the airways of volunteers (see Figure 4, Appendix), it is possible to differentiate between

- extrathoracic deposition (oral-nasal-pharyngeal cavity and larynx) and
- thoracic deposition, which can be subdivided into
- tracheobronchial deposition and
- alveolar deposition.

The particles deposited in the different regions belong to typical size classes (Egan and Nixon 1988, Emmet *et al.* 1982, Foord *et al.* 1978, Lippmann *et al.* 1971, Rudolf *et al.* 1986, 1988, 1994).

In the extrathoracic region, most of the particles deposited belong to the coarse particle fraction. When the particles are inhaled through the mouth, deposition begins at about $2 \mu\text{m}$ (d_{ac}) and increases until, at $d_{ac} \geq 10 \mu\text{m}$, deposition values of over 0.9 are reached. When the particles are inhaled through the nose, deposition begins at d_{ac} values as low as $0.5 \mu\text{m}$ and reaches values of $DE \geq 0.9$ at particle diameters as low as $d_{ac} = 7\text{--}8 \mu\text{m}$ (Heyder and Rudolf 1977) (Figure 5, Appendix).

Tracheobronchial and alveolar deposition are differentiated by making use of the different clearance rates of insoluble particles from these regions (Chan and Lippmann 1980, Langenback *et al.* 1990, Lippmann 1986, Stahlhofen *et al.* 1979, 1980). Thus most of the particles deposited in the ciliated regions of the respiratory tract are transported out of the lungs with an elimination half-time of less than 6 hours. For non-ciliated regions of the lung (“alveolar region”) the elimination half-time is considerably longer, 3 months and more (Camner and Philipson 1978, Stahlhofen *et al.* 1981b). These data apply only for insoluble particles and particles without substance-specific toxicity.

In the ciliated regions of the respiratory tract (tracheobronchial region), particles are deposited on the bronchial mucous secretion and transported with the latter towards the

larynx. They enter the oropharynx and are then swallowed with the mucous into the gastrointestinal tract. Recent studies have shown that not all the particles deposited in the tracheobronchial region are transported out of the lungs within one day. The particles which are not transported out of the lungs within one day via the mucociliary transport mechanisms are eliminated with half-times between 5 and 30 days. For particles with a geometric diameter of about $2\ \mu\text{m}$, the proportion which is eliminated slowly from the tracheobronchial region is about 50%. Particles with a geometric diameter above $7\ \mu\text{m}$ are almost entirely rapidly eliminated (within 24 hours) and the proportion of particles which is eliminated slowly lies under 10% (ICRP 1994, Scheuch *et al.* 1996, Stahlhofen 1988, Svartengren *et al.* 1996).

In the bronchial tree, a collective of particles in the diameter range between 2 and $20\ \mu\text{m}$ (d_{ae}) with a maximum between 5 and $10\ \mu\text{m}$ (d_{ae}) is deposited (Figure 4) (Phalen *et al.* 1988). After inhalation through the mouth the tracheobronchial deposition is greater and displaced slightly towards larger particles than after inhalation through the nose.

In the non-ciliated region (alveolar region), the particles are ingested by macrophages and can, but with low probability, be transported by the macrophages into the ciliated region. Another fraction of the particles can penetrate the interstitial tissue or enter the local lymph nodes via the lymphatic vessels (ICRP 1994). In the form of permanent depots, they are exposed to the effects of the cellular and humoral defence mechanisms of the organism. The half-time of the clearance of the particles from the alveolar region is dependent on the observation time. It has been demonstrated that the clearance of insoluble particles from the alveolar region does not obey a simple exponential function and thus cannot be described by one half-time (Figure 6, Appendix). The longer the observation period, the flatter is the clearance curve and thus the greater is the “half-time” determined. Whereas at the beginning of the observation period, elimination half-times of about 100 days were found, after an observation period of 5 years the elimination half-times had increased up to 2000 days (ICRP 1994, Philipson *et al.* 1995). The alveolar deposition curve has two maxima (Figure 4, Appendix), one in a diameter range of $2\text{--}4\ \mu\text{m}$ (d_{ae}), the other in the submicron range at about $0.03\ \mu\text{m}$ (d_{D}) (Heyder 1982). The ultrafine particles are deposited by diffusion, particles with diameters greater than $1\ \mu\text{m}$ (d_{ae}) mostly by sedimentation. In the range of ultrafine particles $d_{\text{D}} < 0.03\ \mu\text{m}$, alveolar deposition decreases again. The increase in diffusion at small particle sizes results in an increased deposition in the extrathoracic and tracheobronchial regions so that only a smaller fraction of the particles can reach the alveolar region (Cohen and Asgharian 1990, Cohen *et al.* 1990, Heyder *et al.* 1983, Swift *et al.* 1994, Yu and Cohen 1994).

The distribution of an inhaled aerosol among the above-mentioned regions of the respiratory tract is affected not only by the properties of the particles but also by:

1. individual differences in the anatomy of the respiratory tract,
2. individual differences in breathing habits, especially differences in the changing from breathing through the nose to breathing through the mouth during physical activity,

3. differences in respiration rate, respiratory flow and therefore respiratory volumes for various breathing patterns (e.g. breathing at rest, breathing while working, in awkward positions)
4. pathophysiological changes in mucociliary clearance (e.g. caused by tobacco smoke, catarrhal infections)

(Bennett *et al.* 1985, Heyder *et al.* 1982, 1988, Kim 1989, Kim and Eldridge 1985, Kim *et al.* 1988, Schiller-Scotland *et al.* 1994, Stahlhofen *et al.* 1981a, 1989).

2.3 Behaviour of deposited particles

On deposition, the particles come into contact with the liquids which cover the surfaces of the cells in the various regions of the respiratory tract (surfactant, serous and mucous secretions, etc.). Liquid particles are rapidly absorbed, as are readily soluble solids. The solubilization process depends on the particle diameter.

The half-time for the solubilization is given by (Mercer 1967):

$$t_2 - t_1 = 0.206 \cdot \left(\frac{3 \cdot \alpha_v \cdot \rho \cdot d_0}{\alpha_s \cdot k} - t_1 \right)$$

where

t_1 is the time at the beginning of the observation period,

t_2 is the time at which half of the material is dissolved,

$\alpha_v = \frac{m}{\rho \cdot d_0^3}$ volume shape factor,

$\alpha_s = \frac{s}{d_0^2}$ surface shape factor (s is surface area),

ρ is the density of the particles,

d_0 is the initial diameter of the particles (geometric),

k is the solubility constant of the substance,

m is the mass of the observed particle.

The smaller the particles the more rapid is the dissolution process. This can be determined experimentally in a reagent with properties like those of body fluids (Mercer 1967). Insoluble residues of particles and insoluble particles are subject to the mechanical clearance mechanisms (see Section 2.2.2).

2.4 Behaviour of hygroscopic particles

Hygroscopic particles have the capacity to absorb water vapour from gases and to increase rapidly in mass and diameter in the process (Ferron *et al.* 1988, 1992, Hänel and Heyder 1980). This takes place especially in the lungs after inhalation of hygroscopic particles because the relative humidity in the airways is almost 100% (Hicks and Megaw 1985, Hicks *et al.* 1986). The deposition curve plotted for the particle size before inhalation is thus displaced as a whole towards smaller diameters (Figure 7, Appendix) because

the particles inhaled in the dry state increase in size by absorption of water vapour and then display the deposition behaviour of larger particles (Anselm *et al.* 1986, Blanchard and Willeke 1984, Gebhart *et al.* 1987, 1990, Morrow 1986). The curves for regional deposition are also displaced (ICRP 1994).

3 Diseases

Particulate substances encountered at the workplace can cause a variety of diseases of the respiratory tract which are of particular significance because of their number, severity, and the associated socio-economic effects.

The number of new cases of compensatable occupational diseases caused by dust each year is almost 3000, the number of deaths from the same almost 400. The number of cases of occupational diseases caused by dust notified each year is sometimes greater by a factor of 10 or more than the number which result in retirement with a pension [see the annual safety reports of the Federal German Ministry for Employment and Social Affairs “Arbeitssicherheitsberichte des Bundesministeriums für Arbeit und Sozialordnung”].

The main effects included in the occupational medical assessment are the carcinogenic, fibrosis-inducing (fibrogenic) effects and also irritative or systemic toxic effects, allergenic effects and overloading. The effects depend largely on the site of deposition of the inhaled particles in the respiratory tract. The deposition of the particles and the intensity of the effects and rapidity of their onset are determined essentially by the size, shape, surface area, density, chemical and mineralogical composition, hygroscopic properties and crystalline structure of the particles (see Section 2).

The following is a list of examples of such diseases.

Diseases of the nose, pharynx and larynx include:

- malignant tumours caused by dust or droplets of chromium(VI) or nickel compounds or by oak or beech wood dust (see Section III; *List of MAK and BAT Values 1997*),
- obstructive diseases of the upper airways caused by allergenic dusts (allergic rhinopathy),
- perforation of the anterior nasal septum by dust or droplets of chromium(VI) compounds.

Diseases in the region of the tracheobronchial tree include:

- bronchial cancer caused by asbestos fibre dust and dust of arsenic, chromium(VI) or nickel compounds (see Section III; *List of MAK and BAT Values 1997*),
- obstructive disorders of the lower airways caused by chemical irritants or chemical toxins in the form of aerosols.
- cotton dust byssinosis (brown lung).

Diseases of the pulmonary parenchyma include:

- pulmonary fibrosis caused by inorganic fibrogenic dusts, e.g. silicosis, asbestosis, talcosis, hard metal lung, etc.,
- exogenous allergic alveolitis caused by allergenic dusts, e.g. farmer's lung.

Deposition of particular dust particles in the alveolar region or in the interstitial tissue of the lungs causes diseases such as siderosis (caused by iron oxide), baritosis (by barium sulfate), soot lung or tin oxide lung which are designated as storage diseases. Such storage diseases which do not result in fibrosis do not cause functional deficits and therefore are generally not considered to have disease status. Fibrous dusts, e.g. from asbestos, can also cause fibrogenic and malignant changes in the pleura (pleural fibrosis, plaques and mesothelioma).

Deposited particles which are transferred from the respiratory tract to the digestive tract by means of the respiratory clearance mechanisms can be absorbed in the digestive tract and produce toxic effects.

4 Particle Sampling Procedures

4.1 Conventions for measuring concentrations of particles

For the determination of dust fractions with measuring and sampling equipment, five international conventions have been established on the basis of three deposition curves (DIN EN 481). They are based on average experimental data, obtained under defined experimental conditions, for the inhalability and regional deposition of particles (Figures 8, 9, 10 and Table 1, Appendix).

1. **Inhalable fraction (I):** the curve shows the average probability that particles will be inhaled.
2. **Thoracic fraction:** the curve represents the average probability that particles will enter the tracheobronchial and alveolar regions of the lung.
3. **Respirable fraction (R):** this fraction is a part of the thoracic fraction; the curve represents the average probability that particles will enter the alveolar region.
4. **Extrathoracic fraction:** this fraction is obtained by subtracting the thoracic fraction from the inhalable fraction. (Data for this fraction are not tabulated in the convention.)
5. **Tracheobronchial fraction:** this fraction is obtained by subtracting the respirable fraction from the thoracic fraction. (Data for this fraction are not tabulated in the convention.)

For the determination of fibrous dust concentrations, fractions are not defined by aerodynamic criteria. Instead fibre lengths and diameters must be determined micro-

scopically (see *List of MAK and BAT Values 1997*, Section III, Carcinogenic substances, Fibrous dusts).

The definitions of the “inhalable fraction” and “respirable fraction” (HSE 1995, BIA-Arbeitsmappe) are largely equivalent to the definitions of “total dust” (G) and “fine dust” (F) (*List of MAK and BAT Values 1995*) used before 1996 in the establishment of MAK values. Since 1996 the internationally agreed definitions apply. Instead of “total dust”, the term “inhalable fraction” is used (abbreviation “I”). “Fine dust” is now designated as the “respirable fraction” (abbreviation “R”) (Table 2).

These new definitions apply not only to dusts but to all aerosols.

Table 2. Changes in the designation of dusts

Date	Dust fraction	Designation
until 1996	total dust	G
from 1996	inhalable dust	I
until 1996	fine dust	F
from 1996	respirable dust	R

4.2 Additional information about sampling procedures

The adoption of DIN EN 481 does not make it necessary at present to change the sampling devices used for dust collection in Germany (BIA-Arbeitsmappe) although the designation of the total dust fraction has been changed to “inhalable fraction” and the definition altered and extended. In some other countries the devices which were used previously to determine “total aerosol exposure” yielded values for the aerosols at industrially important workplaces which were too low by factors of up to 0.5 when compared with the values obtained with devices for determining the inhalable fraction of the aerosol (Werner *et al.* 1996).

The effects of the new standards on sampling procedures can, however, not yet be finally and quantitatively assessed. Until that is possible, the previously used devices for sampling and determination of “total dust” and “fine dust” are to be used as before.

1. **Devices for determination of the respirable fraction** (previously: “**fine dust sampler**”), which have a fractionation system for which the theoretical separation function matches that defined for sedimentation separators in the Johannesburg convention (50 % of particles with an aerodynamic diameter of 5 µm are retained) can continue to be used. Their deposition curves are within the range of permitted deviations from the deposition curve for the respirable fraction defined in the convention (DIN EN 481). The range is from 0 % to 30 %.
2. **Devices for determination of the inhalable fraction** (previously: “**total dust sampler**”) were required until 1996 to operate at a suction velocity of 1.25 m/s ± 10 %. In the future the deposition function must be determined for these devices as well and compared with that defined for the inhalable fraction in the convention DIN EN 481.

Initial results suggest that the total dust samplers used at present can fulfil the requirements of DIN EN 481 at low wind speeds (3 m/s) (Armbruster and Breuer 1984). Total dust samplers which meet the criteria valid before 1996 can, therefore, be used in future too.

3. For **fumes** the inhalable fraction (I) must be sampled when the fumes contain substances whose threshold values are expressed in terms of the inhalable fraction, previously total dust (G). If the fumes contain only substances whose threshold values are expressed in terms of the respirable fraction (R), previously fine dust (F), then they are sampled as for the respirable fraction.
4. For **mists** the inhalable fraction (I) is to be sampled.
5. If sampling devices are used which collect fractions according to deposition curves which differ from those described above and which do not fulfil the requirements given in 1. and 2., the results must be corrected using a conversion factor which is dependent on the particle size distribution.

In addition, it may be seen in the results of measurements of aerosol concentrations at workplaces that the results differ depending on whether area or personal sampling systems are used. The differences found in the determination of the inhalable and respirable fractions are described in the documentation of the “General Threshold Limit Value for Dust” (this volume) on the basis of the results given in the BIA documentation MEGA.

5 Exposures exceeding the MAK Value

The MAK values for aerosols which are designated “Vg” in the *List of MAK and BAT Values* have been derived from average long-term exposure levels without detectable adverse effects on health.

Impairment of respiratory organ function is a result of long-term effects which are determined largely by the aerosol concentration to which the person is exposed over long periods of time. The MAK values for aerosols, in line with the definition of a MAK value, apply to the concentration values averaged over a single shift. As the average long-term exposure level is an average of variously high shift average levels, the occasional exceeding of the MAK value by single shift average levels can be tolerated. The permitted frequency and extent of the excursions above the MAK value is established on the basis of occupational medical and toxicological findings (see the documentation “Derivation of MAK values for dusts from long-term threshold values” in Volume 11 of the present series). In these cases the peak limitation categories do not apply.

For all other aerosols the peak limitation categories must be observed (see *List of MAK and BAT Values*, Section I Significance and use of MAK values, Limitation of exposure peaks).

6 References

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Appendix

Table 1. Data for the curves defined in DIN EN 481 for the division of the total airborne aerosol into fractions of occupational medical relevance

Aerodynamic diameter [μm]	Inhalable fraction [%]	Thoracic fraction [%]	Respirable fraction [%]
0	100.0	100.0	100.0
1	97.1	97.1	97.1
2	94.3	94.3	91.4
3	91.7	91.7	73.9
4	89.3	89.0	50.0
5	87.0	85.4	30.0
6	84.9	80.5	16.8
7	82.9	74.2	9.0
8	80.9	66.6	4.8
9	79.1	58.3	2.5
10	77.4	50.0	1.3
11	75.8	42.1	0.7
12	74.3	34.9	0.4
13	72.9	28.6	0.2
14	71.6	23.2	0.2
15	70.3	18.7	0.1
16	69.1	15.0	0.0
18	67.0	9.5	
20	65.1	5.9	
25	61.2	1.8	
30	58.3	0.6	
35	56.1	0.2	
40	54.5	0.1	
50	52.5	0.0	
60	51.4		
80	50.4		
100	50.1		

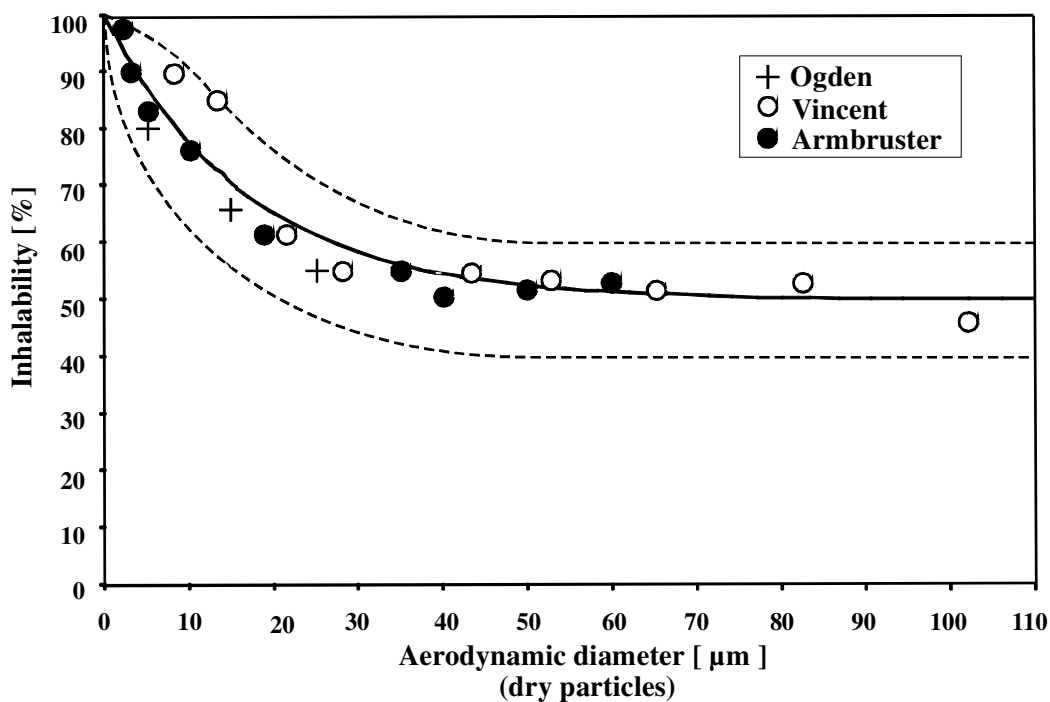


Figure 1. Average probability that a particle will be inhaled (inhalability) as a function of the aerodynamic diameter of the particle. Data from Ogden and Birkett (1977), Vincent *et al.* (1990), and Armbruster and Breuer (1982)

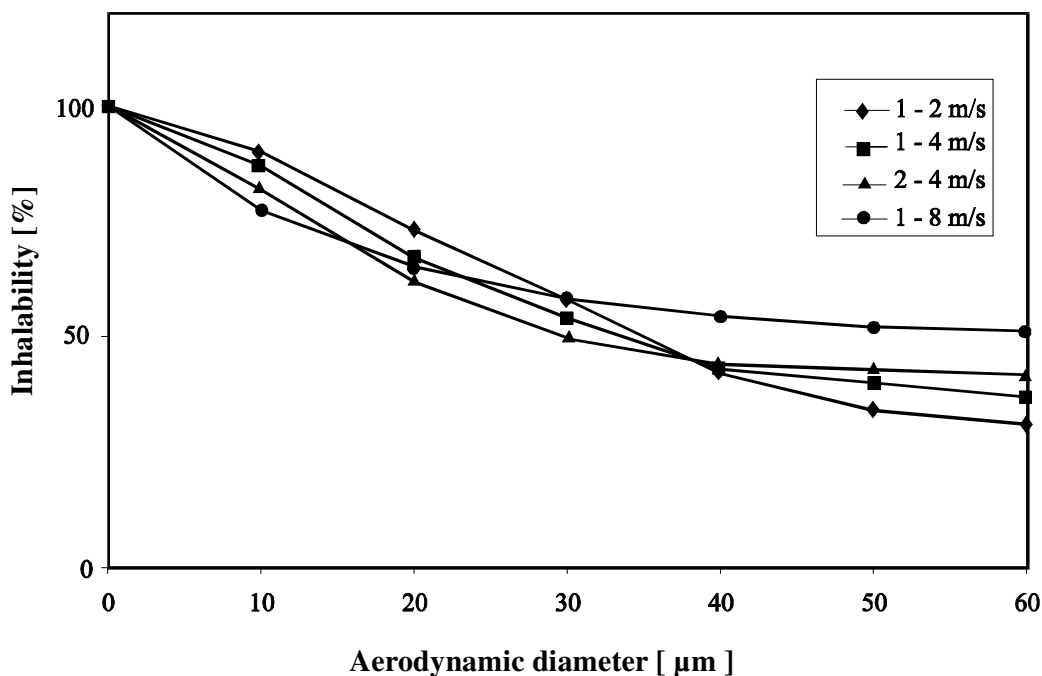


Figure 2. The probability that a particle will be inhaled (inhalability) when breathing through the mouth during normal work as a function of the aerodynamic diameter. The probability values are averaged for various position of the model head in the wind tunnel and averaged over wind speeds between 1–2 m/s and 1–8 m/s.

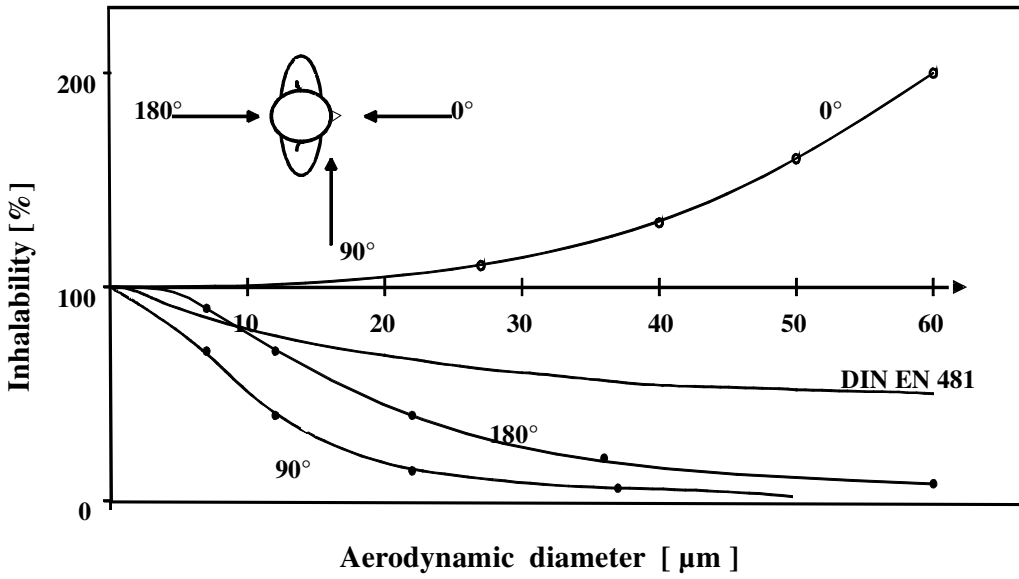
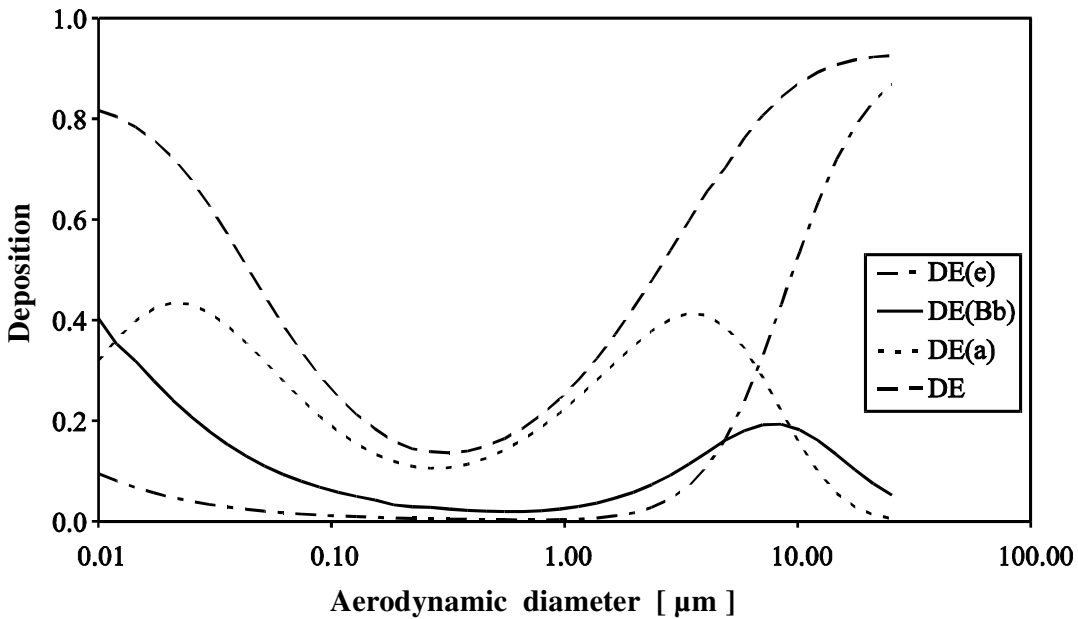


Figure 3. Inhalability of particles as a function of the aerodynamic diameter at a wind speed of 4 m/s for various angles of the head to the wind direction and, for comparison, the relationship of inhalability to particle size as defined by DIN EN 481



DE: total deposition
 DE(Bb): tracheobronchial deposition
 DE(a): alveolar deposition
 DE(e): extrathoracic deposition

Figure 4. Deposition of particles in the respiratory tract as a function of particle diameter, for a respiration rate $Q = 300$ ml/s, a tidal volume of 700 ml and a test person with healthy lungs and functional reserve capacity (FRC) of 3600 ml when breathing through the mouth.

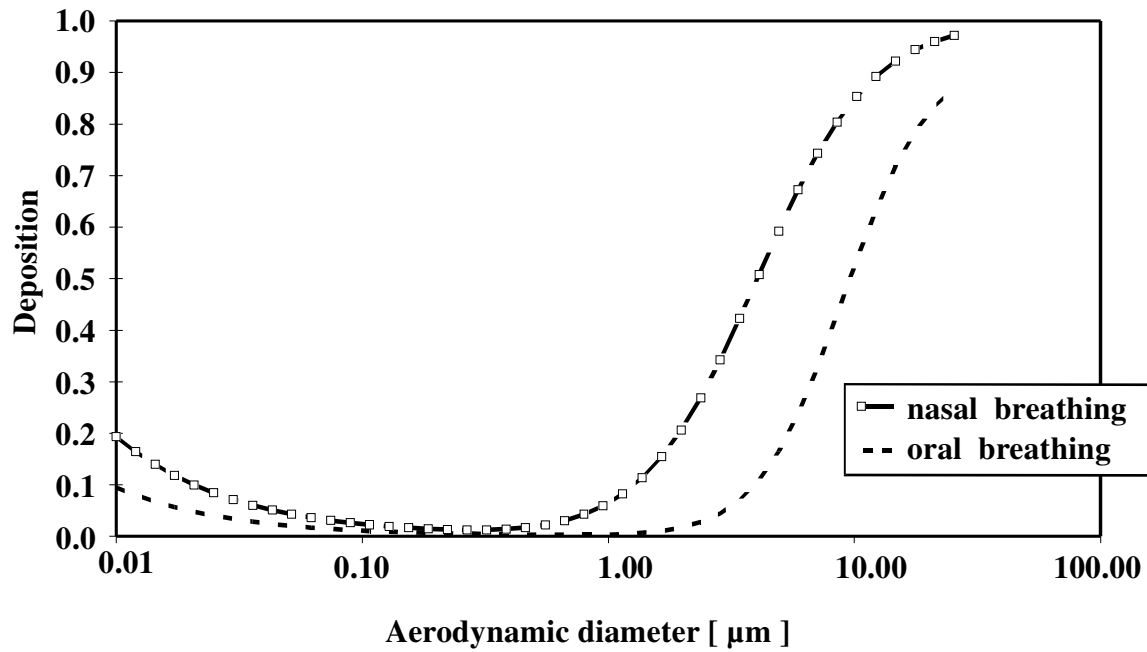


Figure 5. Extrathoracic deposition as a function of the particle diameter after breathing through the mouth and through the nose (for parameters see Figure 4)

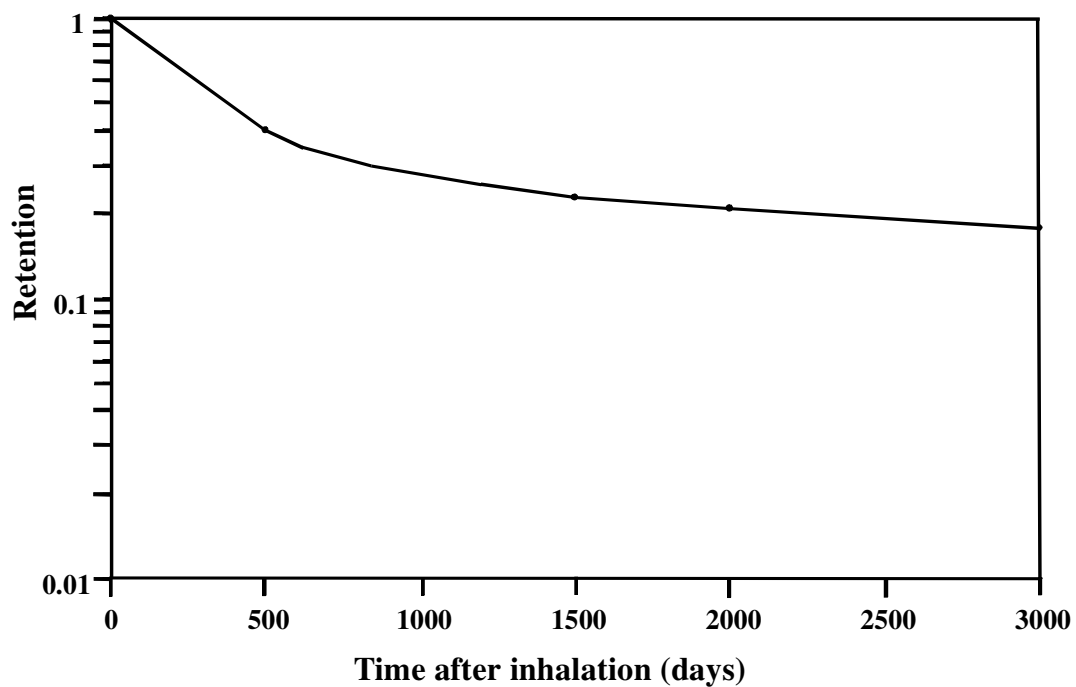


Figure 6. Particle retention in the alveolar region of the lungs as a function of the time after inhalation

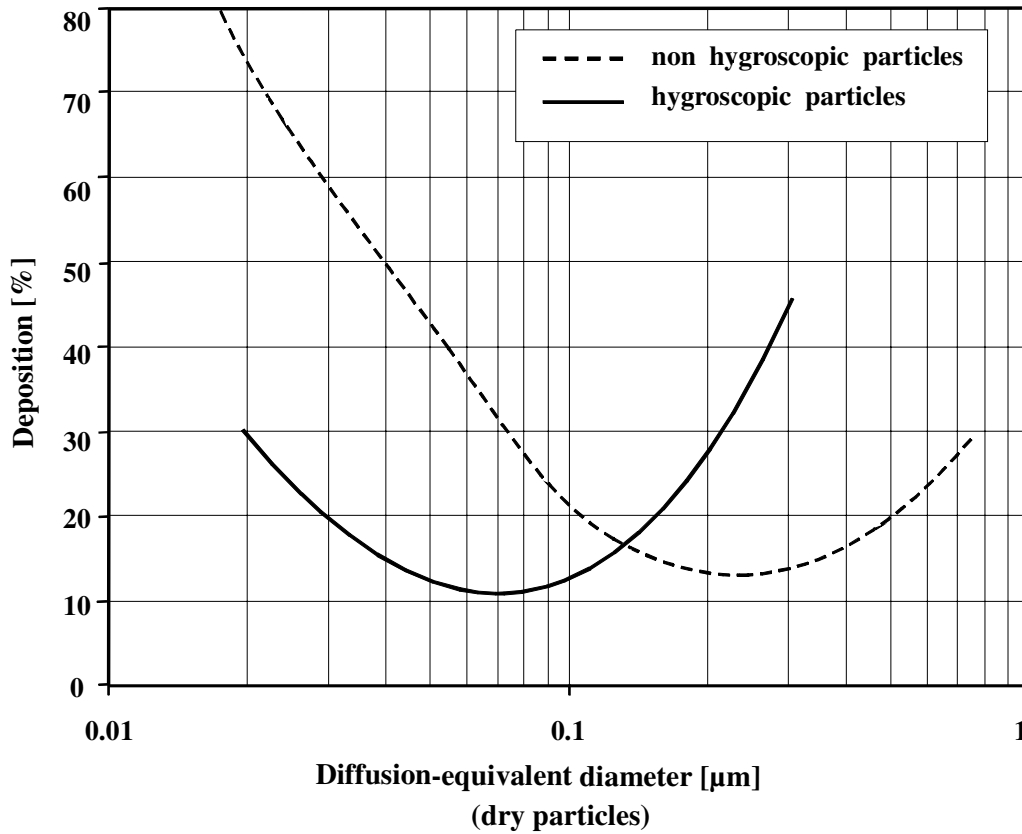


Figure 7. Total deposition of particles in the respiratory tract as a function of the diffusion-equivalent diameter of the dry particles

Definitions

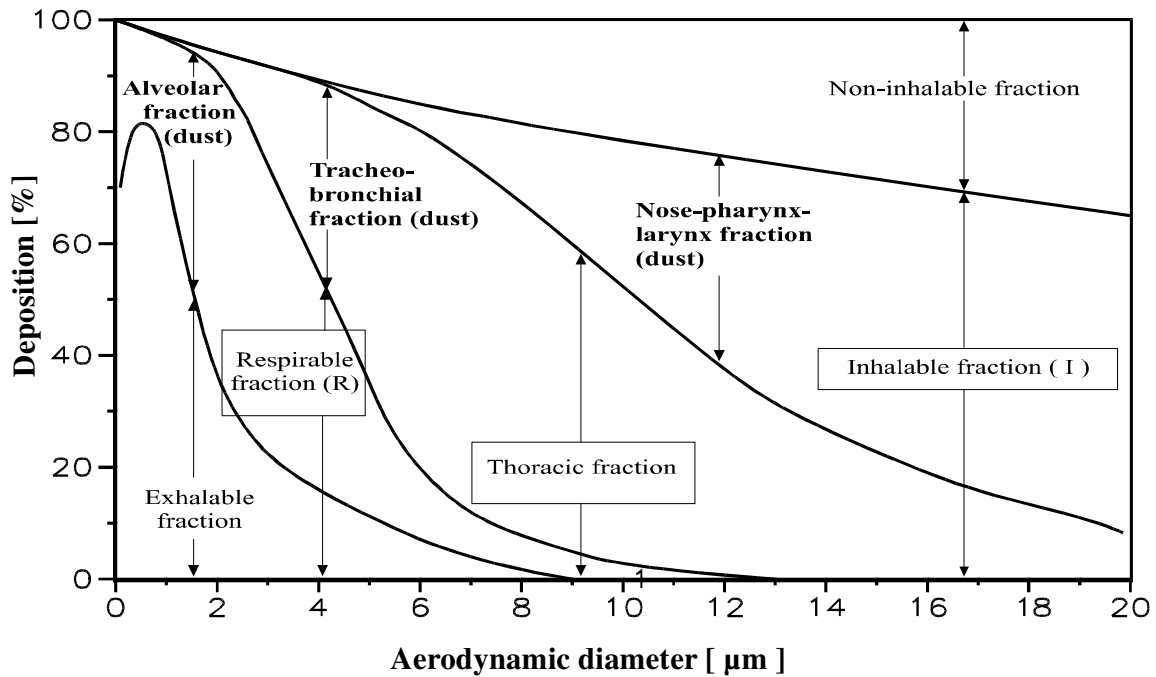


Figure 8. Definition of aerosol fractions as a function of aerodynamic diameter. The aerosol fractions which are important in occupational medicine have been printed in bold type. The boxed fractions are those which can be measured and include in every case the fraction which can be exhaled (see Figure 10).

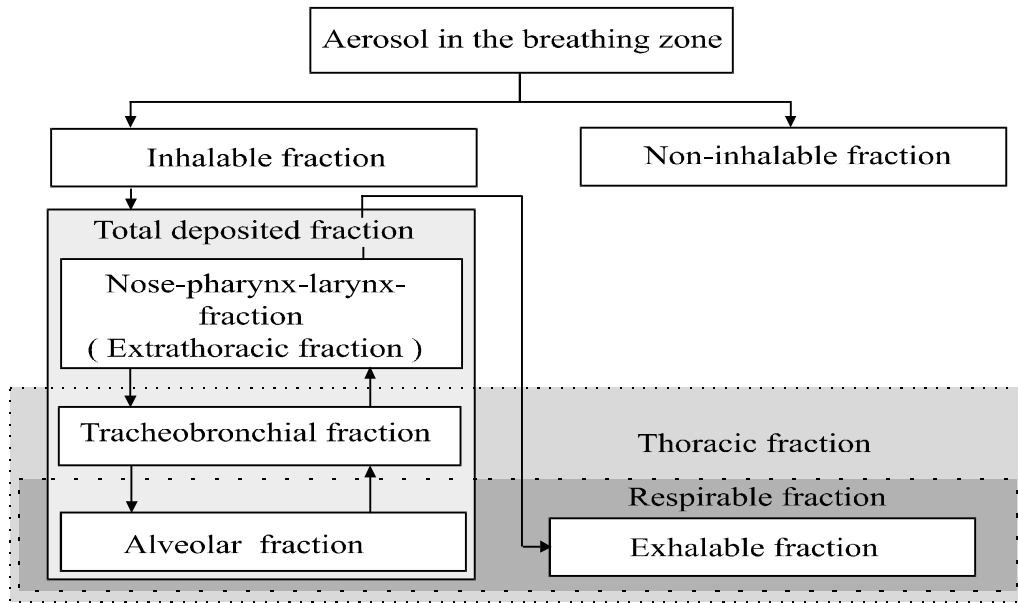


Figure 9. Subdivision of the total aerosol in the breathing zone according to occupational medical criteria. The fraction which enters the thorax includes the (intra-)thoracic fraction and the fraction which can be inhaled. The fraction deposited in the thorax includes the tracheobronchial and alveolar fractions.

Conventions for deposition curves

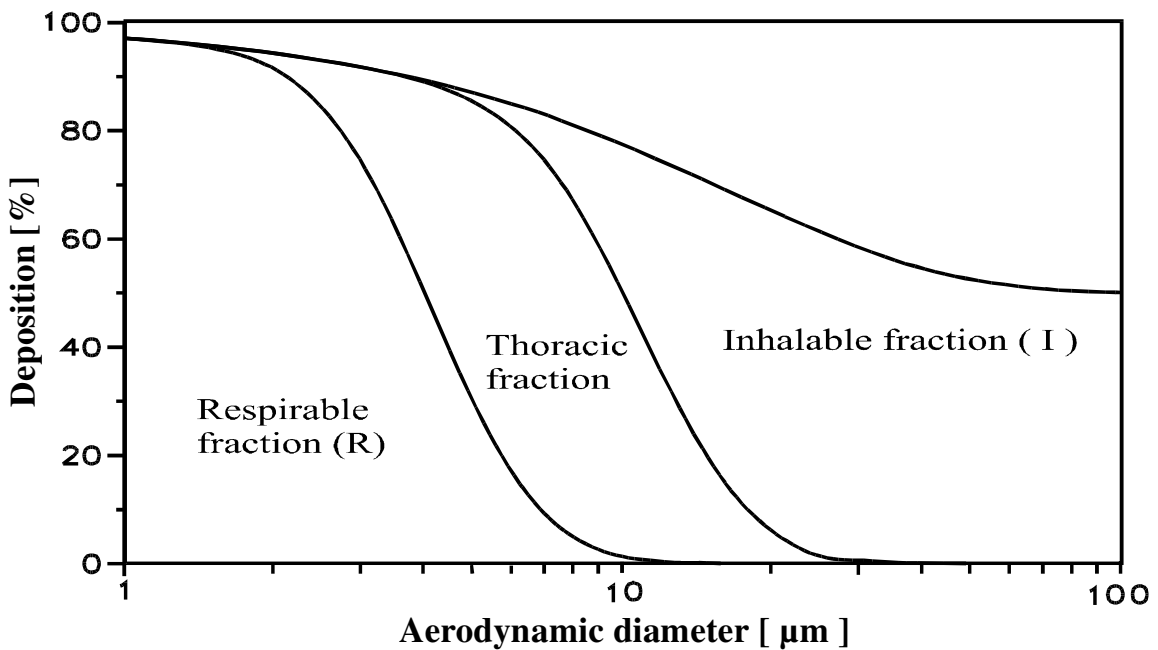


Figure 10. Curves for the separation of the airborne particles in the breathing zone into inhalable, thoracic and respirable fractions. Because the X-axis is on a logarithmic scale, the aerodynamic diameter with the value “zero” cannot be included. The deposition ratio corresponding to this value would be 100 %.