15 Powder hazards

Some of the hazards posed by powders should be obvious and several aspects have been covered in earlier chapters; e.g. powder floods in Chapter 10. From an operating point of view, the strongest motivation for the prevention of a powder hazard is the health and safety of the process operators and those in the surrounding environment. Health and safety legislation varies from country to country, and changes with new legislation and directives, so the following references to standards and procedures should be viewed only as an introduction to this topic. The main hazards posed by powders have been split into two: explosion within a process and personal health hazards.

15.1 Explosion hazards

Eighty percent of organic dusts have been found to be explosive, as are many very fine metal powders, e.g. magnesium and aluminium. Dust explosions are commonplace and it has been estimated that on average there is one accidental explosion per week within the UK. The main distinction between a vapour explosion (*detonation*) and dust is the lower flame velocity found in the latter case. Dust explosions are usually *deflagrations*; i.e. the flame speed is less than sonic velocity and usually ranges from a few m s^{-1} to low 100's m s^{-1}, sonic velocity is approximately 330 m s^{-1}. For explosion five conditions must be met:

- dust must be suspended in air or gas supporting combustion,
- must have a particle size capable of propagating a flame,
- dust concentration must be in the explosive range (the lower threshold ranging from 20 to 50 g m^{-3} for most dusts),
- must be above minimum ignition temperature – but this may be achieved in various ways, so use this only to compare dusts, and
- there must be an ignition source of sufficient energy (which may locally provide the heat for the last point).

The lower flame speed of a deflagration means that there is a better chance of pressure venting to control an explosion with powders, compared to vapour phase detonations.

Particle size is important and, in general, smaller particles are more likely to be explosive. They are also more likely to become airborne. In many cases a dust explosion is followed by a secondary explosion that can have even greater force than the first. This is due to finer particles, which previously rested on ledges and floors, becoming airborne in the first deflagration and creating the secondary explosion. Hence, *good housekeeping* by minimising particles on ledges and floors is important. In the UK powders have been classified into two: Group A and B. Group A is deemed to be the most dangerous and there are standard tests to determine in which group a powder belongs.

However, even the safer Group B powders may be combustible and present a fire hazard.

The rate of pressure rise due to a powder explosion can be estimated by the cube root law, which relates the rate of increase in the pressure to a vessel volume (V_v) and a material dependent constant (K_{ST} in bar m s^{-1})

$$\left(\frac{dP}{dt}\right)_{max} V_v^{1/3} = K_{ST} \qquad (15.1)$$

Nomographs relating the vessel volume, value of K_{ST} and required vent area, based on the above equation, are available – see *The Chemical Engineer*, January, 1989, pp18 – 21 and *'Guide to dust explosion prevention and protection'* – Part 1, IChemE, Rugby, 1984.

15.2 Physiological Powder Hazards

High concentrations of dust in air can cause erosion of tooth enamel, abrasion of skin, etc. Powders that can be absorbed into the body and give rise to chemical, or biochemical, reaction are potentially very dangerous. However, the most significant powder hazards are due to:

- skin – dermatological disease such as dermatitis, which is possible to prevent with the use of barrier creams and protective clothing,
- eyes – cause soreness but not usually permanent damage unless powder dissolves and chemically attacks the eye, and
- lungs – the respiratory system.

Most concern has focussed on the influence of particles on the respiratory system as this can cause both acute (quick acting) and chronic (over a long time period) health effects. Table 15.1 provides a guide to the amount of air required by a person under different conditions at 20°C in litres per minute.

Table15.1 Typical air respiratory requirements for a human

Activity	Air requirement in litres/minute
sleeping	6
sitting	7
standing	8
walking at 2 mph	14 to 26
jogging	43
maximum exertion	65–100

A typical manual labourer will use 1 to 3 m^3 hr^{-1}. Hence, the concentration of an airborne dust in mg m^{-3} can be used to estimate the intake of dust to the lungs. Not all the airborne dust will make it into the mass transfer region of the lungs (the alveoli), only particles in the *respirable range*. This particle size range is illustrated in Figure 15.1, where the diameter is the *aerodynamic diameter*; i.e. the particle size that has the same settling velocity in still air as a particle of relative density of one (i.e. 1000 kg m^{-3}). The remaining dust is removed by the body's natural defences against foreign material.

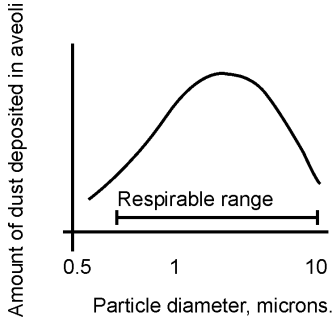


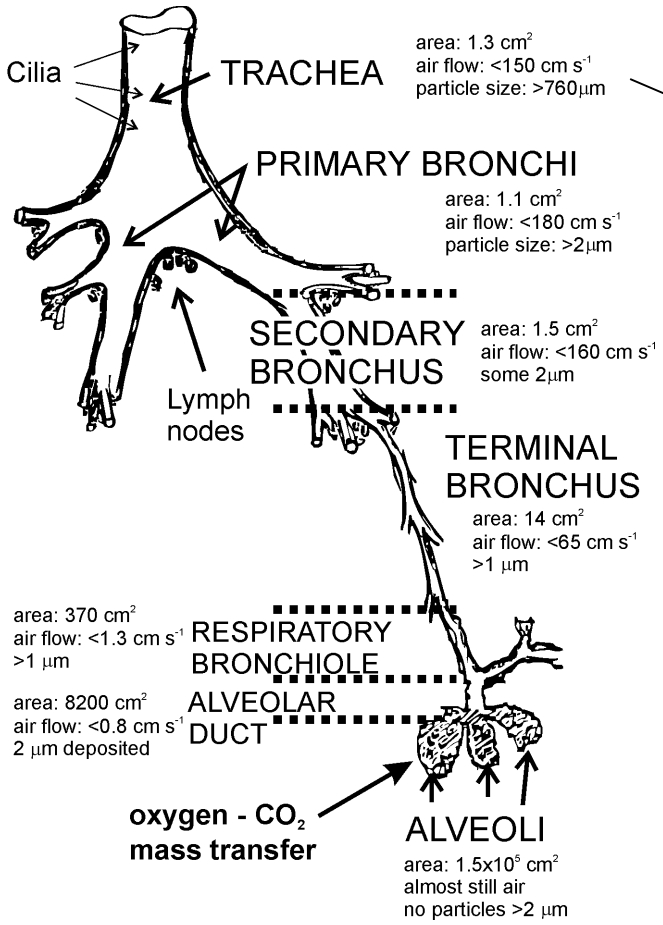
Fig. 15.1 The respirable range

Particles above this range are removed by: impingement on nasal hairs, back of the throat and splits in the respiratory tract, see Chapter 14 for a more comprehensive description of the appropriate mechanisms. Particles in the respirable range may be deposited in the alveoli by sedimentation in the very slowly moving air, or diffusion onto the surfaces. Particles below the respirable range may be exhaled after entering and leaving the lungs. Figure 15.2 provides a large-scale schematic representation of the respiratory tract with data on air flow and particle size of particles that may be found deposited in the

regions. The body has several mechanisms for dealing with particles including:

The *cilia*, which are very thin hairs up to 4 μm long that line bronchi and trachea and catch foreign bodies in the respiratory system. Trapped particles are covered in mucus and passed up into the throat where they are swallowed, sneezed or spat out; N.B. cilia are destroyed at an early stage of smoking induced lung cancer, causing mucous to accumulate in the air passageways and lungs resulting in *smokers cough*.

Phagocytes are cells that surround the particle and reduce its irritation. They can take the particle to the *bronchioles*, and out by the cilia, as above, or into the blood stream and eventually excreted, or into the lymphatic system, and possibly back into blood.



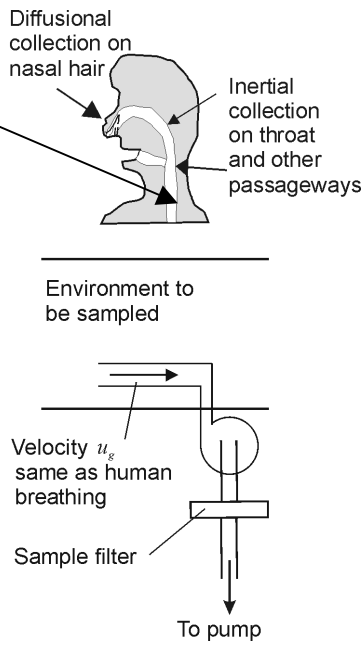


Fig. 15.3 Human head and dust sampling strategy

Fig. 15.2 Schematic diagram of the human respiratory tract

In the UK, the *threshold limit value* (TLV) applies to the dust in the respirable range and were published by the Health and Safety Executive (HSE), but the Control of Substances Hazardous to Health, COSHH, act 1988, introduced the *Maximum Exposure Limit* (MEL) and

the *Occupational Exposure Standard* (OES). For monitoring and protection both standards include such phraseology as: *is reasonably practicable*, but the former is essentially the TLV and the latter is applied to the less well-known materials. See the HSE Guidance Notes EH/40 series, 1989. The old TLV values, defined by different groups of powders, will be discussed below, as they provide a framework on which to base decisions on how hazardous particles may be.

Group I – very dangerous, 0 to 50 μg m^{-3}, as they readily give rise to fibrosis and include: beryllium, silica in the cristobalite form and blue asbestos (5 fibres/cc less than 5 μm in length). Note that asbestos is not classified using aerodynamic diameters; this is because of its unusual fibre like shape.

Group II – dangerous, 50 to 250 μg m^{-3}, including: asbestos (other forms of and with 5 fibres/cc > 5 μm in length), silica such as quartz, and mixed dusts containing 20% or more of silica.

Group III – moderate risk 250 - 1000 μg m^{-3}, including: mixed dusts of less than 20% silica, talc, mica, kaolin, cotton, organic dust, graphite and coal.

Group IV - >1 mg m^{-3}, the least problematical dusts including: cement, limestone, glass, barytes, perlite, iron oxide, magnesia and zinc oxide.

The general term for a dust induced lung disease is *Pneumoconiosis*. Well-known forms of Pneumoconiosis include: asbestosis and silicosis. The prevention of a respiratory hazard includes: keeping dust levels low, switching the process to a powder of lower danger where possible, using dust extraction hoods and *personal protection equipment* such as masks and gloves. It is critically important to monitor the working air quality frequently by taking samples according to approved procedures. This normally means sampling the airflow within ducts *isokinetically*. When sampling is required in a normally still environment, proprietary devices designed to only pass material within the respirable range on to a test filter are available, using an air pump to ensure that the gas velocity within the sample tube is similar to what would be experienced by a human. This is illustrated in Figure 15.3. The resulting concentration can be checked against the appropriate standard.

15.3 Summary

The head and throat regions have very effective means for trapping dust: nasal hair and splits in the respiratory tract provide targets for diffusional and inertial collection. Mucus is used to ensure that the particles do not become re-entrained in the flow. Ultimately, the particles are discharged from the body or become swallowed. This protects the lungs from dust ingress. However, the prevention of dusty environments by good working practices, such as fume hoods and flow booths is an important part of health and safety provision. The likelihood of powder deflagrations is also reduced if powders are not allowed to remain deposited on surfaces within operating environments.