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Airborne particles

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10.1 Introduction

In many parts of the world, atmospheric dust palls can reach concentrations that are deleterious to human and animal health (Figure 10.1). They are variously made up of fine, crystalline and fibrous minerals, mineral aggregates, toxic trace and other chemical elements, organic matter, gases and pathogens.

Natural airborne particles consist of dusts released by weathering and erosion of rocks, unconsolidated sediments, dryland soils, biogenic fibres and residues from forest fires and ash ejected during volcanic eruptions. Sea spray produces aerosols containing particles that are blown inland; the particles are commonly of salt, but can also consist of radionuclides such as those released in discharges during the accident at the Sellafield nuclear power station in north-west England in 1957.

Substantial amounts of particulate aerosols from human activities began to be released, initially as a result of burning and clearing of forests for agriculture in Eurasia from about 8000 years ago, but the quantities increased especially after the Industrial Revolution in Britain in the eighteenth century; such aerosols include dusts from mining and quarrying and the combustion of fossil fuel for energy generation. Exposed agricultural soils are another important source, particularly at local and regional scales. Particulate matter released by biomass burning (for example, from forest clearance and agricultural practices), particularly in developing countries, continues to be important.

There are references to the presence of dust particles in the atmosphere in 3000-year-old Chinese records and in classical Greco-Roman documents in relation to the Mediterranean basin. Dust of Saharan origin over the eastern North Atlantic is mentioned in ships' logs from the eighteenth century and written records of Saharan dust falls in Western Europe have increased steadily since the mid-nineteenth century. The 'dust-bowl years' of the 1930s, which affected the prairies of Canada and the panhandle regions of Texas and Oklahoma, as well as adjacent areas of New Mexico and Colorado, greatly increased public awareness of the threat to health and led to considerable research into particulate aerosols. The dust-bowl conditions arose largely from a combination of several years of drought and intensive agricultural practices.

Prolonged inhalation of mineral particles can damage health and has no known health benefits. Potentially toxic natural particulate dust includes several species of crystalline silica and fibrous silicates, notably asbestos minerals (principally chrysotile, crocidolite (riebeckite), anthophyllite, tremolite, actinolite and amosite), erionite, alkaline salts and dusts containing toxic trace elements (e.g. volcanic ash particles, which hold transition metals and other toxic trace metal on their surfaces: Witham *et al*., 2005). Potentially toxic particulate dust arising from anthropogenic activities includes quartz and other silicates from quarrying and mining, agricultural biomass burning and wild fires and higher-rank coal dust (giving rise to coal-worker's pneumoconiosis) from coal extraction and processing. Other major anthropogenic particle sources include the burning of fossil fuels, combustion of diesel oil and cement manufacture.

Many rocks are made up mainly of silicates and pure silica (predominantly quartz), but understanding the impact of

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Figure 10.1 Before and during a Mongolian dryland dust pall over Beijing, March 2003. Visibility is ca. 50 km (left) and 1 km (right) (*Photos: Edward Derbyshire*)

prolonged inhalation of such dusts comes almost entirely from occupational medicine. It has been known for centuries that workers employed in industries such as mining, quarrying, sandblasting, silica milling and stone masonry are particularly exposed to fine, crystalline quartz dust and can develop inflammation and fibrosis of the lung (silicosis), which is one of the most studied occupational lung diseases. Crystalline silica is also classed as a human carcinogen (International Agency for Research on Cancer, 1997).

In contrast, the impact of high concentrations of naturally occurring silica-rich dust on human and animal health received little attention until recently. The earliest account, based on studies of a small population in the Sahara Desert, was published only half a century ago (Policard and Collet, 1952), although 'desert lung syndrome' (non-occupational silicosis with asthmatic symptoms including dyspnoea (breathlessness) and fatigue) has been known for over a century; evidence of it has been found in ancient Egyptian mummies (Tapp *et al*., 1975). In the past half-century this condition has been reported from several dryland regions including Pakistan, California, Ladakh (north India), the Thar Desert of Rajasthan (north-west India) and northern China.

Large quantities of silica and silicates, together with a range of chemicals including potentially toxic trace elements, are released during some volcanic eruptions. Inhaled ash can exacerbate symptoms in people who are susceptible to asthma and respiratory disease. Such an increase in health problems followed the 1980 eruption of Mount St Helens in the United States, when total suspended particulates (TSPs) exceeded 1000 $\mu g/m^3$ for 7 days, compared with a mean ambient 80 $\mu g/m^3$ (Baxter *et al*., 1983). Increased wheeze symptoms in children have also been reported from Montserrat, in the West Indies, following the Soufrière Hills volcanic eruptions (1995 to the present) (Forbes *et al*., 2003). It is still not clear, however, if exposure to volcanic ash is associated with chronic diseases such as silicosis (Horwell and Baxter, 2006); given that approximately 9 per cent of the world's population live near a historically active volcano, this is an important question.

Asbestosis is a progressive, incurable chronic lung disease, which has been known for at least a century and is attributable to prolonged exposure to fibrous silicates known collectively as asbestos (see Section 10.5.1). Unfortunately, the important insulation and fireproof properties of asbestos saw its widespread use in many industries, including building construction, ship building and industrial refrigeration plants, despite the known link to serious lung disease. World production and use of asbestos did not decline until the mid-1970s (Wagner, 1997), but asbestos continues to be used quite extensively in the automotive and other transport industries in brake shoes and pads in cars, trains and other vehicles. Asbestiform particulate matter from rocks, sediments and soils may also be transported naturally by wind. Cases of non-occupational asbestosis in people exposed to agricultural soil dusts have been reported in several European countries, including Italy, Turkey and France (Corsica) (Baris *et al*., 1987; Selçuk *et al*., 1992; Viallat *et al*., 1999; Magnani *et al*., 1995) and in villagers in the Troodos region of Cyprus who had no involvement in the open-cast mining of asbestos on that island (McConnochie *et al*., 1987). Pleural mesothelioma can occur after brief exposure to relatively low levels of amphibole asbestos such as crocidolite or tremolite. Prolonged exposure is not necessary (see Section 10.5).

Toxic trace elements are commonly found in, or adsorbed on to, dust particles. Their impact on lung tissue is poorly understood, but they are associated with inflammation of lung airways, giving rise to acute conditions such as asthma, bronchitis and rhinitis, as well as chronic conditions including fibrosis. Pathogens, including bacteria, fungi, pollen and spores, have been found in cavities in airborne mineral particles. They have been associated with a wide range of environmental impacts including Saharan dust palls acting as a carrier of the meningitis pathogen (*Neiseria meningitides*) (Molesworth *et al*., 2003). Other bacterial pathogens found in Middle East dust palls (Lyles *et al*., 2005) raise the question of the role of dust-transported pathogens in epidemics and pandemics. A particular case associated with Gulf War Syndrome and the health of military veterans involves the use of munitions containing depleted uranium (DU) during conflicts in the Middle East and the Balkans. The concern involves inhalation of DU aerosol particles and the toxicity of the uranium. Inhaled particles can enter the bloodstream by way of the lungs, ultimately affecting kidney function. Alternatives to DU, such as tungsten, may be just as toxic.

The annual release of fine silt and clay particles, commonly with alkali salts, by wind action on seasonally dry and former lake depressions (palaeo-lakes and artificially drained water bodies such as Owens Lake (USA) and the Aral Sea (Uzbek Republic)) also constitutes a health risk, causing acute irritation of the lungs (Gomez *et al*., 1992).

There are adverse health impacts from the extraction and burning of coal. It has been known for decades that chronic exposure to coal dust can give rise to incurable fibrotic lung diseases. Coal-worker's pneumoconiosis is not a response to a specific mineral; the presence of quartz in the coal was thought to influence the toxicity of coal dust, but this is only true when very high concentrations of quartz are present, when silicosis develops (see Section 10.5.2; McCunney *et al*., 2009). However, the presence of clay minerals may inhibit the toxicity of quartz in coal dust. Mineralised coal, including much of the coal in China (United States Geological Survey, 2000) may be enriched in toxic trace elements such as arsenic, fluorine, mercury, antimony and thallium as well as radioactive elements such as uranium and its decay products such as radium and radon. Burning such coal in unventilated stoves exposes families to toxic smoke (Finkelman *et al*., 2003) and combustion in coal-fired power stations exposes both local and regional populations.

Emissions from industrial coal combustion have been greatly reduced in most rich countries, but emission levels in countries such as China and India remain high. Incomplete coal combustion releases inhalable, unburned carbon and other particles potentially containing toxic trace elements (TTEs). The health impacts can be severe, as in the London smog of November 1952 when a temperature inversion trapped fog and particles from burning coal for several days, leading to 4000 excess deaths. The incident led to the UK Clean Air Acts of 1956 and 1968, which legislated for smokeless fuels, relocation of power stations away from urban areas and construction of tall chimneys to disperse pollution.

Emissions from diesel road and rail vehicles release polluting gases and particles that consist mainly of carbonaceous soot. The clustered spherules are ultra-fine (mean diameter 10–80 nm) and, as about 95 per cent of dust exhaust particles are less than 1 μm in diameter, most remain in suspension and are readily inhaled. Exhaust particles make up about 40 per cent of the particulate matter finer than 10 μm (PM_{10}) aerodynamic diameter in the airborne dust fraction in cities such as Los Angeles (Diaz-Sanchez, 1997).

Biomass burning, as a common agricultural practice or as a result of lightning strikes, has a strong impact on temperate, savannah and some equatorial landscapes, to the extent that some ecosystems depend on periodic fires. In addition to gases, particles emitted include organic carbon and silica from the tissues of plant species such as grasses and sugar cane (Le Blond *et al*., 2008).

The impact of high levels of pollutants in smoke from wild or bush fires, although sometimes intense, is generally short-lived (Morawska and Zhang, 2002), so that healthy people generally recover quickly from acute exposure. The effects can be more serious in people with pre-existing conditions such as pulmonary and cardiovascular diseases, as well as the old and the young.

Whole communities in central and eastern Asia are exposed to the adverse effects of natural airborne dusts at exposure levels encountered elsewhere only in some high-risk industries. The level of exposure to the dust hazard is considerable in China's semi-arid north and west, which includes a range of silt-rich dynamic terrain types, from large river flood plains, where silt is deposited during flash flooding events, to annually replenished sedimentary fans and a plateau covered by extensive loess (wind-lain mineral dust deposited in the past 2.6 million years) with a total area of about 0.75 million km^2. Farmers and herders are at particular risk and, in this huge region in which most domestic dwellings are built of *adobe* (a mixture of fine silty clay and water), indoor mineral dust can reach high levels, usually exacerbated by open-fire smoke from cooking hearths. Burning of low-grade coal, wood and animal dung inside such dwellings increases the impact of airborne particulate matter on human health over large tracts of China, northern India, Tibet and Africa.

The prime target of mineral-related disease is the lungs and lung-lining by inhalation of airborne dust, leading to the respiratory and related diseases that form the focus of this chapter. However, there are other exposure routes, such as absorption of ultra-fine volcanic clay minerals through the feet, leading to podoconiosis, an endemic disease in Africa, characterised by chronic lymphatic irritation, inflammation and collagenesis, leading to obstruction and lymphoedema of the lower limbs (Davies, 2003).

10.2 Hazardous properties

The effect on the human body from inhaling atmospheric particulate matter varies widely in response to many factors, including certain characteristics of the particulate matter and the nature of

any pathogens attached to them, the dust concentration, the length of the exposure period and an individual's personal vulnerability.

The hazardous properties of airborne dusts vary with their geochemistry, mineralogy, degree of crystallinity, size, shape, density, solubility and reactivity with human fluids and tissue (Guthrie, 1997). Fine particles may remain suspended in the atmosphere for hours to weeks depending on their size, form and density. They may also be easily re-suspended by natural and human action. All particulate matter finer than 10 μm aerodynamic diameter is referred to as the respirable fraction or PM_{10} within which the fine fraction (<2.5 μm, or $PM_{2.5}$) and the ultrafine fraction (<100 nm) are of particular importance to pollution and public health.

10.2.1 Crystalline silica

Crystalline silica, as fine particles of quartz, cristobalite and tridymite, constitutes a major health hazard. Quartz is the commonest and by far the most studied polymorph, but both cristobalite and the rarer tridymite are regarded as being equally or more pathogenic. Crystalline silica is a highly fibrogenic agent in lung tissue, by a process that appears to arise from the reactivity of particle surfaces in contact with lung-lining fluid and cells. Reactivity of quartz surfaces is greatest when particle surfaces are freshly fractured (either by artificial or natural processes, Table 10.1). Non-crystalline (amorphous) silica, present in diatoms, phytoliths and testate amoebas, generally lacks surface radicals and thus is regarded as benign when inhaled. However, there is a minority view that the presence in wind-blown dust of impurities and particles such as soil-derived sponge spicules with aerodynamic diameters of between 5 and 3 μm (thus meeting the definition of hazardous mineral fibre: Skinner *et al*., 1988) 'raises the possibility that they may present a respirable silica hazard in dust-affected areas' (Clarke, 2003). Potential pathogenicity of crystalline quartz is moderated or nullified by aging of particle surfaces, by the presence of adhering minerals or coatings and by the presence of small amounts of contaminant metals, notably aluminium and iron. The presence of aluminium, commonly in the form of clay minerals such as kaolinite, substantially moderates quartz toxicity.

Crystalline silica is odourless and a non-irritant when inhaled, with no noticeable symptoms, but protracted exposure over long periods (the dose) may lead to progressive thickening and scarring of lung tissue, although radiographically visible symptoms of silicosis may take many years to appear (see Section 10.5.2). Prolonged progression may result in death owing to cardiopulmonary failure. Cell response to silica varies widely, both among and within its polymorphs, such that silica cannot be regarded as a single, discrete hazardous material (Donaldson and Borm, 1998).

The processes by which particles induce pulmonary pathogenicity are complex and incompletely understood. Particles in the lungs must be cleared rapidly if the gas-exchange system is to work effectively. Problems arise when particles such as crystalline silica or asbestos are not cleared, primarily because of complex interaction with cellular components, or because the shape–size characteristics inhibit particle transport within the pulmonary airways. Free-radical release and the formation of reactive oxygen species (ROS) are thought to be primary processes inducing inflammation. In the case of crystalline silica, breaking of the bond between Si and O by homolytic and heterolytic cleavage generates dangling bonds (reactive surface radicals $Si^{\bullet}$ and $SiO^{\bullet}$) and surface charges (Si^{+} and SiO^{-}), respectively (Fubini *et al*., 1995) (Figure 10.2). Particle-derived ROS from crystalline silica particles in contact with epithelial cells enhance both oxidative stress and inflammation (Fubini, 1998a, 1998b; Fubini and Hubbard, 2003; Figure 10.3).

Traces of iron on crystalline silica surfaces sustain production of free radicals in a series of catalytic stages (the Haber Weiss cycle) involving the Fenton reaction in the lungs (formulas 10.2–10.4).

Table 10.1 Characteristics of particulate matter determined by its origin (Modified after Fubini, 1998b)

Comminution of crystals e.g. grinding through industrial or natural (e.g. volcanic and glacial) processes	Combustion e.g. coal fly ash, diesel particulates	Biogenic e.g. diatoms, sponge spicules
Sharp edges and corners	Spherical particles	Retention of organism shapes
Irregular surface charges	Smooth surface	Indented, irregular surfaces
Surface radicals	No surface radicals	High surface area, particularly internal surface area
Hydrophilic	Hydrophobic	No surface radicals
Contamination by grinding processes and co-existence with other minerals	Contamination by carbon and other component processes	Very hydrophilic
		Sometimes contains alkaline, alkaline earth and iron ions from original material

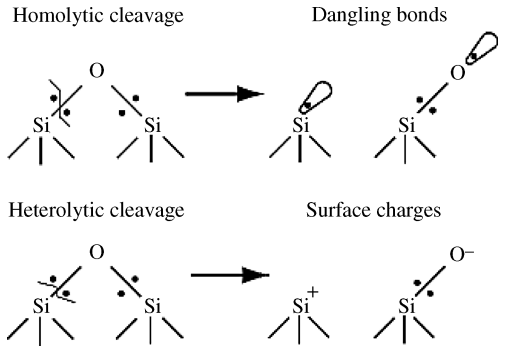

Figure 10.2 Diagram to show homolytic and heterolytic cleavage of silica tetrahedra. Silicon/oxygen radicals and surface charges are generated during cleavage of atomic bonds due to, for example, fragmentation of crystalline silica during quarrying or volcanic eruption (After Fubini *et al.*, 1995)

The hydroxyl radical ($HO^{\bullet}$) is produced through the Fenton reaction (10.4, below) (Fubini and Otero Arean, 1999):

$$Fe^{3+} + \text{reductant} \rightarrow Fe^{2+} + \text{oxidised reductant} \qquad (10.1)$$

$$Fe^{2+} + O_2 \rightarrow Fe^{3+} + O_{2^-}\cdot \qquad (10.2)$$

$$O_{2^-}\cdot + 2H^+ + e^- \rightarrow H_2O_2 \qquad (10.3)$$

$$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + OH^- + HO^{\bullet} \qquad (10.4)$$

Trivalent Fe on crystalline silica surfaces in contact with H_2O_2 in the body is converted to divalent Fe to generate highly reactive hydroxyl radicals ($HO^{\bullet}$), leading to a cycle of increasing cell damage (Fubini and Otero Aréan, 1999). Free-radical generation can cause mutations in DNA, leading to inflammation and cancer (Hardy and Aust, 1995). Particle-induced inflammation is central to a wide range of occupational diseases (Figure 10.4).

The progression from chronic inflammation to fibrosis and silicosis following long-term exposure to fine crystalline silica particles also has deleterious effects on the immune system. For example, the link between silicosis and tuberculosis was established decades ago (Snider, 1978), the risk to silicotic patients of developing tuberculosis being up to 20 times the level found in the general population (Westerholm, 1980). There is a reduction in the ability of the macrophages to inhibit the growth of tubercle bacilli responsible for tuberculosis. Some rheumatic, as well as chronic renal diseases also show higher than average incidence in individuals exposed to silica and it is likely that increased susceptibility to some mycobacterial diseases is due to impaired function of macrophages in silicotic lungs (Snider, 1978).

10.2.2 Asbestos

Asbestos and asbestiform fibres constitute a severe health hazard. Inhalation over a period of years can give rise to fibrosis (asbestosis) in industrial situations and also in some natural environments. Crocidolite is the most pathogenic form of asbestos. Together with chrysotile and amosite, it can induce pulmonary fibrosis, bronchogenic carcinoma, mesothelioma and a number of pleural diseases (Wagner *et al.*, 1960). In addition, amphibole asbestos and particularly crocidolite, causes pleural mesothelioma. The increased toxicity of amphibole asbestos over that of serpentine asbestos (chrysotile) is due in part to the high aspect ratios (i.e. length to width ratio greater than 3) and durability of the amphibole asbestos, where the fibres are long, thin and needle-like and are biopersistent (see Section 10.5.1). Asbestiform compounds have mineral aggregates with the distinctive features of amphibole asbestos, namely discrete, long,

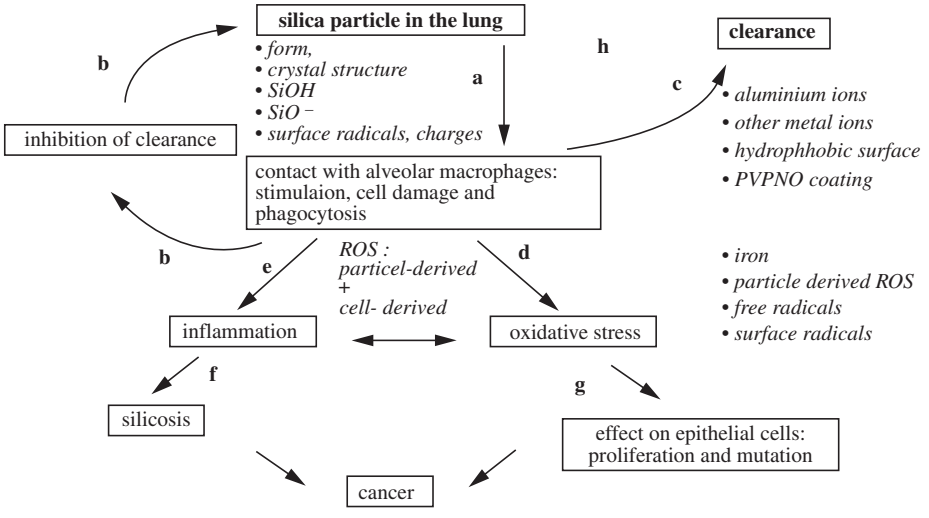

Figure 10.3 Sequence of events leading to quartz pathology, based on animal studies and cells *in vitro* (After Fubini, 1998a, modified from Donaldson and Borm, 1998)

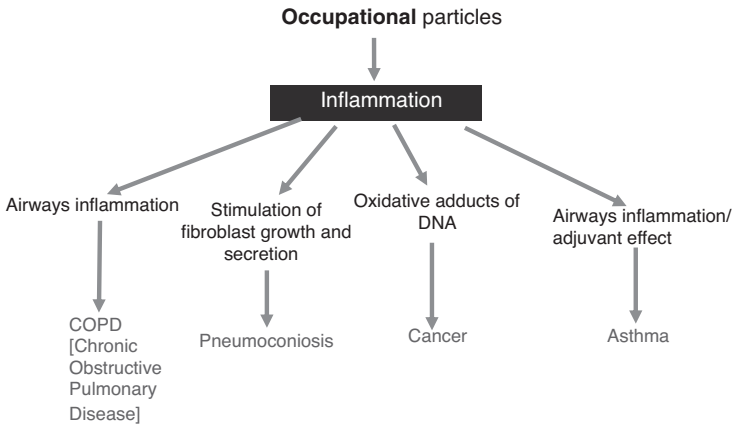

Figure 10.4 The central role of inflammation in a number of occupational diseases (Courtesy of Professor K. Donaldson)

thin, strong and flexible fibres in bunches or mats. Fibrogenesis arising from inhalation of asbestos fibres spreads out along elements of the lung structure, rather than as discrete nodules as occurs with silicosis. Some fibres penetrate tissue and remain in the lungs, lung lining and abdominal cavity. On deposition within the conducting and alveolar regions, their surfaces are modified by adsorption of macromolecules which may enhance cell damage due to generation of free radicals. To count as a 'fibre', the World Health Organisation states that an asbestos particle must have an aspect ratio greater than 3:1, but the most important single property for respirability is fibre diameter, which should be less than 3 µm (World Health Organisation, 1986). Once in the lung, fibre length, surface chemistry, solubility and other physical and chemical properties control biological activity.

Response to grinding varies with species. For example, prolonged grinding deactivates chrysotile but in other asbestos fibres grinding may raise reactivity, releasing the reactive oxygen species that enhance oxidative stress, perhaps because of exposure to small amounts of ferrous iron (Neijari *et al*., 1993; Fubini, 1997). Oxidative stress induced by asbestos fibres increases with the rate of free-radical production (Ghio *et al*., 1998). Pathogenicity may be affected by adsorption of metal ions on mineral dusts. For example, the presence of iron on asbestos fibres is thought to influence the catalysing release of free radicals (Hardy and Aust, 1995).

Although not a member of the asbestos group, the fibrous zeolite mineral erionite is regarded as a serious environmental health threat, being between 200 and 1000 times more carcinogenic than asbestos minerals (Hill *et al*., 1990; Carbone *et al*., 2002). In the Cappadocia region of Turkey, malignant mesothelioma accounts for more than 50 per cent of deaths in some villages (Dogan *et al*., 2008), caused by exposure to natural wind-borne erionite-rich particles. Incidence rates can be 10 000 times greater than in the general population, though it has been claimed that epidemiological evidence points to genetic susceptibility as an additional factor (Roushdy-Hammady *et al*., 2001).

10.2.3 Toxic trace elements

Another group of particles that occur as airborne dust comprises toxic trace elements (TTEs) (see also Chapter 4). These include the well-studied elements mercury, lead, arsenic, cadmium and iron and also fluoride, radioactive elements and other trace metals such as copper and zinc. They may occur adsorbed on airborne mineral dust and as integral constituents of such particles. Most knowledge of the adverse health effects of inhaled and ingested TTEs comes from occupational medicine. Toxic trace elements can cause both upper and lower airway injury and sensitivity (Cook *et al*., 2005). Arsenic and mercury can cause rhinitis and sinusitis, while mercury and zinc and many other TTEs can cause tracheitis, bronchitis and asthma. Inhalation of elements such as cadmium causes inflammation, oedema and fibrosis of the parenchyma. Iron can act catalytically in free-radical generation, leading to greater oxidative stress and epithelial cell damage. Trace amounts of iron help to generate ROS, leading to DNA damage, cell transformation and pulmonary reaction.

The potential of TTEs to cause damage is related to their solubility: high solubility reflects dissociation and dispersal deep into lung tissues and a degree of insolubility is associated with deposition in the airways (Newman, 1996). A recent study of selected TTEs in a dryland dust pall over Beijing, using an *in vitro* plasmid assay, showed that dust-particle bioreactivity derives mainly from the water-soluble fraction (Shao *et al*., 2006). Total water-soluble concentrations of aluminium, vanadium, chromium, manganese, iron, cobalt, nickel, copper, zinc, arsenic and lead were found to be higher in the more strongly bio-reactive samples and water-soluble zinc was the element responsible for most plasmid DNA damage. However, the chemical processes by which metals cause pathophysiological responses, especially as compounds, remain poorly understood (Cook *et al*., 2005).

As mentioned above, iron can act catalytically in free-radical generation, leading to greater oxidative stress and epithelial cell damage. Trace amounts of iron help to generate ROS, leading to

DNA damage, cell transformation and pulmonary reaction. However, bioreactivity varies with the type of iron oxide and other factors. Toxic reaction of lung tissue to dust particles is also proportional to the total water-soluble concentration of trace elements in the dust, a factor that affects oxidative capacity.

As with other natural particles, the potential toxicity of volcanic particles depends not only on mineral composition, grain size and surface area but also on the presence of TTEs and complex factors relating to the interaction of the TTEs, particle surface, lung tissue and cells (i.e. surface reactivity). It is known that basaltic ash produces particularly high numbers of hydroxyl free radicals through interaction of reduced iron on the surface of ash particles with hydrogen peroxide (Horwell *et al*., 2007). Fluoride is injected into the atmosphere by volcanic activity and other natural processes, as well as from anthropogenic inputs. Airborne fluoride is toxic if high concentrations are inhaled or ingested. It is rapidly absorbed following intake and has a high affinity for calcified tissue, including bones and teeth. Indoor combustion of fluoride-rich coal can lead to high concentrations of particles and fluoride, as is seen in some Chinese rural communities. High concentrations of urinary fluoride have been noted in fluoride-rich coal-burning regions and, in some Chinese villages, all elementary and junior high-school students between 10 and 12 years old were found to have dental fluorosis (Ando *et al*., 1998).

Manganese (Mn) is an essential trace metal, but excessive exposure is associated with neurotoxicity, notably in mining, the ferroalloy and battery industries and welding (Lucchini *et al*., 1999). Released manganese-rich dust has been implicated in manganese-induced Parkinsonism (Koller *et al*., 2004), inhaled Mn dissolving in body fluids to be deposited in the striatum of the brain (involving damage to the basal ganglia structures). Common symptoms are weakness, tremor, gait abnormality and slowness of speech. Manganese-induced Parkinsonism can be differentiated from other forms of Parkinsonism using clinical and imaging techniques (Cersosimo and Koller, 2006).

10.2.4 Diesel fuels

The emissions from the burning of liquid fossil fuels have been causally linked with a large number of adverse health effects. The principal path into the body is respiratory and, once inside the lung, particles can translocate to numerous and diverse locations within that organ. The particles possess the intrinsic hazardous property of being harmful as well as acting as carriers for bioreactive compounds that have condensed on their surfaces, or metals that have been trapped within the carbonaceous structure of the spheres. Exposure to these particles ranges from short-term, high-intensity exposure (usually occupational), which can result in irritation to the eyes and the respiratory tract, to longer-term exposure resulting in compromised lungs, coughing and breathlessness. There is good evidence to support the view that long-term exposure increases the risk of lung cancer, COPD, heart attacks, strokes and numerous other conditions (Neuberger *et al*., 2007). Human exposure investigations have shown that roadside exposure to traffic fumes results in decreased lung function in asthmatic children (Delfino *et al*., 2009).

10.2.5 Biomass and wild fires

Air pollution arising from natural fires (or bushfires) and the seasonal burning of vegetation can have a severe impact on large populations locally and sometimes at long distances. Combustion of vegetation releases solids, hydrocarbons, organic compounds and gases, but particulate matter and polycyclic aromatic hydrocarbons (also called polynuclear aromatic hydrocarbons) are of particular concern with respect to human health (World Health Organisation, 1999), especially as amorphous species may be converted into crystalline minerals during combustion. A majority of discrete particles are ultra-fine, with most of the particle mass in the less than 2.5 μm range. The smaller, fine particulate matter consists of up to 70 per cent organic carbon (Morawska and Zhang, 2002).

10.2.6 Micro-organisms

The impact on lung tissue of airborne mineral dust may be exacerbated by the presence within fine dust particles of bacteria, fungi and other micro-organisms (Kellogg *et al*., 2004). The global extent of the fine-dust transport system has been implicated in damage to plants and animals, including impairment of human health. Although susceptible to destruction by ultraviolet radiation, a proportion of any included micro-organisms may survive in cavities and cracks within suspended dust particles. Dryland dust can act as a carrier for the meningitis pathogen. A study in Mali (Kellogg *et al*., 2004) found that, of 95 dust-borne bacteria identified, 25 per cent were opportunistic pathogens. No outbreaks of disease attributable to fungi in dust palls have been reported, but the prevalence of fungi in dryland soils and dust storms indicates diverse communities including pathogenic genera and species. More than 40 fungal colony-forming units or spores have been found within dust palls (Griffin, 2007); most of the genera known to contain pathogenic species are derived from the Sahara, Sahel and Middle East. A wide range of human diseases is caused by pathogenic or opportunistic pathogens and some fungi listed include mild to potent allergens. Short-range transmission of infectious human viruses is well documented, but little is known about long-range transmission within dust storms (Griffin, 2007).

10.3 Sources

10.3.1 Natural sources

10.3.1.1 Crustal dust

World dust emissions from arid and semi-arid terrains can reach as much as 5 billion tonnes per year (Schultz, 1980). Sources of natural dust occur on all continents, but the most extensive

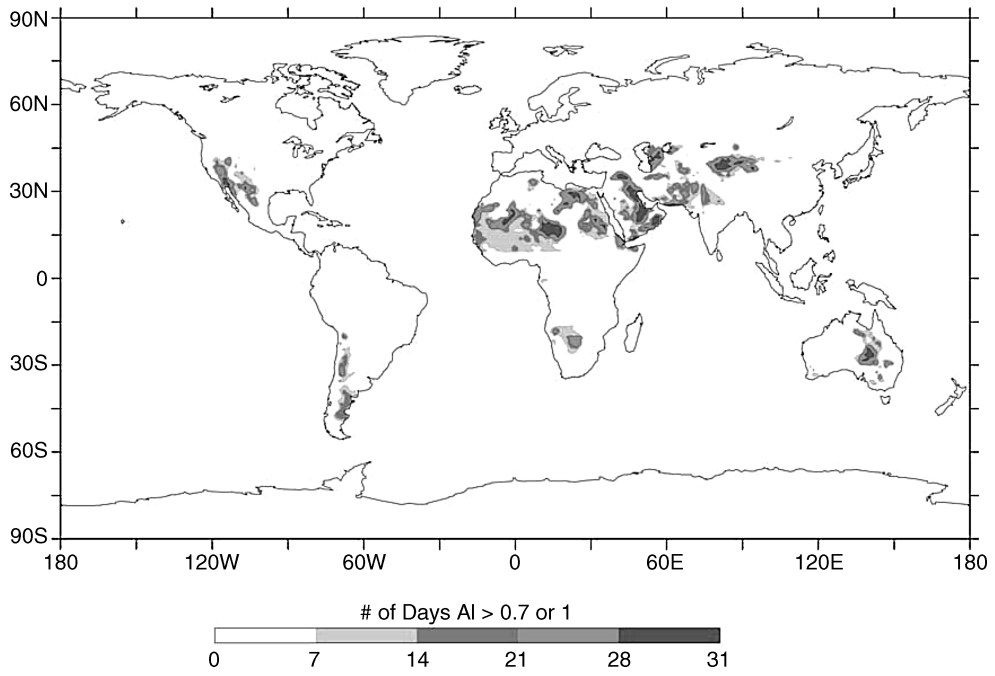

Figure 10.5 Total ozone mapping spectrometer (TOMS) indicating main dust source regions. UV spectral contrast is used as an absorbing aerosol index (AAI). An AAI threshold of 1.0 is used in the 'global dust belt' (West Africa to China) and 0.7 elsewhere (After Prospero *et al.*, 2002, with kind permission)

source region stretches from the western Sahara, through the Middle East and central Asia to the Yellow Sea (Figure 10.5). The distribution of palaeo-lake beds closely matches the major source areas of atmospheric dust (Ginoux *et al.*, 2001; Tegen, 2003: Figure 10.6). The lake deposits in the Bodelé depression (formerly mega-lake Chad, North Africa) include abundant broken particles of diatomite (siliceous shells of a eukaryotic algae: Figure 10.7); and fluxes from this source can

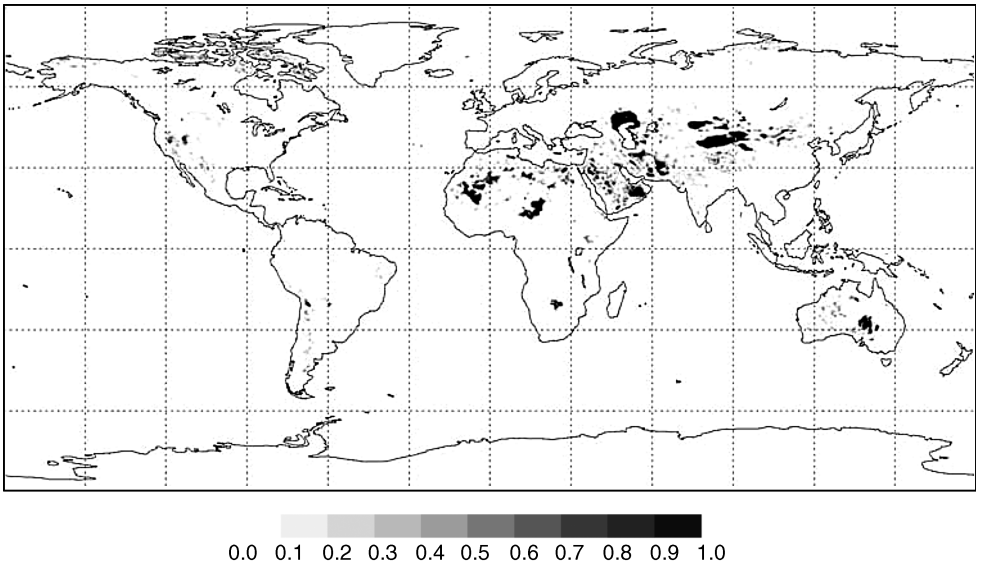

Figure 10.6 Areal coverage of preferential dust sources, calculated from the extent of potential lake areas, excluding actual lakes (After Tegen, 2003)

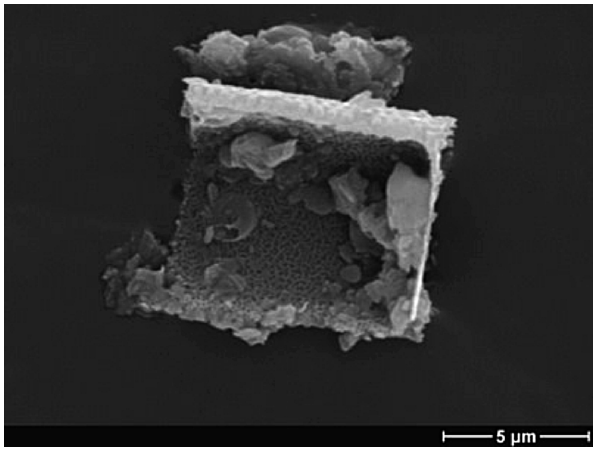

Figure 10.7 Fragment of a diatom abstracted from a Saharan dust fall on Gran Canaria (Canary Islands) on 4 March 2004 (*Photo Edward Derbyshire*)

reach 1.18 ± 0.45 Tg per day, with an estimated contribution to global dust emissions of between 6 and 18 per cent (Todd *et al*., 2007).

The massive deposits (>300 m thick) of wind-lain mineral dust known as loess in northern China are impressive evidence of sustained, semi-continuous deposition of the coarser airborne dust fractions (silt and sandy silt) for at least the past 2.6 million years. The loess formation of north-west China, made up of alternating silt (loess proper) and clayey silt palaeosols (fossil soils), marking, respectively, substantial dust depositional rates and reduced dust depositional rates with soil profile development, is supplied by sources lying to the north and west (see Figures 10.12 and 10.13), a natural process that has continued at varying intensity for at least the past 7 million years (Ding *et al*., 2001). Huge loess units such as the Loess Plateau of north-west China are subject to natural erosion by streams and rivers and mass movements (landslides) as well as to anthropogenic actions such as deforestation, excessive irrigation, intensive agricultural practices and overgrazing. When the loess surface is breached by such actions, it is highly susceptible to wind erosion, making such degraded loess a notable but secondary source of present-day wind-blown dust.

The toxicity of natural dust is greatly influenced by its composition, which varies according to its provenance (Krueger *et al*., 2004). The percentage of quartz in airborne dust samples from North Africa and China varies from less than 20 per cent to more than 60 per cent, clay minerals from less than 10 per cent to more than 40 per cent and carbonates up to 40 per cent.

There are relatively few studies of natural particulate matter in major dust source regions using systematic sampling and analysis. One study obtained a year's airborne samples from 15 sites across the Middle East: Djibouti, Afghanistan, Qatar, United Arab Emirates, Iraq and Kuwait (Engelbrecht *et al*., 2009). All of the sites exceeded the WHO guidelines for maximum ambient particulate matter exposure (20 µg/m^3 for PM_{10} and 10 µg/m^3 for $PM_{2.5}$) and all contained mixtures of silicate minerals, carbonates, oxides, sulphates and salts. Dust events were found to yield short-term maxima of the major soil-forming elements silicon, aluminium, calcium and manganese, as well as magnesium, potassium, titanium, vanadium, iron, ruthenium, strontium, zirconium and barium. All quartz and other silicate mineral particles were thinly coated with a silicon-aluminium-magnesium layer made up of clay minerals. Despite the high dust concentrations, none of the quartz particles had fractured surfaces; they were partly rounded and coated with clay minerals and iron oxides suggesting that the dust is of low toxicity. No asbestos fibres were found.

10.3.1.2 Asbestos

Sources of asbestos-rich dust occur in both the natural and built environments. Asbestos occurs in metamorphic rocks in orogenic belts around the world. Chrysotile (white asbestos) occurs in ultramafic rocks that have been altered to serpentinite. The other main types of asbestos are the amphibole minerals crocidolite (blue asbestos) found in feldspathoid rocks and amosite (brown asbestos; fibrous anthophyllite) found in metamorphosed basic and ultrabasic igneous rocks. The Jurassic to Cretaceous metamorphic rocks of north-east Corsica, including outcrops of *schistes lustrés,* are rich in amphiboles, serpentinite and chrysotile. There is no history of occupational contact with asbestos, but it is reported that 94.6 per cent of villagers born in north-east Corsica have bilateral plaques in the lung, compared with only 5.4 per cent of subjects in villages in the north-west. The disease has been linked to the presence of abundant airborne chrysotile fibres (Boutin *et al*., 1986). Erionite, a fibrous zeolite, occurs in volcanic tuffs and agricultural soils in the Cappadocia region, central Turkey. Weathered rock and fine soil particles make up dust palls that affect the health of village communities in dry periods, a situation exacerbated by the use of erionite-bearing rocks and soils as building stone and as a whitewash on buildings.

10.3.1.3 Volcanic ash

Substantial volumes of silica and silicate minerals may be emitted during volcanic eruptions, depending on the type of magma erupted. Basic (e.g. basaltic) eruptions produce silica-poor (<52 wt per cent) ash, which contains mafic minerals such as calcium-rich feldspar, pyroxenes and olivine, whereas acidic (e.g. rhyolitic) eruptions produce silica-rich (>69 wt per cent) ash with high concentrations of felsic minerals such as quartz, potassium-rich feldspar and silica glass. Basaltic volcanoes are usually found above oceanic hot spots such as the Hawaiian island chain and where Earth's crust is thin. Volcanoes erupting more evolved magma, with more silica (such as andesitic, dacitic and rhyolitic volcanoes) are generally found around Earth's subducting plate margins, e.g. the Pacific Ring of Fire which has given rise to the volcanic chains stretching from Chile, Mexico, around the western US coast, eastern Russia, Japan, Philippines, Indonesia and south to New Zealand.

The amount of crystalline silica generated varies substantially depending on the type of volcano, with basaltic volcanoes producing no free silica. The andesitic (~57–63 wt per cent silica) Soufrière Hills volcano, Montserrat, West Indies (part of the Lesser Antilles island arc) generates abundant crystalline silica in the form of cristobalite, formed by vapour-phase crystallisation and devitrification of volcanic glass within its volcanic dome. Superheated, silicon-rich vapours escaping from the volcano pass through pore networks and cracks in the lava, depositing cristobalite as a metastable mineral in voids. At the Soufrière Hills volcano, the ash produced during collapse of the dome contains in the region of 15 wt per cent cristobalite (Horwell *et al.*, 2010).

The quantity of respirable material (<4 μm aerodynamic diameter) varies greatly among different types of volcanic eruptions, related to magma composition, with increasing explosivity correlating with increasing silica content and viscosity. Basaltic eruptions, which are generally effusive, produce coarse particles with <5 vol. per cent respirable material. Andesitic, dacitic or rhyolitic eruptions, which are usually more explosive, produce finer particles. The eruption of Vesuvius, Italy, in AD 79 produced ash with around 17 vol. per cent respirable material. Dome-forming eruptions such as the Soufrière Hills volcano, Montserrat, can generate ash with about 10–15 vol. per cent respirable material during dome collapse (Horwell, 2007).

The crystalline silica content of volcanic ash is the main cause of concern in terms of health hazard but, in recent years, other potential hazards have been identified. For example, volcanic ash can generate substantial quantities of hydroxyl radicals through the Fenton reaction. Iron-rich basaltic ash has greater reactivity than iron-poor ash (Horwell *et al.*, 2007).

10.3.2 Anthropogenic sources

Human activities that emit fine dust include agriculture, mining and quarrying and combustion of fossil fuels for energy generation and transport. The expansion of agriculture over the past 200 years, especially in the world's sub-humid and semi-arid marginal lands, has involved ploughing fragile soils with heavy machinery, leading to the re-suspension of loess and fine soil particles. The problem is exacerbated by the use of fertilisers and pesticides and the large-scale diversion of water for irrigation and domestic use from rivers and lakes, which impacts on the toxicity of the dusts and enhances erosion.

Fertilisers and pesticides contaminate groundwater and some are sorbed on soil particles that are entrained by strong winds and transported for long distances. The addition of trace elements, such as arsenic, cadmium, chromium, copper, molybdenum, nickel, lead, uranium, vanadium and zinc in fertilisers such as phosphates further increases the toxicity of soil-derived dust palls. The Aral Sea in Kazakhstan has been progressively diminished since 1960 by diverting feeder streams and canals for irrigation, with reduction of the water surface area by 80 per cent. The input of fertilisers and pesticides across the Aral basin has made the periodic dust palls more toxic. Morbidity and mortality rates have increased greatly across the region. Over 50 per cent of illnesses reported in children were found to be of respiratory type (O'Hara *et al.*, 2000; Wiggs *et al.*, 2003; Figure 10.8).

The extractive industries, which provide minerals for the metallurgical, energy, aggregate, cement and brick-making industries, generate dusts during crushing and grinding. Dust is

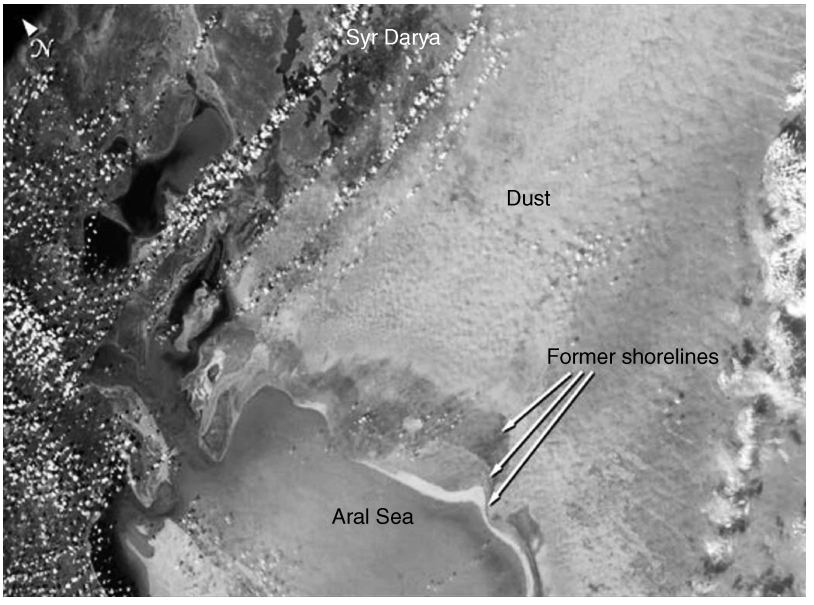

Figure 10.8 The diminishing Aral Sea on 30 June 2001, showing a pall of toxic dust, driven by a westerly wind (Reproduced by kind permission of Earth Sciences and Image Analysis Laboratory, Johnson Space Centre, USA)

also deflated from waste piles (tailings) if they are not kept moist. Such dust palls may represent a high risk to communities nearby and sometimes some distance away if TTEs are present in the tailings dust.

Public awareness of such hazards has increased recently. Following closure of a vermiculite mine in Libby, Montana, USA in 1990, it was found that the vermiculite was contaminated with tremolite-actinolite asbestos and radiographic pleural and interstitial abnormalities were found to be present in 51 per cent of former mine workers, increasing with age and exposure. Abnormalities in non-miner groups (3.8%) were also higher than elsewhere (United States Environmental Protection Agency, 2003).

Coal and oil combustion is also a major source of particulate matter, coal fly ash being widespread in industrial, many urban and some natural environments (Jones *et al*., 2009). Fly ash can contain a component of unburnt organic matter but, if this exceeds certain levels, it is common practice to re-inject this material into the furnace for secondary combustion. The smaller-sized fly-ash particles, typically around a few microns in diameter, are usually spherical and often display gas-expulsion holes. Larger particles tend to have a more blocky appearance and often have numerous smaller glass spheres adhering to the surface. In modern coal-burning factories or power stations, the vast majority of fly-ash particles are collected post-furnace by electrostatic collectors or in bag rooms. The fly ash is then usually dumped on nearby ash piles; thus, the potential exists for this fly ash to be blown into the atmosphere (Brown *et al*., 2009). The chemistry of the fly ash is controlled by the original chemistry of the coal (mineral impurities) and the operating conditions at the furnace. Although the coal minerals are completely melted into glass, after solidification there is often a small amount of recrystallisation producing quartz, mullite and haematite. The fly ash itself is not particularly reactive (Wlodarczyk *et al*., 2008), most having limited pozzalinic properties; usually it has to be mixed with Portland cement to make any sort of building material. However, it does contain a range of TTEs that can be leached out in certain chemical environments. These include arsenic, mercury, lead, barium, cadmium, chromium, copper, fluorine, nickel, selenium, zinc and uranium and its decay products.

The human-health implications of fly ash produced by domestic coal burning are currently a subject of concern. This is especially so in poorer countries where the coal is burnt in poorly ventilated rooms in order to retain as much heat as possible and, consequently, the fumes are also retained. In northern China, for example, the domestic burning of local Permian coals appears to have resulted in clusters of lung cancer (Shao *et al*., 2009). The relatively few studies of coal fly ash toxicity have yet to provide convincing evidence of human lung inflammation and there is a continuing debate about the relative importance of this material versus its TTE components. However, the absence of fibrosis in subjects exposed to coal fly ash indicates that the glass spheres are less bio-reactive than equivalent doses of crystalline silica (Borm, 1997).

There is compelling evidence that residual oil fly ash (ROFA) may be more harmful to human health than coal fly ash. ROFA is mostly less than 2.5 μm in diameter, chemically complex, largely inorganic and rich in metals, especially vanadium. Under the electron microscope, ROFA is seen to consist mostly of clusters of tiny carbon spheres; it is, therefore, soot produced by the burning of the oil. Lung inflammation, eye and throat irritation, cough, dyspnoea, rhinitis and bronchitis, have all been found in workers (notably in power-generating plants) exposed to high ash concentrations. Given the similarity of the symptoms associated with ROFA and vanadium exposure, transition metals, especially vanadium in ROFA, may be involved in Fenton-like chemical reactions in the lungs. Exposure to vanadium in the mining, steel and chemical industries is mainly by inhalation, however, suggesting the presence of ROFA particles in ambient air (Ghio *et al*., 2002).

Petrol and diesel-powered vehicles are an important source of gaseous and particulate atmospheric pollution. This is a field that has constantly changing trends, resulting in constantly changing particulate emissions. For petrol engines, the most important change in recent years has been the decrease in atmospheric lead levels following the ban on the use of leaded petrol in most developed countries (Cook and Gale, 2005). For diesel engines, there are a number of important trends that include significant increases in domestic diesel cars, as opposed to petrol cars, the development of the so-called new-generation diesel engines with sophisticated fuel-injection systems and tailpipe after-treatment devices, the promotion of low-sulphur diesel and the introduction of biofuels. The nature of the particles generated by diesel combustion is therefore a function of the type of fuel, the technology employed in the engine and exhaust systems, the running condition of the engine and ambient atmospheric conditions (Maricq, 2007). An overloaded truck straining to get up a steep hill will produce different particles from those released by the same truck driving along a flat road under normal operating conditions.

All types of liquid fossil fuels produce combustion particles (soot). These particles consist of nanometre-scale carbon spheres that can exist individually but, more commonly, rapidly aggregate into larger chains or clusters. Confusion exists as to whether consideration of the behaviour of these particles should take account of the size of the individual spheres (nanoparticles) or the whole clusters that can be PM_{10} or more in diameter. A pragmatic approach is to classify the particles as 'nanostructured', which means that the size of the cluster is considered. However, it is understood that these particles can, both chemically and biologically, behave like the constituent nanoparticles. The overall size of the particles is significant, as it has implications for collection methods as well as health issues such as the ability of the particles to penetrate deeply into the lung. The individual spheres have an onion-like structure of perturbed (turbostratic) small graphitic structures, which can be imaged by high-resolution techniques such as transmission electron microscopy. The spaces between the graphitic molecules in the primary carbon spheres can trap metal sourced from the fuel, engine and exhaust systems. This includes platinum-group

metals from the catalytic converters. The surfaces of the carbon spheres act as substrates for the condensation of a large variety of organic species, heavier-end hydrocarbons and inorganic species such as sulphates (Clague *et al.*, 1999). In addition to the carbon-sphere-based particles, other particles are generated by the direct condensation of both organic and inorganic species as the hot emission fumes cool to ambient air temperatures.

10.4 Global pathways

The detachment of mineral dust from the ground surface ('deflation') and its transport by the wind are functions of several variables, including the wind speed (notably the critical wind speed, or threshold velocity, required to dislodge particles), the degree of instability of the atmosphere, the size of the particles, the roughness and moisture content of the land surface and the degree of particle exposure. A recent classification of dust concentrations in relation to wind velocities in north-east Asia claims that sustained wind velocities of 10 m/s or more can generate total suspended solid loads in excess of 6000 $\mu g/m^3$ with PM_{10} fractions as high as 5000 $\mu g/m^3$ (Song *et al.*, 2007).

Silt-sized particles in the 10–50 μm range are readily picked up by the wind from dry, unvegetated surfaces, but, because of the high inter-particle cohesive forces typical of very fine (colloidal) materials, entrainment of clay particles (<2 μm) usually occurs as cohering silt-size aggregates or as attachments to silt-size grains. Critical wind threshold velocities vary markedly, those for the semi-arid and sub-humid silt-covered terrains of northern China being approximately twice those required to initiate dust storms in the Sahara (Wang *et al.*, 2000). It is important to discriminate between source-proximal and source-distal dust plumes (Pye, 1987; Figure 10.9). The mean size of entrained particles diminishes with transport distance because of fallout of larger and denser particles. Thus, the proportion of a dust plume consisting of the respirable fractions increases progressively with distance from the source, although the absolute mass of the respirable fraction is greatest close to source. Median diameters less than 10 μm characterise dust particles transported over long distances. The finest fractions (<1 μm) settle more slowly under gravitational force when in moving air streams, often staying at high altitudes for weeks. This is true of very fine-grained particulate pollutants, such as asbestos, which has been traced thousands of km from source.

Clearance of lower atmospheric dust by deposition occurs mainly as dry deposition, in which high atmospheric pressure is dominant, but it can also occur as wet deposition when dust particles are drawn down by raindrops in the unstable conditions associated with vigorous cells of low atmospheric pressure (depressions). Wet dust deposition is known regionally as 'loess rain' in eastern China and 'blood rain' in Mediterranean Europe.

The natural processes by which mineral dust is injected into the atmosphere are usually episodic (e.g. as in volcanic eruptions), often strongly seasonal and, in the case of dust deflated from the land surface, located mainly in sub-tropical arid and semi-arid regions. Such major dust sources cover about 30 per cent of the total land area of the Earth. The finest particulates are carried long distances along the following four main pathways (Tanaka *et al.*, 2005):

- the monsoon and westerly systems across Asia and trans-Pacific;
- the sub-tropical easterly system (Trade Winds: Figure 10.10), modified by seasonal westerlies in North Africa and trans-Atlantic;
- the westerlies from the Middle East to East Asia (Figure 10.11);
- the westerly system across North America, South America and Australasia.

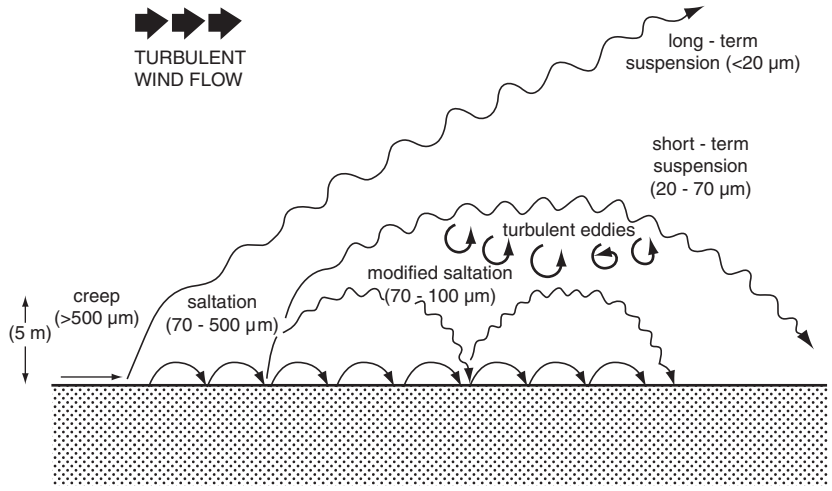

Figure 10.9 Modes of particle transport by the wind. The indicated particle-size ranges in different transport modes are those typically found during moderate wind storms [$\varepsilon = 10^4$–10^5 cm^2/s^1] (After Pye, 1987)

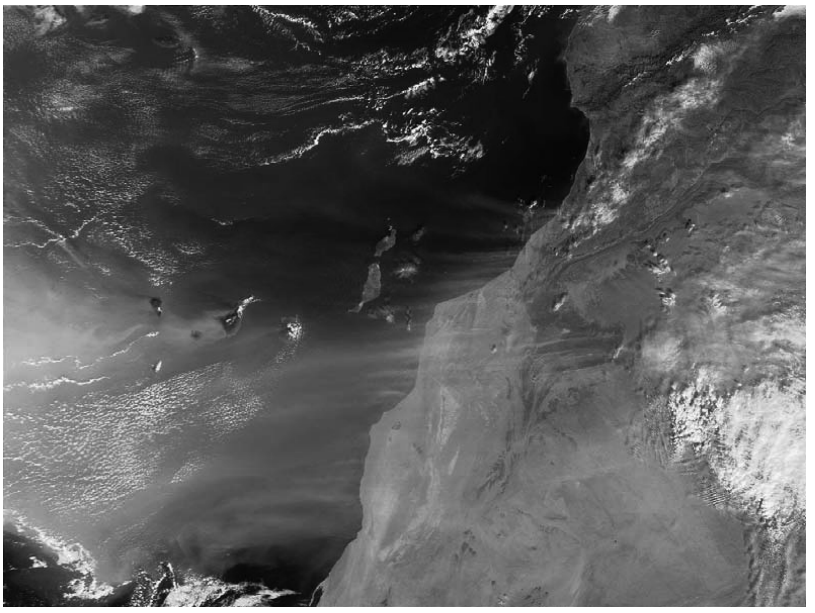

Figure 10.10 Easterly pall of Saharan dust over north-west Africa (southern Morocco, western Algeria, Western Sahara and Mauritania), covering the Canary island archipelago and extending into the eastern Atlantic and beyond in the northern winter (January, 2002) (Reproduced by kind permission of NASA)

In the planet's two most abundant dust-source regions (North Africa and Central and East Asia), dust pathways and deposition are influenced by distinctive and seasonally variable meteorological factors, including the subtropical trade winds, the monsoons and the westerlies. It should also be noted that aeolian dust is a vital source of ocean ecosystem nutrition. The dominant input of iron to oceanic life, for example, is supplied by wind-borne dust from the world's drylands

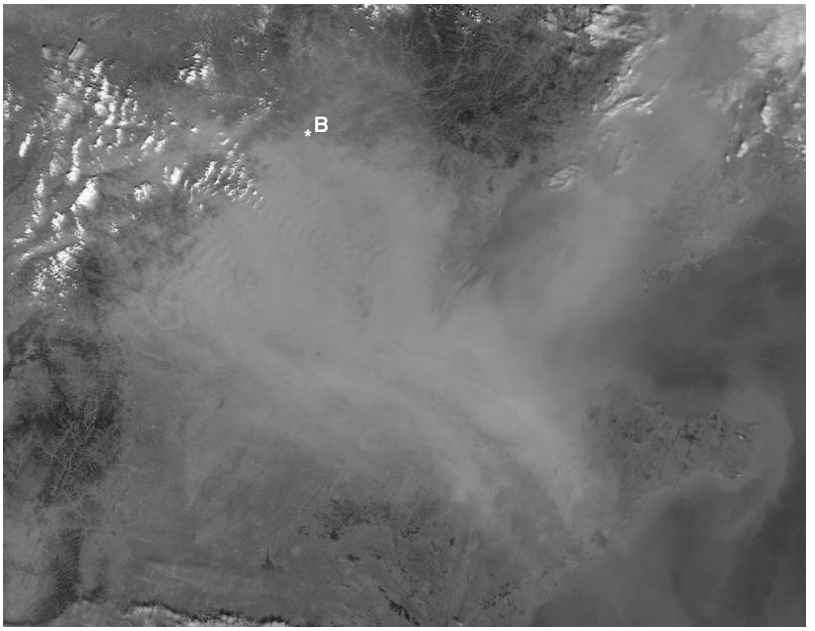

Figure 10.11 Dense outbreak of dust from the drylands of Mongolia and northern China in the northern winter–spring transition period (March 2009). Westerly airflow carries dust eastwards over the north China plain (*B = Beijing) including the Shandong Peninsula, the Bohai Gulf and Yellow Sea and the Pacific Ocean (Reproduced by kind permission of NASA)

(Jickells *et al*., 2005). Both African and Asian dust pathways are global in distribution.

Dust transport in central and eastern Asia is influenced by both westerly and Asian monsoon climatic systems. An extensive high-pressure system (the Siberian High) dominates the cold, dense but stable atmospheric conditions in northern Eurasian winters; but between late winter and early summer, the Siberian High progressively diminishes in size and stability as westerly-driven depressions and associated low-pressure troughs track along its receding southern margins. Mineral dust in the lower troposphere, driven by westerly frontal systems, is sourced from mid-latitude drylands in Mongolia and western China. Two major source-pathway systems (Mongolia to North China and Taklamakan Desert to the northern margins of Tibet) drawing dust palls in the lower troposphere to Beijing, Nanjing and beyond have recently been determined using Nd-Sr isotopic composition (Chen *et al*., 2007; Li *et al*., 2009; Figure 10.12).

The dust deposition sequence in East Asia begins on the north and north-west China plateau regions, with the coarsest fractions falling on the loess lands of Gansu, Shaanxi and Shanxi provinces. Progressively finer dust fractions are transported eastward to the densely populated north China plain, Korea and Japan. The city of Xi'An on the southern margin of the Chinese Loess Plateau in Shaanxi province receives dust palls from seven different pathways (Wang *et al*., 2006; Figure 10.13).

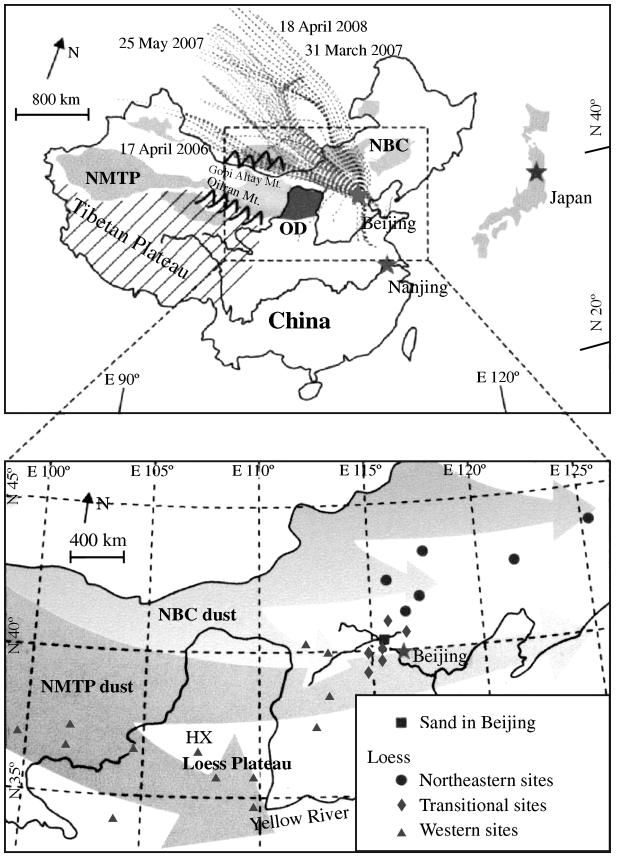

Figure 10.12 Two major source and pathway systems with three distinctive Nd-Sr isotopic signatures across northern and western China. Thirty six hour back trajectories from Beijing were calculated using the HYSPLIT method (Draxler and Rolph, 2003). After Li *et al.* (2007)

Lower-troposphere dust palls usually travel considerable distances beyond China, crossing the Pacific to deposit in North America (McKendry *et al*., 2007). Both lower and upper troposphere dusts from East Asia have been found in the Greenland ice sheet (Bory *et al*., 2003) and the French Alps (Grousset *et al*., 2003). Recently, an upper troposphere dust stream from the Tarim Basin was traced along a $\sim 450^{\circ}$ meridian circuit crossing the eastern Pacific at the 120°E meridian twice, with a dust transport mass flux decay of an order of magnitude from 75 to 8 Gg (gigagrams: Uno *et al*., 2009).

In North Africa, the nature and extent of pressure cells over the region vary with the season. Winter outbreaks of Saharan dust (February–March) are associated with the seasonal high pressure system over North Africa and its characteristic mobility, in which major cells can be found from the eastern Atlantic to the Mediterranean basin. This circulation pattern draws prolonged, high concentrations of mineral dust into the lower troposphere from the Sahelian zone south of the Sahara. Outbreaks during summer (June–August) include dust palls of low frequency and density ($\sim$0.75 mg/m^3 of PM_{10}) but high persistence (15–30 days). The thermal low pressure that characterises the Sahara in summer shifts the anticyclone above the 850 hPa isobar level (approximately 1500 m a.s.l.), with the result that summer outbreaks of Saharan dust origin occur at higher tropospheric levels and do not intrude into the oceanic boundary layer. This semi-permanent dust layer (the Saharan Air Layer; Prospero and Carlson, 1972), clearly seen on orbital imagery, sustains an almost continuous westward dust flow across the Atlantic (Figure 10.14). A third, autumn–winter type of outbreak occurs at low frequency in October–November. In this season, the anticyclone is at ground level, the Sahelian surface having being cooled by lower temperatures and more frequent rainfall events, so inhibiting large-scale dust injections. However, locally violent dust storms sometimes raise PM_{10} dust densities of up to 400–500 mg/m^3. In general, these systems undergo dry deposition, but unstable atmospheric conditions involving draw-down of Saharan dust to the surface by intense low-pressure cells offshore of the Western Sahara can lead to wet deposition.

Saharan dust takes about a week to cross the Atlantic Ocean, typically reaching north-eastern South America, including the lower Amazon basin, in the (northern) late winter and spring and the Caribbean, Central America and the south-eastern United States in summer and early autumn (Prospero and Nees, 1986). Depending on season and meteorological conditions, particulate dust advected westwards from the Sahara influences air quality in North America (Prospero, 1999), the Caribbean, South America, Europe, the Middle East and Asia and affects the nutrient dynamics and biogeochemical cycles from northern Europe to South America. It has been estimated that 13 million tonnes of African dust falls on the north Amazon Basin every year (Griffin *et al*., 2001, 2002). On the Caribbean island of Trinidad during

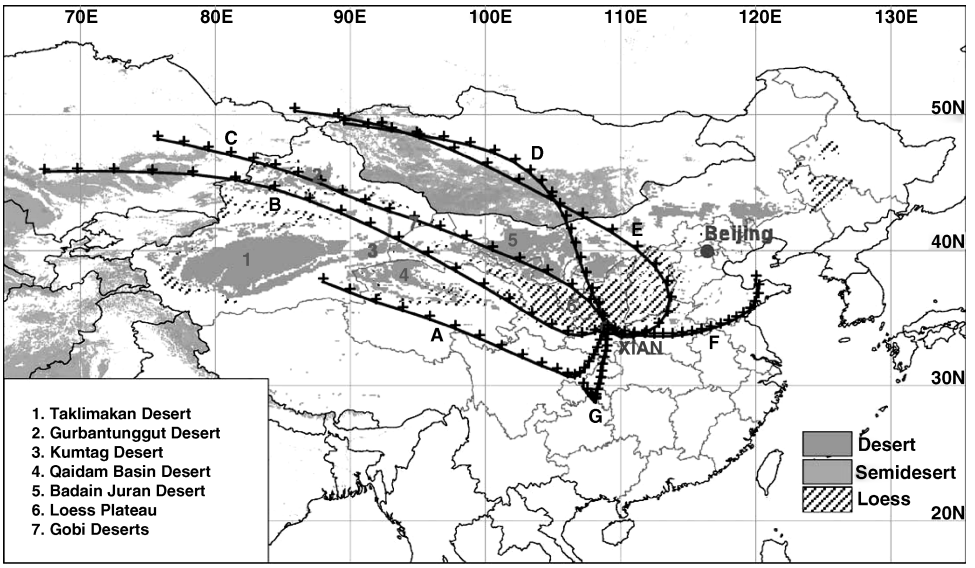

Figure 10.13 Back trajectories (HYSPLIT: Draxler and Rolph, 2003) of pathway clusters of springtime dusts deposited on the city of Xi'An in 2001–2003 (Wang *et al.*, 2006). Pathway clusters A, B and C, traversing the major sources of Asian dust, show mean PM_{10} loadings higher than the three spring months' average (159 $\mu g/m^3$); they account for 8 per cent, 16 per cent and 29 per cent of all trajectories depositing at Xi'An. Clusters D and E (the more northerly sources, centred on the Badain Juran desert) and F are less important routes for transport to Xi'An. No dust sources lie along Cluster G, the deposits being anthropogenic dust only. The major dust sources for Xi'An and Beijing are different, with north-westerly sources more important for Xi'An and arid and semi-arid regions in Mongolia more important for Beijing. PM_{10} loadings in springtime at Xi'An are usually less than those observed in Beijing (194 $\mu g/m^3$)

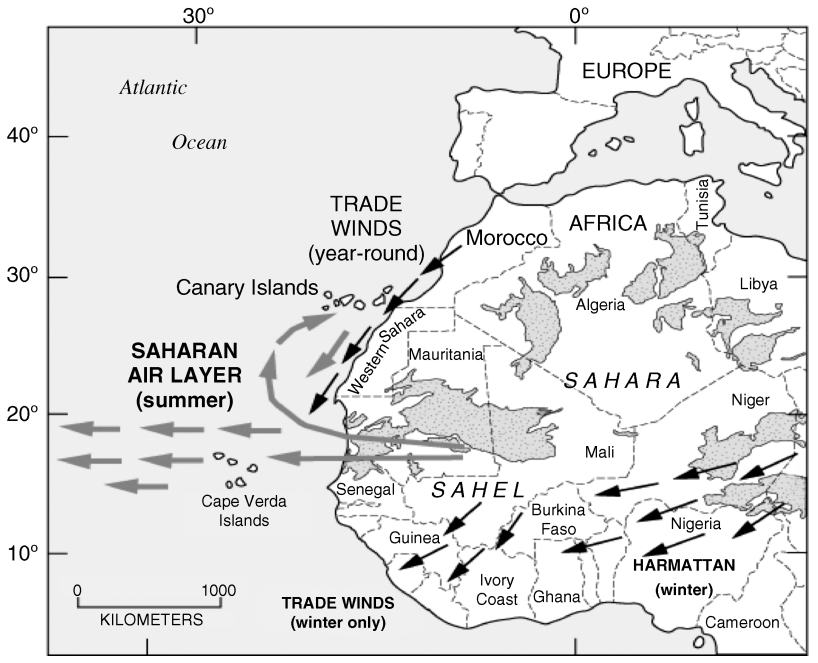

Figure 10.14 Main dust-transporting winds and their transport seasons, over the western half of the Sahara. Grey stippled areas indicate major active sand seas (From US Department of Agriculture, Natural Resources Conservation Service: http://www.soils.usda.gov/use/worldsoils/mapindex/order.html), based on FAO-UNESCO Soil Map of the World (Food and Agriculture Organization of the United Nations, UNESCO, 1974)). After Muhs *et al.* (2010), with kind permission of the first author

influx of Saharan dust, PM_{10} values of 135–149 $\mu g/m^3$ were registered (four times the known values for non-Saharan days), leading to dramatically increased paediatric asthma admissions (Rajkumar and Chang, 2000). At five sites in Spain, it has been shown that the daily mean concentration of $PM_{2.5}$ is doubled (from 4 to 11 $\mu g/m^3$) during influx of Saharan dust (Viana and Averol, 2007).

Isotopes ^{137}Cs and ^{40}K have been successfully used as tracers of PM_{10} in the Marine Boundary Layer offshore of the Sahara on the island of Tenerife (Karlsson *et al*., 2008). In the Mediterranean, North African sub-regional dust sources and inferred pathways have been clearly discriminated using strontium, neodymium and lead isotopic ratios (Grousset and Biscaye, 2005; Figure 10.15)

A recent study has reported detection of north-east African and Middle East dust over Japan, implying trans-Eurasian movement of natural mineral particles from Africa eastward as well as westward. In March 2005, for example, Saharan dust was traced across Asia and the Pacific, reaching the western coast of Canada in just 10 days (McKendry *et al*., 2007). Earlier (March 2003), rainwater samples in Japan contained dust and nanometre-scale particles that differed in both composition and shape from typical Chinese dust. In addition, carbon-bearing nanoparticles in the rainwater were consistent with the crude-oil fires raging in the Middle East at that time (Tanaka *et al*., 2005).

Volcanic plumes can also travel great distances from the source volcano. The global extent depends on the volcano's location (plumes circulate in the hemisphere within which the volcano is located, following high-level wind patterns) and on the volume of erupted material and the height of the plume. On 15 June 1991, a climactic eruption of Pinatubo volcano, Philippines, sent fine-grained ash, gas and aerosol 35 km into the stratosphere, where it then circled the globe at equatorial latitudes in 22 days. Most of the ash mass from eruptions is made up of large particles which rapidly sediment out of the plume, leaving particles with diameters of less than 2 μm in the stratosphere. The ash component rapidly aggregates, however, and most of it falls to earth within 1–2000 km of the source; in the case of Pinatubo, ash fall was recorded as far away as Malaysia and Vietnam (~2500 km). The Pinatubo plume that circulated the globe was, therefore, mainly aerosol composed of sulphuric acid droplets (H_2SO_4) produced from SO_2 in the plume which condensed on to nuclei such as existing sulphuric acid particles and remaining ash particles. This type of aerosol scatters incoming solar radiation causing cooling effects at the Earth's surface and warming in the stratosphere.

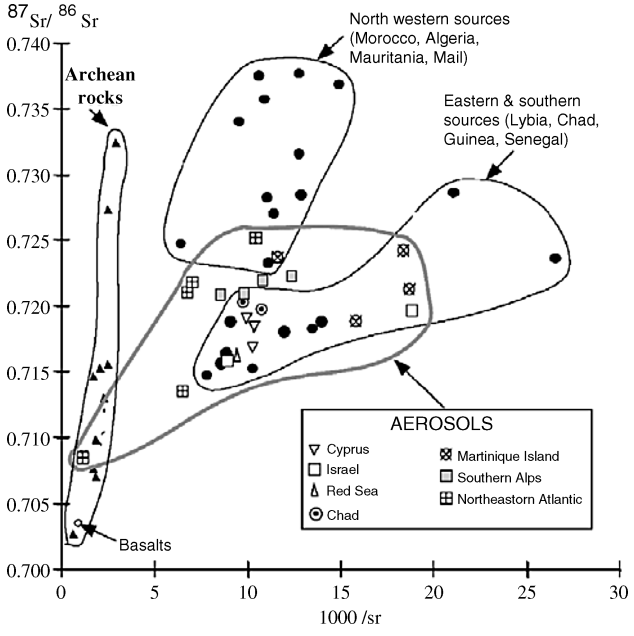

Figure 10.15 Strontium, neodymium and lead isotope ratios of Saharan aerosol dusts and their source regions (After Grousset and Biscaye, 2005)

10.5 Health effects of inhaled particulate material

10.5.1 Asbestos

Asbestos has been of practical use to mankind for over 4,000 years. The special properties of asbestos were recognised in Finland 4500 years ago, where they incorporated anthophyllite fibres into clay pots, presumably to give the vessels added strength (see Gibbs, 1996). The Greeks have used asbestos since the first century BC; the word asbestos is derived from the Greek, 'unquenchable' or 'inextinguishable', reflecting its durability under extreme conditions. Industrial mining of asbestos began in the 1850s and, as large deposits of asbestos were discovered in Canada, Russia and South Africa and as novel asbestos products began to be developed, the industry grew rapidly in the latter half of the nineteenth century. It was at the turn of the nineteenth century and the beginning of the twentieth century that the potential health effects of inhaled particulate material, including asbestos, became apparent. Those most at risk have been, or are, involved in mining of the material as well as being exposed during production, repair and destruction of asbestos-containing materials; the greatest human health risk is to the lungs by way of inhalation.

In 1907, Murray was the first to report a case of asbestosis and a detailed description was later provided by Cooke (1924) who coined the term asbestosis. Merewether (1930) addressed the hazards of working with asbestos and made recommendations on exposure. Soon afterwards, Gloyne (1935) noted a relationship between asbestosis and bronchogenic carcinoma, which was subsequently confirmed (Merewether, 1949; Doll, 1955) and the relationship between exposure to low levels of asbestos and a rare, untreatable form of lung cancer, malignant mesothelioma, was first described by Wagner *et al*. (1960). Asbestos exposure has devastating consequences on the respiratory system, causing pulmonary fibrosis, bronchogenic lung cancer, mesothelioma

and pleural plaques. Although legislation to limit exposure was first introduced in Britain in the 1930s, following the report of Merewether and Price, legislation was not introduced into the USA until the 1970s. The exceptional qualities and versatility of asbestos meant that it was widely and increasingly, used for many more years before regulatory control was introduced in the UK in the 1980s (Asbestos Licensing Regulations, 1983; Control of Asbestos at Work Regulations, 1987) and, ultimately, importation to the UK was prohibited in 1992 (Asbestos Prohibition Regulation, 1992; The Control of Asbestos at Work (Amended) Regulations Act, 1992). Owing to the long latent period of asbestos-related diseases (some 30–40 years), the incidence of diffuse malignant mesothelioma, which has the longest latency, is predicted to reach its peak in Europe in 2020 (Peto *et al*., 1999). Diagnosis of asbestosis continues to rise and, consequently, the deaths from these untreatable diseases are significant.

The nature of the disease depends, at least in part, on the type of asbestos to which individuals are exposed (Berman and Crump, 2008). There are two main types of asbestos, which have distinct physical differences: serpentine (chrysotile) and the amphiboles (crocidolite, amosite, anthophyllite, tremolite, actinolyte) (Table 10.2). The amphiboles are straight and needle-like, or form flakes. These fibres tend to retain their structure within the lung tissue; their needle-like shape and biopersistance is believed to be an important factor in their pathogenicity, penetrating parenchymal lung tissue and accessing the pleural surface (Donaldson, 2009). In contrast, chrysotile asbestos is made up of hundreds of small fibrils that form fibres, which are flexible and fray at the ends. These fibres can wrap around the lung structures, depending on the site of deposition; they are less durable than the amphiboles. The deposition and retention of asbestos in the lung critically depends on fibre size and physicochemical format. Long fibres are trapped in the large airways, where they may be phagocytosed by macrophages or trapped in the mucus and transported to the throat where they will be swallowed or expectorated. Very long particles are sometimes engulfed by many macrophages (frustrated phagocytosis) and develop into asbestos bodies (Figure 10.16), readily seen by light microscopy. These usually contain amphibole asbestos and high levels of asbestos bodies in lung secretions obtained by bronchoalveolar lavage indicate high tissue levels. Smaller, respirable particles less than 10 µm aerodynamic diameter, will reach the peripheral, respiratory zone. Macrophages will phagocytose single particles and particle aggregates in the micron range and will clear the particles from the lung via the mucociliary escalator or the lymphatic system. Particles that escape phagocytosis and the normal clearance mechanisms of the lung will access, and cause injury to, the lung tissue.

The Great Britain Asbestos Survey of 98 117 asbestos workers (Harding *et al*., 2009) confirmed the known association between exposure to asbestos, asbestos-related lung diseases and the high mortality rates due to asbestosis, lung cancer (e.g. bronchogenic carcinoma), as well as pleural and peritoneal cancers and malignant mesothelioma. Pleural plaques are also common in asbestos workers but are not in themselves a cause

Table 10.2 Chemical formulae of asbestos and some asbestiform minerals. There are many other fibrous zeolites, such as mesolite, mordenite, natrolite, paranatrolite, tetranatrolite, scolecite and thomsonite

Name	Other names	Mineral type	Formula
Asbestos minerals			
Chrysotile	White asbestos;	Serpentine	$Mg_3(Si_2O_5)(OH)_4$
Amosite	Brown asbestos; cummingtonite-grunerite (solid solution series)	Amphibole	$Fe_7Si_8O_{22}(OH)_2$
Crocidolite	Blue asbestos; fibrous riebeckite	Amphibole	$Na_2Fe^{2+}{}_3Fe^{3+}{}_2Si_8O_{22}(OH)_2$
Tremolite		Amphibole	$Ca_2Mg_5Si_8O_{22}(OH)_2$
Anthophyllite		Amphibole	$(Mg,Fe)_7Si_8O_{22}(OH)_2$
Actinolite		Amphibole	$Ca_2(Mg,Fe)_5(Si_8O_{22})(OH)_2$
Some other asbestiform minerals			
Erionite		Zeolite	$(Na_2,K_2,Ca)_2Al_4Si_{14}O_{36}{\cdot}15H_2O$
Fluoro-edenite		Edenite	$NaCa_2Mg_5(Si_7Al)O_{22}F_2$
Richterite		Amphibole	$Na(Ca,Na)(Mg,Fe^{2+})_5(Si_8O_{22})(OH)_2$
Winchite		Amphibole	$(Ca,Na)Mg_4(Al,Fe^{3+})(Si_8O_{22})(OH)_2$
Balangeroite		Serpentine	$(Mg,Fe^{2+},Fe^{3+},Mn^{2+})_{42}Si_{16}O_{54}(OH)_{36}$
Carlosturanite		Serptentine-like	$(Mg,Fe,Ti)_{21}(Si,Al)_{12}O_{28}(OH)_{34}$
Nemalite	Fibrous brucite	Magnesium hydroxide	$Mg(OH)_2$
Palygorskite	Attapulgite	Clay	$(Mg,Al)_2Si_4O_{10}(OH)^{\bullet}4(H_2O)$
Wollastonite		Pyroxenoid	$CaSiO_3$
Sepiolite		Clay	$Mg_4Si_6O_{15}(OH)_2{\cdot}6H_2O$

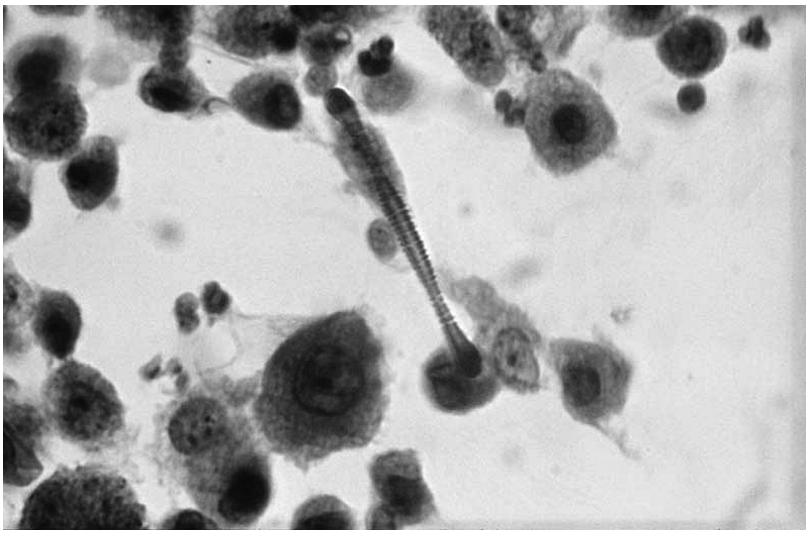

Figure 10.16 Photomicrograph of an asbestos body recovered during diagnostic bronchoscopy. Asbestos bodies consist of a central core fibre of asbestos, coated with a layer of iron-protein-polysaccharide, giving this beaded, dumb bell appearance. The fibre is surrounded by alveolar macrophages and other inflammatory cells, which were recovered during bronchoalveolar lavage (Courtesy of Professor T. D. Tetley)

of mortality (Weiss, 1993). Those who work in the insulation industry were found to be almost 7 times more likely to die from asbestos exposure than those who were in manufacturing; those involved with asbestos removal, a more recent occupation, were also found to be more likely to die than those in the manufacture of asbestos (Harding *et al*., 2009; Antonescu-Turcu and Shapira, 2010). There is evidence to suggest that smoking is an important additional risk factor in cases of asbestos-related lung cancer and asbestosis, but not for mesothelioma (Harding *et al*., 2009; Henderson *et al*., 2004). Smoking has been described as being both additive and synergistic, but its influence probably reflects the complexity of the combination of differing pathogenicity of the asbestos, the type of cigarette smoked, genetic background and other environmental factors. The most recent report on British asbestos workers, the largest survey of its kind, shows a synergistic effect (Harding *et al*., 2009). Churg and Stevens (1995) suggest that smoking delays clearance of the asbestos fibres from the lung, increasing retention within the airway mucosa and contributing to airways obstruction; increased accumulation of asbestos fibres is also likely to enhance the possibility of carcinogenic changes in the lung.

All types of lung cancer have been related to asbestos exposure, including small-cell and non-small-cell carcinomas. As mentioned above, the effect of concurrent smoking has been shown to be synergistic, with a significant, manifold increase in development of lung cancer. Since it is not possible to distinguish tumours related to asbestos exposure from those induced by tobacco smoke, well-controlled epidemiological studies have been essential in establishing synergy (Henderson *et al*., 2004). It is suggested that cigarette smoke acts as a tumour promoter (Mossman and Gee, 2010) or co-carcinogen. There is also some debate regarding an association between asbestosis and the incidence of lung cancer. This is further complicated by the difference in relative potency of chrysotile and amphibole asbestos: both can induce lung cancer, but amphibole asbestos has at least six times greater carcinogenic potential (Berman and Crump, 2008). Another important factor is fibre length; the presence in lung tissue of asbestos fibres of 10 μm and above (but not shorter fibres) have consistently been associated with development of lung cancer.

Asbestosis causes an interstitial pneumonitis and fibrosis as a result of fibre inhalation and deposition within the lung (Antonescu-Turcu and Shapira, 2010; Becklake *et al*., 2007; Gibbs, 1996). The condition develops over many years and initially there are few symptoms; it will be diagnosed only some 20–30 years following the peak exposure, the risk and incidence relating to the cumulative dose of asbestos exposure and the time since the first exposure. Patients present with breathlessness and dry cough, reduced gas diffusion capacity and oxygen desaturation on exertion. Lung function in advanced disease shows restrictive disease and reduced total lung capacity and vital capacity. Lung pathology shows usual interstitial pneumonia; high-resolution tomography shows significant remodelling of the parenchyma and honeycombing in advanced disease. There is no treatment for asbestosis; the condition gets progressively worse, even after exposure has ceased.

Malignant mesothelioma is an aggressive tumour of the pleura with a prognosis of only 6–18 months following diagnosis, depending on the cellular origin of the tumour (Churg, 1998). Again, there is a long latency between exposure and symptoms, up to 50–60 years following exposure and, unlike asbestosis, exposure to very low levels of amphibole asbestos can induce mesothelioma. Thus, exposure to low levels of amosite or crocidolite (but not chrysotile) induces mesothelioma. Fibres above 10 μm are associated with mesothelioma, but short fibres are not (Berman and Crump, 2008). A complication has

been that samples of chrysotile asbestos may be contaminated with amphiboles or even erionite, a potent inducer of mesothelioma. Erionite-induced mesothelioma was thought to be confined to a small region of Turkey, but cases have recently been described in the United States (Dogan *et al*., 2008; Kliment *et al*., 2009). Unlike bronchogenic cancer and asbestosis, smoking is not a risk factor (Harding *et al*., 2009). It is difficult to diagnose the condition, as the symptoms are nonspecific. The patients report breathlessness, cough and chest pain, possibly associated with weight loss. The tumour can be seen as dense white tissue encasing the lung. Over time, it penetrates the respiratory tissue, destroying the pulmonary architecture and may impact on other structures such as the oesophagus and superior vena cava. Because of the ubiquitous, aggressive nature of the tumour and late diagnosis, there is no effective treatment.

10.5.2 Silica

Since silica is one of the most common constituents of the Earth's crust, people have always been at risk of exposure to pathologically significant levels, particularly in an occupational setting. Silica is derived from the Latin *silex,* which describes a flint, a stone tool containing silica. Pneumoconiosis, including silicosis, has been shown in Egyptian mummies, in sixteenth-century Bohemian miners and in eighteenth-century stone cutters (see Gibbs, 1996). The industrial revolution in Britain and increased, unanticipated occupational diseases, led to serious study of conditions related to occupation, including those generating a dusty atmosphere. Thus, in 1832 Thackrah noted that sandstone workers died prematurely, usually before 40 years of age (Thackrah, 1832). Peacock (1860) and Greenhow (1865) reported silica in the lungs of stone workers. The first to use the term silicosis was Visconti, in 1870 (Rovida, 1871); silicosis was previously known as miner's phthisis, grinder's asthma, potter's rot and other terms relating to occupation. It was often confused with tuberculosis (TB; also termed phthisis), until the TB bacillus was discovered by Koch in 1882.

Numerous reports in the early twentieth century correlated high rates of silicosis amongst South African gold miners, as well as in miners of tin, metal, slate, sandstone and quartz in the United Kingdom and Australia, with exposure to silica dust. These studies were the first to propose that crystalline silica (rather than amorphous silica) was responsible (Lanza, 1938). The early twentieth century also saw the introduction of sandblasting and the use of abrasive mechanical equipment to produce fine particulate silica dust, often without adequate protection. Of particular relevance was the Hawk's Nest Tunnel disaster at Gauley Bridge, West Virginia, in the 1930s (Madl *et al*., 2008). The project entailed blasting large natural rock formations to construct a source of hydroelectric power. Many of the workers died of respiratory disease during the project (called 'tunnelitis') and an epidemic of silicosis occurred subsequently. It was discovered that the rock consisted largely of pure quartz which, when processed, resulted in prolonged exposure to high concentrations of pure silica dust. Consequently, the Air Hygiene Foundation of America Inc. was founded and the first occupational exposure limits (OEL) were established. Regulations have continued to be tightly controlled; nevertheless, respiratory disease related to silica inhalation still occurs, particularly in underdeveloped countries.

There is a wide range of industries in which there may be exposure to silica, including mining and milling of metals, iron and steel foundries, quarrying of granite, stone and slate and the glass, pottery and ceramics industries. The construction industry, shipbuilding and maintenance and some agricultural activities are significant activities that involve contact with silica dust at some stage. Consequently, respiratory morbidity and mortality due to inhalation of silica continues, albeit at low levels (Madl *et al*., 2008). There are now fewer than 30 deaths per year in the UK (Health and Safety Executive UK) and 200–300 deaths per year in the USA (American College of Occupational and Environmental Medicine, 2005).

Silica exists in a number of forms, which have different pathogenicity. Whilst the non-crystalline forms (e.g. diatomaceous earth (diatomite), fume silica, opal and mineral wool) are not considered to be pathogenic, the crystalline forms are. Quartz, cristobalite, moganite, some forms of tridymite, melanphlogite, coesite and stishovite are all crystalline forms of silica; quartz is by far the most common. Piezoelectric properties, where the silica exhibits opposite electrical charges at opposite ends of the crystal under pressure, are believed to contribute to their pathogenicity, particularly that of quartz (Greenberg *et al*., 2007; Rimal *et al*., 2005). Furthermore, toxicity is greatest when the particles have been freshly generated, as the resulting increase in redox potential at the surface favours strong reactions with hydrogen, oxygen, carbon and nitrogen. Oxygen free radicals at the surface of freshly mined silica and stimulation of cellular oxidative stress have been implicated in the cellular damage and lung injury that occurs in affected individuals. These processes are discussed in more detail in Section 10.2.

Chronic (nodular, classic) silicosis is the most common of the respiratory conditions caused by exposure to silica. It usually occurs after 20 or more years of exposure to relatively low concentrations of silica; some cases may occur at 5 or 10 years, described as accelerated silicosis, due to exposure to higher concentrations of often freshly generated silica. The lungs contain numerous, firm nodules, which vary in colour depending on the type of silica exposure. The nodules may vary from a few millimetres in diameter to larger regions of fused nodules in the upper lobes of the lung. In progressive massive fibrosis (PMF), a subset of chronic silicosis, there is marked conglomeration of nodules to create a large, hard fibrotic mass, most commonly observed bilaterally in the upper lobes (Gibbs, 1996; Greenberg *et al*., 2007; Rimal *et al*., 2005). The nodules contain silica crystals and many have an area of necrosis centrally, due to inadequate blood supply, surrounded by whorls of fibroblasts and peripheral, silica-laden macrophages and other inflammatory cells, including lymphocytes. In PMF, the fibrotic masses may show cavitation due to necrosis.

Clinically, the diagnosis is based on the patient's history of exposure and the presence of nodules on chest radiographs or, preferably, by computerised tomography. Classically, the nodules are approximately one centimetre in diameter; the patients have relatively few symptoms, although there is some evidence of airflow obstruction and cough, sputum production and breathlessness. These may be symptoms of associated conditions, such as bronchitis. In contrast, complicated silicosis, with PMF, shows the larger fibrotic regions and sometimes basilar emphysema, on radiography and computerised tomography. These patients are often hypoxic at rest and may have mycobacterial infections such as tuberculosis. They are breathless on exertion and exhibit both restrictive and obstructive lung disease, as well as decreased diffusing capacity. They also develop spontaneous pneumothoraces and die of respiratory failure. Those with accelerated silicosis present with the same symptoms as those with chronic silicosis and may rapidly progress to complicated silicosis, but are also likely to exhibit a variety of autoimmune disorders.

Acute silicosis most often occurs when the silica dust is extremely fine and affects occupations involving sandblasting and rock drilling. It develops rapidly over 4–5 years and may be fatal. It is very different from chronic silicosis and is also referred to as silicoproteinosis. It is rare, but was prevalent in the Hawk's Nest disaster. Individuals may present with breathlessness, fever, pleuritic pain and weight loss. It is important to diagnose quickly as the condition can progress very rapidly to respiratory failure and death. The airspaces become filled with fluid containing eosinophils and enriched with lipids and proteins. This partly reflects increase in numbers of alveolar epithelial type 2 cells, which line the respiratory units and secrete large amounts of proteins and lipids, being the source of pulmonary surfactant which lubricates and protects the respiratory units under normal conditions, but which is significantly elevated in acute silicosis.

The relationship between silica exposure and cancer has proved difficult to establish conclusively. In 1997, the International Agency for Research on Cancer (IARC) rated silica as a Group 1 carcinogen (with 'sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite') (International Agency for Research on Cancer, 1997). However, this has been contested by others (Madl *et al*., 2008; Hessel *et al*., 2000) who used many of the same studies together with studies that took place subsequently, but applied alternative statistical analysis (Hessel *et al*., 2000) which did not show an association between exposure to silica and cancer. One problem has been separating silica exposure and silicosis from exposure to other known carcinogens, including cigarette smoke (Madl *et al*., 2008; American College of Occupational and Environmental Medicine, 2005). Other factors include differences in the socioeconomic status of the subjects and whether the control groups were appropriate. Thus, the risk of developing cancer increases in those with silicosis who smoke (American College of Occupational and Environmental Medicine, 2005), but evidence of a risk of cancer in those without silicosis is inconsistent (Ulm *et al*., 1999; Checkoway and Franzblau, 2000). It has been suggested that the relationship between exposure to silica, silicosis and lung cancer may never be resolved (Madl *et al*., 2008).

As alluded to previously, a number of recent studies suggest that occupational exposure to silica may be associated with autoimmune diseases, such as rheumatoid arthritis, scleroderma, progressive systemic sclerosis and lupus erythematosis, as well as kidney disease. However, it is difficult to know the exact risks since these diseases are also dependent on other environmental and genetic factors, as well as being more common in women, so that epidemiological investigations are not easy to control. The evidence has been interrogated (American Thoracic Society Committee of the Scientific Assembly on Environmental and Occupational Health, 1997; NIOSH, 2002) and the conclusion is that there is not enough evidence and more research is needed before reliable conclusions can be drawn. A similar situation exists in relation to silica exposure and kidney disease (NIOSH, 2002).

10.5.3 Coal

Coal has been mined in the UK for over a thousand years. The first mention of coal was in 852, in the Saxon Chronicle of Peterborough (Gibbs, 1996). During the nineteenth century and the industrial revolution, coal became an important source of energy. Coal-workers' pneumoconiosis (CWP) was first described by Laennec (1819). The term anthracosis was introduced (Stratton, 1838) to describe the blackened lungs of coal miners from Fifeshire, Scotland. A particularly high incidence of CWP was found in miners in South Wales, which led to a survey, set up by the Medical Research Council, leading to a series of reports in the early 1940s (Gibbs, 1996). These were followed up by further investigations by the MRC and the National Coal Board, which culminated in setting standards for dust exposure (Hurley *et al*., 1982). NIOSH have more recently recommended levels of exposure aimed at preventing or, at the least, limiting CWP (NIOSH, 1995). During the latter half of the twentieth century, these controls, together with a decline in coal mining, have resulted in a significant reduction in the incidence of CWP in Europe and North America. However, coal production is increasing in developing countries, where it is an important source of energy and contributes to the economy of the country. In China, coal is used to generate approximately 70 per cent of the electricity (Liu *et al*., 2009) and the incidence of CWP is high. The predicted increase in mining and use of coal in Asia and Africa is causing concern that there will be a parallel increase in respiratory health problems (McCunney *et al*., 2009).

CWP was originally believed to be a form of silicosis, but most coal has relatively little free crystalline quartz and there is a poor correlation between quartz content and CWP (McCunney *et al*., 2009). Thus, the incidence of CWP was found to be low in miners who mined coal with a comparatively high level of silica, whilst there was a high incidence of CWP in collieries with comparatively low levels of silica. A series of epidemiological studies in Britain during the 1970s and 1980s demonstrated that development of CWP was related to exposure to coal dust but did

not correlate with the silica content (McCunney *et al.*, 2009; Hurley *et al.*, 1982; Maclaren *et al.*, 1989). Similar findings were found in studies from Europe. Furthermore, although the diseases may have similar radiographic patterns, they are pathologically very different. It is not entirely clear exactly what properties in coal cause CWP. It is well known that the rank of the coal – low, medium or high, reflecting the carbon content – is important. High rank coal is the hardest and oldest, containing the most carbon, the least volatile matter and least silica; it is retained more readily in the lung and is associated with severe CWP. When the incidence of CWP was tracked against the rank of the coal, CWP was found to be 6- to 10-fold higher in those exposed to high-rank coal (>90 per cent carbon) than in those exposed to the low-ranked coal (<80 per cent carbon) (Attfield and Morring, 1992). Furthermore, the amount of bioavailable iron has also been related to development of CWP (Zhang and Huang, 2002). There is no evidence that coal dust induces cancer.

CWP is largely due to inhalation of coal dust during the process of mining. The disease is described as simple CWP (SCWP) or complicated CWP (CCWP), depending on the pathology and severity (Gibbs, 1996; McCunney *et al.*, 2009; Ross and Murray, 2004). As mentioned earlier, anthracosis is the accumulation of the coal pigment/particles in the lung; this is asymptomatic. When the lung becomes overloaded with coal-dust particles, the defence mechanisms are overwhelmed. The phagocytic macrophages are the first line of defence, internalising coal-dust particles and migrating away from the airways to the throat where they are swallowed or expectorated, or to the lymphatic system to remove excessive material. During heavy or chronic exposure, macrophage migration and particle clearance is reduced so that there is an accumulation of macrophages in hot spots of particle deposition in the peripheral respiratory zone; there is relatively little collagen deposition, unlike that seen in silicosis and asbestosis. These focal areas of macrophages and particles are known as coal macules, which are defined as being less than a centimetre in diameter and are a characteristic of the disease. Macules are mostly situated in the upper regions of the lung and many may not be observed radiologically. Accumulation of coal dust and activation of macrophages leads to tissue injury and, consequently, fibrosis in an attempt to repair the damage. Thus, fibrotic nodules may also be present in SCWP, which are larger and far fewer in number than the macules. If the process of nodule enlargement continues and there is fusion of nodules to create fibrotic lesions many centimetres in size, CCWP or PMF occurs; this may involve a whole lobe, or many lobes, of the lung. Emphysema, which is a loss of lung tissue showing enlarged airspaces associated with reduced surface area and elasticity, is often present in the lung tissue surrounding the macules, nodules and fibrotic regions. Emphysema is a component of chronic obstructive pulmonary disease (COPD), a disease that affects approximately 15 per cent of tobacco smokers, but which the evidence suggests can also be one of the effects of exposure to coal dust (Coggon and Newman, 1998; Ross and Murray, 2004).

The diagnosis of CWP is based on history of coal-dust exposure and radiology in the first instance, but the macules and nodules cannot always be visualised so that computed tomography scanning may be performed. SCWP is largely asymptomatic, showing only small changes, if any, in lung function. In those with CCWP who present with cough and breathlessness, the ventilatory capacity is reduced in proportion to the presence of diseased tissue, as is the gas exchange capacity and increasing hypoxemia. There is also evidence that coal miners have symptoms of chronic bronchitis, chronic airflow limitation and emphysema, all features of chronic obstructive pulmonary disease (Coggon and Newman, 1998). The severity of CWP is related to the duration and intensity of exposure.

Once the disease has progressed to PMF it continues to progress regardless of removal from the coal-dust stimulus. Thus, if SCWP has been diagnosed, it is suggested that the individual changes jobs to an environment where there is low, or no exposure to avoid progression to CCWP. As smoking has an additive effect, this is to be discouraged. Depending on the degree of breathlessness and hypoxemia, the patient could have oxygen therapy.

10.5.4 Bioaccessibility and bioavailability of inhaled particulate material

The bioaccessibility and bioavailability of inhaled particles depends on numerous factors. Bioavailability refers to the difference between the amount of airborne particulate matter to which one is exposed and the actual amount that the lung retains. Bioaccessibility refers to subsequent events whereby the particles are processed by body fluids and cells to facilitate absorption by, and assimilation into, the lung. Thus, on exposure to airborne particulate matter, particle size and shape, chemistry, crystallinity, surface properties, biopersistence and durability are all important, impacting on the particle burden and reactivity within the lung and development of lung disease.

10.5.4.1 Particle deposition and retention

Deposition of inhaled particles is determined by their density, as well as their size and aspect ratio, which dictate their aerodynamic diameter. The smaller the aerodynamic diameter and/or density, the greater is the likelihood of deposition in the distal, respiratory region of the lung. In addition, the anatomy of the lung favours deposition under gravity or impaction of particles greater than 5 μm in the large, conducting airways; in contrast, a high proportion of particles smaller than 5 μm can reach the respiratory units and may be deposited on the alveolar epithelium. However, very long fibres with high aspect ratios may be deposited in the deep lung.

The ability to clear deposited particles depends on both physicochemical and physiological mechanisms. Large particles which deposit in the conducting airways are cleared by way of the mucociliary escalator to the throat, where they may be swallowed or expectorated. In addition, airway macrophages will phagocytose particulate material (optimally <5 μm), which

will then be cleared by the same route. However, long fibres greater than the diameter of the macrophage (>20 μm), cause 'frustrated phagocytosis', involving the efforts of numerous macrophages to internalise the fibre. These chronically-activated macrophages contribute to generation of reactive oxygen species and other mediators of inflammation (Oberdorster, 2010).

Clearance of particles from the distal, epithelial air-liquid interface of the respiratory region of the lung will also involve macrophage phagocytosis and clearance by the mucociliary escalator. In addition, particles may translocate into the interstitium, due to physical breaching of the alveolar barrier or as a result of active epithelial uptake processes (e.g. endocytosis). Alternatively, particulate material may pass across the epithelial barrier paracellularly, following the flow of water under negative interstitial water pressure. Interstitial particles can enter the lymphatic system under negative pressure and translocate to the pleural surface or to the tracheobronchial lymph nodes, where they may accumulate (Dodson *et al*., 1991; Miserocchi *et al*., 2008). The difference between the amount of particulate material that is deposited and the amount that is cleared is the particle retention, which accounts for the lung burden.

10.5.4.2 Interaction with lung-lining fluid

Another significant factor in the bioaccessibility and biopersistence of inhaled particles is their reaction with components of lung-lining fluid. Mineral particles, such as asbestos and metal oxides, were shown to adsorb proteins from serum many years ago (Desai and Richards, 1978); the amount and nature of protein adsorption depends on the surface chemistry and surface area of the particles, as well as the flexibility of the protein. Protein adsorption by nano-sized particles has recently been described as forming a 'corona', where strong protein binding forms a 'hard corona' (Lundqvist *et al*., 2008). Again, the surface properties (i.e. charge), size and surface area of the particles are important. It is interesting that, to date, there appears to be no predictable pattern or profile of serum protein adsorption based on nanoparticle physicochemistry. Previous studies of ambient environmental particulate matter have shown agglomeration of particles which deposit in human lung-lining liquid due, in part, to alveolar epithelial cell-derived surfactant protein D (SPD) (Kendall *et al*., 2004, 2002) and very precise interactions with macrophages to induce phagocytosis. In addition, surfactant protein A (SPA) adsorbed to inhaled particles may enhance epithelial uptake and translocation of particles less than 1 μm in diameter that deposit at the epithelial surface (Kemp *et al*., 2008) and which escape macrophage phagocytosis. Dipalmitylphosphatidylcholine, a major component of lung surfactant, which has high surface reactivity, also binds to inhaled ambient particulate matter (Kendall, 2007); it is believed to be more likely to enhance particle dispersion rather than agglomeration. Thus, adsorption of components of lung-lining liquid may have protective or detrimental effects on particle reactivity, cellular uptake, translation into the pulmonary interstitium and retention in the lung.

10.5.4.3 Asbestos

There are marked differences in lung retention between serpentine (chrysotile) and amphibole asbestos (Churg and Wright, 1994; McDonald, 1998; Bernstein and Hoskins, 2006). Amphibole asbestos is found in the lung in greater quantities than chrysotile asbestos. Particle retention will reflect a mixture of the dose inhaled and rate of clearance from the lung. Clearance is compromised when there is an exposure overload, which, in experimental animals, occurs when a single bolus of a high concentration is introduced into the airways. It is not clear how relevant particle overload is to human exposure. Nevertheless, inhalation studies in experimental animals (i.e. not involving particle overload) show that, for the same exposure dose, chrysotile is cleared from the lung more effectively than amphibole asbestos, the former being cleared in a matter of days ($T_{1/2} + 0.3$–11 days) whereas the latter takes years ($T_{1/2} > 500$ days). Studies in people are less exact, but show that retention of amphibole asbestos, particularly fibres with a high aspect ratio and a longest diameter greater than 20 μm long, is significantly greater than chrysotile asbestos (Bernstein and Hoskins, 2006; Churg and Wright, 1994; McDonald 1998), even when the amphibole asbestos (e.g. tremolite: McDonald and McDonald, 1997) is a small impurity in a predominantly chrysotile exposure dose. Regardless of the significant difference in the exact amounts of chrysotile retained in the lung compared to the amphibole asbestos, there is still a correlation between the concentration of chrysotile and amphibole asbestos with the estimated cumulative dust exposure. It has been suggested that the increased biopersistence (i.e. fibre retention within the lung, despite the lung's physiological clearance mechanisms) and durability (i.e. ability to resist dissolution under a wide range of experimental *in vitro* conditions; Maxim *et al*., 2006; Muhle and Bellmann, 1995) of the long, thin amphibole asbestos fibres may be one reason why these fibres cause mesothelioma.

An important factor in the rapid clearance of chrysotile asbestos from the lung is its solubility. The crystal structure of serpentine consists of layers of octahedrally coordinated magnesium (brucite sheets) and layers of tetrahedrally coordinated silica; these sheets are mismatched so they curl and form scrolls, with the magnesium on the outside (Bernstein and Hoskins, 2006; Fubini, 1997). The structure is susceptible to environmental pH, in that the outer, magnesium, brucite-like sheets dissolve in mildly acid conditions. The silica matrix is susceptible to acid pH, a situation which exists in the phagolysosomal apparatus and in the immediate vicinity of the cell surface membrane of macrophages (Fubini, 1997; Wypych *et al*., 2005). Consequently, regardless of the initial length of fibre, chrysotile deposited at the air–liquid interface of the lung disintegrates into fragments that can readily be internalised and cleared by macrophages and other clearance systems in the lung. If this occurs, it is unlikely to be a significant health hazard. In contrast, amphibole asbestos has chemical resistance similar to that of quartz. The fibres, each of which is a double chain of tetrahedral silicate structures, are weakly associated lengthwise by a variety of ions, depending on the host rock. These ions bond

the fibres together, but it is at these weakly-bonded surfaces that the fibres will break apart to generate fibres with greater aspect ratios than the original and these are highly insoluble at any pH (see Bernstein and Hoskins, 2006) and may retain their structure in the lung even after many decades. This is why amphibole-asbestos fibres are significantly more biopersistent than chrysotile.

10.5.4.4 Silica and coal

Unlike fibrous asbestos minerals, silica and coal are, for the most part, small, respirable particles finer than 5 µm, often as small as 1 µm, which readily access the deep lung during inhalation. Macrophages play an important role in clearance of both silica and coal and are a key factor in controlling the lung burden. However, phagocytosis of particles and induction of macrophage cell death leads to release of the intracellular particles, which are then re-ingested by fresh macrophages. This continuous cycle of particle phagocytosis and macrophage death contributes to inadequate particle clearance and retention in the lung, which, in turn, perpetuates the inflammatory process. However, the reactivity of coal and various types of silica differs according to their crystallinity (silica) and associated contaminating material, such as clay and pyrite (Donaldson and Borm, 1998).

Inhaled silica deposits in the alveolar ducts and bronchiolar region of the deep lung consist of particles that are, on average, 1.5 µm in diameter. Particles which escape removal by macrophage phagocytosis, as described above, interact with and injure the epithelium, when the particles are then translocated into the lymph nodes (Hemenway *et al*., 1990). In the Hemenway study, the clearance of cristobalite was far less than that found for quartz, while the inflammatory response was 30 per cent greater, lasting over several months. This coincided with increased lung hydroxyproline, as an indicator of fibrosis. The variability in the bioreactivity of silica is well documented (Donaldson and Borm, 1998; Fubini, 1997) and relates to its surface properties. In addition, particle surfaces may be modified by components of lung-lining liquid, as described above. Orr and colleagues (Orr *et al*., 2010) recently showed that uptake of nano-sized amorphous silica by macrophages depends on macrophage scavenger receptor A, by way of clathrin-dependent mechanisms and suggested that the physical agglomeration state influenced the mode of cellular trafficking. In this study, the uptake of silica nanoparticles by pulmonary A549 adenocarcinoma cells (often likened to alveolar epithelial type 2 cells) critically depended on protein adsorption (Stayton *et al*., 2009). Proteins adsorbed to the particles very rapidly and cellular uptake was consequently far slower, approximately one third that of the naked particles. Interestingly, the particles were also slowly expelled from the cells, though at a slower rate than uptake, resulting in cellular retention. Such complex interactions, between silica, macrophages and epithelial cells will impact on the overall bioaccessibility of silica within the lung, at the gas-liquid interface and within the interstitium.

Coal dust is less life-threatening than silica. Exposure to moderately high doses of coal dust, with its significant biopersistence in the lung, does not have the same impact on health as exposure to silica. It is interesting that, although there is a massive accumulation of coal dust in the lungs of miners, related to exposure dose, the health effect, in the absence of exposure to tobacco smoke, is lower than that observed with equivalent exposure to silica. Furthermore, although silica is found in coal dust at a level that might be considered to be detrimental to health, the expected pathogenicity is not observed. Within the lung, factors related to particle deposition, uptake and translocation will apply to all inhaled particulate matter, as discussed above. Clearly, there are some important differences between coal dust exposure and that of mineral fibres and silica (and other dusts described in this chapter). Some clues to these differences lie in the finding that water-soluble extracts of coal dust and both low- and high-aluminium clays (kaolin and attapulgite) inhibit the cellular reactivity of quartz (Stone *et al*., 2004; Clouter *et al*., 2001); such events might explain why occupational exposure to coal dust containing less than 20 per cent quartz does not induce classic silicosis. It is not yet clear whether the influence of components of coal dust simply depresses the bioreactivity of silica or whether they influence the bioaccessibilty, relocation and subsequent reactivity of silica.

10.6 Risk reduction and future trends

This chapter has shown that airborne particulate matter includes material from both natural and anthropogenic sources and that the impact on the environment and human health varies in intensity, scale and timing. The hazards of airborne dust have been described and the potential for reduction of the risks of inhaling particles is considered here. The risk takes into consideration the vulnerability and resilience of local populations, the potential economic impact and the likelihood of the hazard occurring.

10.6.1 Natural dust palls

Dust palls arising from natural sources may have local, regional and even global impacts, their periodicity varying with source type and location.

10.6.1.1 Dust storms

A key strategy in risk reduction is control, minimisation or prevention of the hazard. Dust storms of anthropogenic origin are increasing in many parts of the world. Continuing global deforestation and desertification correlate quite closely with increases in the severity and frequency of dust storms. For example, desertification in China increased total desert area by 2 to 7 per cent in the second half of the twentieth century. This resulted in disproportionately large areas of potential

sources of soil dust, which have increased troposphere dust loading in the mid-latitudes and generated 10 to 40 per cent more dust storms. Any reversal of the soil-dust load by re-establishing grassland in the sensitive 200–400 mm annual rainfall belt of north-west China would require regional cooperation (Gong *et al*., 2004) upon which the degree of risk prevention would depend.

Asian dust storms are known to occur at any time of the year but the prime period is spring to early summer. In contrast, dust palls sourced in North Africa occur throughout the year, dust reaching North America and the northern Caribbean in the northern summer and South America and the southern Caribbean in the northern winter. Substantial regional dust palls derived from some of the world's smaller dust source regions may have a lower mean frequency. For example, the major dust storm that originated in Australia's Northern Territory in September 2009 and which extended beyond New Zealand into the Southern Ocean, was described as the largest dust storm event for over 40 years (Figure 10.17).

Such wide-ranging differences in the magnitude and frequency of naturally occurring dust palls have a bearing on human-health impacts. There is a clear link between frequent exposure to high-density airborne particles over several decades and pneumoconiosis and related conditions; this precludes the application of effective risk reduction strategies, a situation made more complex by the distribution of human population density in relation to regional and global dust pathways. For example, up to 50 per cent of the annual dust flux from eastern Asia derived from its two global sources (the Taklamakan Desert in western China and the Mongolia/North China region) is deposited in China, Korea and Japan, which have urban population densities of between 100 and more than 500 people per km^2 (Figure 10.18); some parts of western China have a siliceous pneumoconiosis incidence of 7 per cent, rising to three times that proportion in people over 40 years old (Xu *et al*., 1993). While available research results are overwhelmingly concerned with occupational pneumoconiosis, there is increasing recognition that non-occupational pneumoconiosis is a serious problem in China (Yin, 2005). Regions in the Caribbean and North and South America affected by Saharan dust have moderately dense populations (>40 per km^2 with some regional centres having higher densities of >100 km), but dust palls are more frequent than the mean for eastern Asia. Saharan regional-scale dust reaches moderately dense populations in Europe, but the limited extent, low density and/or low frequency of many of these incursions reduces their potential health impact.

Given that dust storms have the potential to affect large areas, reduction of risk is most likely to be achieved by adopting local mitigation measures such as small-scale forestation projects, long-term use of dust masks and even relocation of populations. Direct action has been undertaken in China, designed to stem the frequency and magnitude of dust storms around dryland margins; trees have been planted, including, most notably, the Green Great Wall initiative. However, judging from evidence of a

Figure 10.17 Dust storm progression eastwards from a central Australian source in 2009. Top: Advancing dust storm front approaching a children's playground in Alice Springs, 22 September 2009. Photograph source: unknown. Bottom: Changing air quality over the city of Brisbane, Queensland, some 2000 km ESE of source. Upper: Before the storm, early September 2009. Middle: 11.07 a.m. on 23 September. Lower: 11.44 a.m. on 23 September (Set of three photographs courtesy of Ben Garratt, University of South Australia)

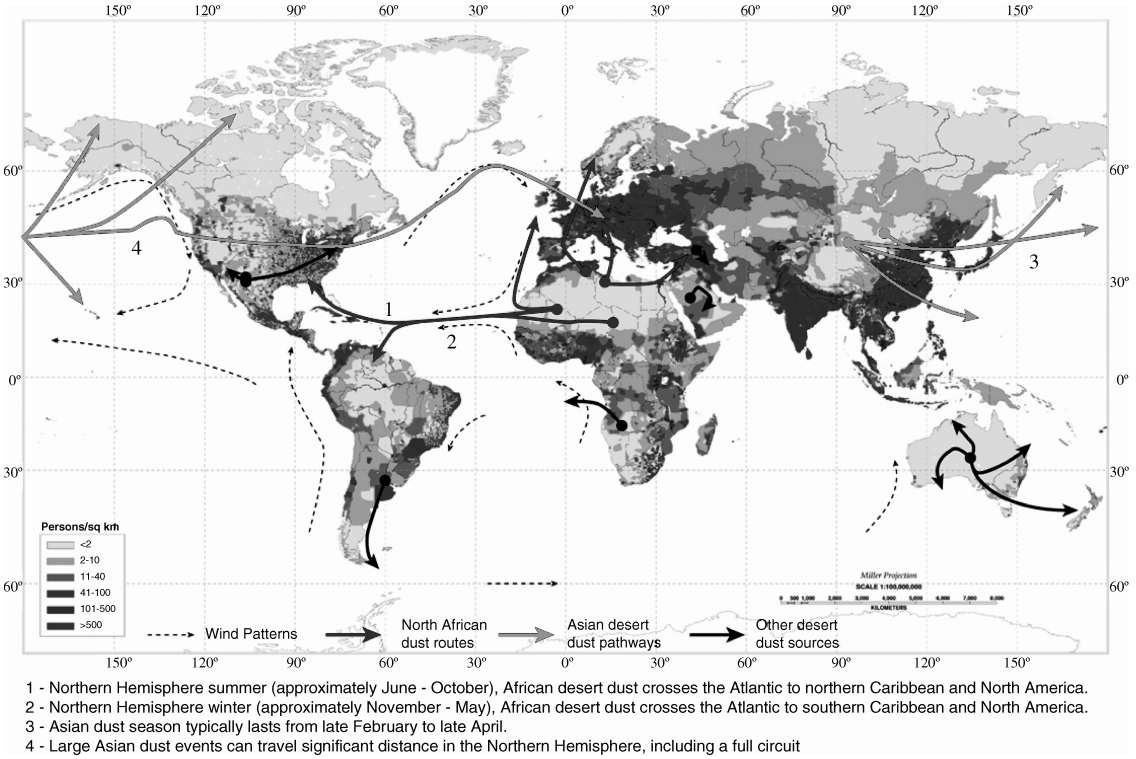

Figure 10.18 Primary sources of mineral dust and their atmospheric pathways (modified after Griffin, 2007) in relation to world population density 1994 (United States Department of Agriculture) (After Derbyshire (in press))

continuing increase in dust storms in the past quarter of a century, the degree of success has been limited. Nevertheless, success in some local communities has been achieved by considerably reducing the number of grazing animals and limiting their grazing range, together with improvement of water conservation practices (Normile, 2007). At the urban scale, rapid growth and increasing dependence on motor traffic, as well as growth of manufacturing, power and other industries, has accelerated a rise in particle-rich air pollution in many cities in eastern China and in settlements in dryland areas of intense farming activity. Most direct action to reduce risk has generally been a matter of personal initiative in response to governmental and company-management advice, notably in the use of cotton face-masks during periods of high dust density.

10.6.1.2 Volcanic plumes

A large volcanic eruption can spew ash into the stratosphere, into the path of jet streams, thereby enabling the ash to circle the globe. Occasionally, the impact of such an eruption can be significant, causing global temperature change which, in turn, can lead to droughts, crop failures and famines. The hazard itself is uncontrollable, so risk reduction can be achieved only through international economic and logistical scenario planning.

More frequently, volcanoes emit substantial quantities of ash which falls on local populations and may remain in the environment for months, years or, occasionally, decades, if the eruption is sustained. For example, the Soufrière Hills volcano, Montserrat, West Indies, started erupting in July 1995 and is still active. The population has been subjected to frequent ash falls over the past 15 years. Those most likely to develop respiratory problems are workers who are occupationally exposed to the ash (e.g. outdoor workers) and detailed risk analyses have been carried out to assess the likelihood of specific occupations developing chronic diseases (Hincks *et al.*, 2006). Assuming continuing volcanic activity, outdoor workers, as the group most at risk, are thought to have a 2–4 per cent risk of silicosis after 20 years of exposure to the cristobalite-rich ash. Risk reduction is also achieved through education of local populations, taking into account cultural issues and vulnerabilities. This includes teaching safe ways to clear ash off roofs, to clean and seal houses and to maintain respiratory health through the use of dust masks.

10.6.2 Anthropogenic dust

Biomass burning and some bush fires, the former being part of the annual agricultural cycle in many parts of the world, can generate major dust palls and smoke plumes with local to regional impact. Fires in several locations in recent years, including those in Indonesian tropical forests and Siberian boreal woodlands, led to dust palls that could be traced over several thousand kilometres. The burning of sugar cane at harvest has

been common practice worldwide, but the environmental and human impact has led to new legislation in Brazil, banning pre- and post-harvesting burns from 2010 (Le Blond *et al*., 2010).

Poor air quality arising entirely, or largely, from human action at local to regional scales is of broad provenance, including particulate matter derived from industrial, transportation, agricultural and domestic sources. Progress in reducing risks to health posed by airborne particles, largely by means of state legislation aimed at imposing measures to improve air quality, has advanced in the past half-century, although there is some way yet to go. Examples of legislation include the United States' Air Pollution Control Act of 1955 and further Acts between 1963 and the present, the United Kingdom's Clean Air Acts of 1956 (demanding use of smokeless fuel in industry) and 1968 and similar actions in several other countries. Progress in setting up air-quality standards has been much slower in less-developed countries, however, in an era in which development is advancing rapidly.

The main cause of air pollution in modern cities is vehicular traffic and the introduction of traffic-reducing or calming measures may have had an indirect influence on the levels and distributions of particles from those vehicles. London is an appropriate case study, where a congestion charge was introduced in February 2003. Atkinson *et al.* (2009) reported that, while there did not appear to be an overall reduction in pollution in London as a direct result of introducing the scheme, there was 'evidence of relative reduction' of PM_{10}. However, within London's charging zone, Beevers and Carslaw (2005) reported that PM_{10} emissions were reduced by 11.9 per cent and by 1.4 per cent on London's inner ring road. They attributed these reductions to increases in vehicle speed, as this is as important in reducing emissions as changes in vehicle numbers. Conversely, traffic calming measures such as road humps and width barriers can have a negative effect on emission levels in the immediate proximity because drivers 'aggressively' brake and accelerate through such systems (Kyoungho and Hesham, 2009).

Legislation designed to encourage decreased use of cars and to create financial advantages by using smaller and less polluting vehicles have had mixed success. High petrol taxes have been justified on the grounds that they would decrease car use and, therefore, emission levels. However, a study in Southern California and Connecticut in the USA revealed that environmental taxes on fuel resulted in only minimal reductions in driving levels (Sipes and Mendelsohn, 2001). There are also significant developments in producing viable electrical and hybrid vehicles and promoting their use. For example, the Irish government has plans for 10 per cent of all vehicles to be powered by electricity by 2020, resulting in significant emission reductions (Brady and O'Mahony, in press). In addition to new technology in the vehicle fleet, the development of new fuels offers the chance of reduced emission levels. The use of biofuels is rapidly increasing and, although there are concerns about decreased engine efficiency using these new fuels, there is generally a marked decrease in gaseous and particulate emissions. A study by Ozsezen and Canakci (2011), using a direct-injection diesel engine fuelled with canola-oil methyl ester (COME) and waste (frying) palm oil methyl ester (WPOME), showed a decrease in engine power and increased fuel use, although smoke opacity decreased by 56–63 per cent.

10.6.3 Future trends

This chapter has shown that the processes involved in the interaction of particulate matter and lung tissue, from the derivation, transport pathways and composition of airborne particles to the exposure of individuals to inhalation and the likelihood and degree of impact that may lead to lung morbidity and mortality, are compound and complex. Uncertainties arising from such complexity, especially those concerned with matters of cause and effect, are present throughout the process.

For example, the geochemical content of mineral dust is strongly influenced by its source and its complexity is increased by the presence of multiple sources arising from several factors as diverse as land surface moisture regime and variations in the weather. This and the manifold re-suspension of dust, has led to increasing use of the single-particle analytical method, rather than those based on bulk samples; as a more sensitive means of characterising both individual and multiple dust sources, the single particle method has considerable future potential (Stevens *et al*., 2010).

Global diversity in the mode and time-span of dust collection, dust measurement and statistical analysis of pollutant data has also been a limiting factor in comparative analysis between regions and in respect of community sub-groups. Other non-standard practices may include variability of criteria used in diagnosis of certain conditions (Monteil, 2008) and the assessment of the relative importance for human health of local, regional and global components in dust pollution when data on the health effects of global-scale mineral dust are sparse and opinion remains contentious (Monteil and Antoine, 2009). Future long-term, standardised studies are needed at all scales, with collaborative, comparative global programmes on the impact on health of both semi-continuous and seasonal dust palls, providing higher quality background data than is currently available.

At the other end of the spectrum, information on the precise mechanisms involved in mineral-particle pathogenesis is relatively sparse. For example, the condition of quartz-particle surfaces and the presence both upon and within them of toxins affects pathogenicity. However, while metallic iron is known to reduce quartz toxicity and thus lung inflammation, traces of ferric or ferrous iron stimulates oxidative stress on quartz surfaces that leads to damage of epithelium cells (Donaldson and Borm, 1998).

Evidence is accumulating of the presence of cause-and-effect links between far-travelled dust and morbidity arising from micro-organisms, including bacteria, fungi, pollen and viruses attached to soil particles (e.g. Griffin *et al*., 2001; Kellogg *et al*., 2004; Griffin, 2007), but the role of airborne pathogens in epidemics remains poorly understood.

Understanding of the role of airborne particles in environmentally linked health conditions has rested to varying degrees

upon epidemiological results, while definitive studies of the part played by physico-chemical processes are sparser. These and other gaps in knowledge currently legitimise doubt as to whether or not chronic exposure to naturally derived dust particles, such as those of regional to global scale, is necessarily a *direct* cause of disease.

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