COAL DUST

1. Exposure Data

1.1 Chemical and physical data

Coal is a heterogeneous, carbonaceous rock formed by the natural decomposition of plant matter at elevated temperature and pressure in the earth's crust. The subject of this monograph is 'coal dust', itself a heterogeneous by-product of the mining and use of coal.

1.1.1 *Coal types and classification*

Coal exists in various forms, ranging from lignite and brown coals (soft coals) to bituminous coals and anthracite (hard coals). Most classification schemes for coal were developed for geological and commercial reasons; the various schemes apply different weights to the many different chemical and physical properties of coal. Consequently, classifications vary widely and differ in different countries. For example, the British system relies heavily on the coking properties of the coal, whereas the system in the United States of America is based on the percentage of carbon in the coal and its calorific value. An international system does exist, and this uses a three digit code to represent the degree of volatility and 'caking' (coking) properties (Speight, 1994).

Despite these apparent differences, on closer examination it is clear that most systems demonstrate an underlying consistency with each other, in that they all reflect the geologic age of the coal. In this regard, a widely used and convenient term is coal rank. Coal rank varies from high to low; high rank coals are generally older, have the greatest fixed carbon, have the least volatile matter, the lowest moisture content, and the highest calorific value, and vice versa. The highest rank coal is anthracite, followed by the bituminous and sub-bituminous coals, and ending up with the brown coals and lignite (**Table 1**).

Two other parameters are frequently used to classify coal: ash content and sulfur content. Ash content, the residue following low temperature combustion, is commercially relevant. This can vary substantially (3–20%), but is not necessarily related to coal rank. Sulfur content is also commercially (and environmentally) important, but again is not strongly correlated with coal rank.

Table 1. Classification of coal according to rank[a]

Class	Group	Limits of fixed carbon or Btu, mineral-matter-free basis	Requisite physical properties
I. Anthracite	1. Meta-anthracite	Dry FC, ≥ 98% (dry VM, ≤ 2%)	
	2. Anthracite	Dry FC, 92–98% (dry VM, 2–8%)	
	3. Semi-anthracite	Dry FC, 80–92% (dry VM, 8–14%)	Non-agglomerating[b]
II. Bituminous[c]	1. Low-volatile bituminous coal	Dry FC, 78–86% (dry VM, 14–22%)	
	2. Medium-volatile bituminous coal	Dry FC, 69–78% (dry VM, 22–31%)	
	3. High-volatile A bituminous coal	Dry FC, < 69% (dry VM, > 31%); and moist Btu, ≥ 14 000[d,e]	
	4. High-volatile B bituminous coal	Moist Btu, 13 000–14 000[e]	
	5. High-volatile C bituminous coal[f]	Moist Btu, 11 000–13 000[e]	
III. Sub-bituminous	1. Sub-bituminous A coal	Moist Btu, 11 000–13 000[e]	Both weathering and non-agglomerating
	2. Sub-bituminous B coal	Moist Btu, 9500–11 000[e]	
	3. Sub-bituminous C coal	Moist Btu, 8300–9500[e]	
IV. Lignite	1. Lignite	Moist Btu, < 8300	Consolidated
	2. Brown coal	Moist Btu, < 8300	Unconsolidated

From ASTM (1991); FC, fixed carbon, VM, volatile matter; Btu, British thermal units

[a] This classification does not include a few coals that have unusual physical and chemical properties and that come within the limits of fixed carbon or Btu of the high-volatile bituminous and sub-bituminous ranks. All these coals contain less than 48% dry, mineral-matter-free fixed carbon or have more than 15 500 moist, mineral-matter-free Btu.

[b] If agglomerating, classified in low-volatile group of the bituminous class.

[c] It is recognized that there may be non-caking varieties in each group of the bituminous class.

[d] 'Moist Btu' refers to coal containing its natural bed moisture but not including visible water on its surface.

[e] Coals having ≥ 69% fixed carbon on the dry, mineral-matter-free basis shall be classified according to fixed carbon regardless of Btu.

[f] There are three varieties of coal in the high-volatile C bituminous coal group: variety 1, agglomerating and non-weathering; variety 2, agglomerating and weathering; variety 3, non-agglomerating and non-weathering.

1.1.2 *Bulk coal composition*

The predominant constituent of coal is carbon. The carbon content of various types of coal is shown in **Table 2**. Because of its origin, some organic functional groups (e.g. –COOH, –OH) are retained to a greater or lesser extent depending upon the coal rank. They are present at the surface of the coal and affect surface reactivity. A wide range of minerals are also found in the coal, including clays, carbonates, sulfide ores, oxide ores, quartz, phosphates and heavy minerals. The mineral matter may be intrinsic to the coal, as in the silica grains in the coal matrix, or may lie in pockets or layers, having been originally washed in with the plant matter or having later percolated in and been deposited in cracks and fissures in the coal (Speight, 1994).

Table 2. Carbon content of coals

Coal type	Rank	Composition (%) (dry mineral-matter-free basis)		
		Carbon	Hydrogen	Oxygen
Peat		50–65	5–7	30–40
Lignite	(Low)	65–75	5–6	20–30
Sub-bituminous	↓	75–80	5–6	13–20
Bituminous	(Intermediate)	80–90	4.9–5.7	5–15
Semi-bituminous	↓	90–92	4.5–5.9	4–5
Anthracite	(High)	92–95	2–4	2–4

From Parkes (1994)

The proportion of minerals in the coal, and their relative composition varies widely from coal seam to coal seam, and often within the same seam. **Table 3** illustrates the marked difference in composition between two seams in Kentucky in the United States (Braunstein *et al.*, 1977). In general, the most common clay minerals found in coal are kaolinite and illite. With regard to the other constituents, calcite and siderite are common carbonates, and pyrite a common sulfite (Speight, 1994). Ten inorganic oxides commonly found in coal ash are shown in **Table 4**. **Table 5** gives the appropriate distribution of elements and trace elements in coal.

Organic compounds in coal include methane, benzene, phenols, naphthalenes, acenaphthalenes and 3-, 4- and 5-ring polycyclic aromatic hydrocarbons. The latter include benzo[*a*]pyrene, chrysene, cyclopentanochrysene and benz[*a*]anthracene derivatives (Falk & Jurgelski, 1979).

1.1.3 *Coal dust composition*

Virtually all of the information available on the composition of coal dust comes from industrial hygiene studies in coal mines. In this section, data on exposures to crystalline silica (quartz) (see also the monograph on silica in this volume) as a component of the dust in coal mines are presented. For other exposure data, see Section 1.3.

Table 3. Some minerals occurring in coals, expressed as a percentage of total mineral matter

Classification	Mineral constituents	Elkhorn No. 3 seam, Kentucky	Hartshorne seam, Kentucky
Silicates	Kaolinite	3–40	1–10
	Illite	Trace	1–10
	Chlorite	Trace	1–10
	Mixed-layer illite, montmorillonite	Trace	
Carbonates	Siderite		30–40
Oxides	Quartz	40–50	1–10
	Haematite	ND	ND
	Rutile	1–10	
Sulfates	Gypsum	1–10	1–10
	Thernardite	ND	ND
Sulfides	Pyrite	1–10	1–10

From Braunstein *et al.* (1977)
ND, no data available

Table 4. Elemental composition of mineral matter in coal ash

Constituent	Representative percentage
SiO_2	40–90
Al_2O_3	20–60
Fe_2O_3	5–25
CaO	1–15
MgO	0.5–4
Na_2O	0.5–3
K_2O	0.5–3
SO_3	0.5–10
P_2O_5	0–1
TiO_2	0–2

From Speight (1994)

Coal mine dust is a complex and heterogeneous mixture containing more than 50 different elements and their oxides. The mineral content varies with the particle size of the dust and with the coal seam. Airborne respirable dust in underground coal mines has been estimated to be 40–95% coal (Walton *et al.*, 1977; United States National Institute for Occupational Safety and health, 1995); the remaining portion consists of a variable mixed dust originating from fractured rock on the mine floor or roof or from within the coal seam. Mineral dust can also be introduced into the mine atmosphere through

Table 5. Elements and trace elements in coal

Constituent	Range (percentage)	Constituent	Range (ppm)
Aluminium	0.43–3.04%	Arsenic	0.5–93 ppm
Calcium	0.05–2.67%	Boron	5–224 ppm
Chlorine	0.01–0.54%	Beryllium	0.2–4 ppm
Iron	0.34–4.32%	Bromine	4–52 ppm
Potassium	0.02–0.43%	Cadmium	0.1–65 ppm
Magnesium	0.01–0.25%	Cobalt	1–43 ppm
Sodium	0–0.2%	Chromium	4–54 ppm
Silicon	0.58–6.09%	Copper	5–61 ppm
Titanium	0.02–0.15%	Fluorine	25–143 ppm
Organic sulfur	0.31–3.09%	Gallium	1.1–7.5 ppm
Pyritic sulfur	0.06–3.78%	Germanium	1–43 ppm
Sulfate sulfur	0.01–1.06%	Mercury	0.02–1.6 ppm
Total sulfur	0.42–6.47%	Manganese	6–181 ppm
Sulfur by X-ray fluorescence	0.54–5.4%	Molybdenum	1–30 ppm
		Nickel	3–80 ppm
		Phosphorus	5–400 ppm
		Lead	4–218 ppm
		Antimony	0.2–8.9 ppm
		Selenium	0.45–7.7 ppm
		Tin	1–51 ppm
		Vanadium	11–78 ppm
		Zinc	6–5350 ppm
		Zirconium	8–133 ppm

From Ruch *et al.* (1974)

operations other than coal cutting, such as in roof bolting or in the distribution of rock dust (a low-silica limestone dust) to prevent explosions. In addition, the presence of diesel equipment underground will lead to a substantial amount of fine particulate (< 1 μm) in the dust, the composition of which would be fairly typical of diesel exhaust from industrial machines (see IARC, 1989). Certain jobs in underground mines involve exposures to isocyanates and urethanes.

The coal component of respirable dust at surface coal mines can be highly variable. This variation depends on the stage of the mining operation at such opencast sites (United States National Institute for Occupational Safety and Health, 1995).

Those involved in sampling dust in coal mines have usually concentrated on assessing those constituents associated with pneumoconiosis, the major health hazard of coal mining. These constitutents have included mixed respirable dust, quartz (silica), kaolin and mica, coal rank (percentage carbon) and ash (these components are not mutually exclusive and do not add to 100%).

Compositional data for airborne coal mine dust collected in British collieries[1] are presented in **Table 6**. About one-third of each dust sample was non-coal material. On average, quartz made up about 4% of the dust (range, 0.8%–6.9%), and this corresponds to a gravimetric airborne concentration of about 0.17 mg/m^3. Quartz levels tended to vary inversely with coal rank, being the greatest in low-rank coal seams. Kaolin and mica constituted 14% of the airborne dust overall, or about 0.6 mg/m^3 (Jacobsen *et al.*, 1971; Walton *et al.*, 1977). Quartz levels at eight other British mines for 1970–75 ranged from 1.5% to 10.3% (Crawford *et al.*, 1982).

Table 6. Compositional data for airborne dusts in British coal mines prior to 1970[a]

Coalfield	Colliery	Mean environmental data[a]			
		Carbon (%)	Non-coal (%)	Quartz (%)	Kaolin and mica[b] (%)
Scottish	SC1	84.1	36	4.3	15.7
	SC2	85.4	42	5.5	12.2
	SC4	82.0	62	5.8	23.0
	SC5	82.6	43	3.0	17.1
Northumberland	NH1	84.0	43	3.0	12.5
Cumberland	C1	86.9	44	6.8	11.5
Durham	D1	86.3	35	3.4	12.6
	D2	89.7	33	5.9	8.6
Yorkshire	Y1	85.3	43	6.2	14.2
	Y2	85.2	51	7.8	17.5
Lancashire	L1	87.8	19	1.2	7.3
North Wales	NW1	84.9	39	6.9	15.1
Nottingham	NT1	81.1	51	5.1	32.8
Warwick	W1	81.8	42	4.2	9.3
South Wales	SWA1	94.0	31	3.2	8.8
(anthracite)	SWA2	92.7	19	0.8	11.4
South Wales	SWS1	91.2	18	2.2	21.1
(steam coal)	SWS3	91.9	20	2.3	8.4
South Wales (bituminous coal)	SWB1	90.6	28	2.8	6.8
Kent	K1	88.6	32	2.0	16.3
All collieries		86.8	36	4.1	14.1

From Jacobsen *et al.* (1971); Walton *et al.* (1977)

[a] Percentages are not necessarily additive and should not total to 100%

[b] Computed from quotient of cumulative exposures to kaolin and mica and cumulative exposure to mixed dust by the Working Group

[1] In Britain, the word mine tends to refer to a surface mine, while pit and colliery tend to refer to underground mine. In the United States a mine can be an underground or a surface mine, while the word pit could refer to a surface excavation. In this section the terms are used as they appear in the original papers.

Tomb *et al.* (1995) reported on an extensive programme of sampling for crystalline silica (quartz) that took place in underground mines in the United States between 1985 and 1992. **Table 7** shows the average percentage of quartz detected in this study in personal dust samples for 10 underground occupations. The mean level over the 10 occupations was 4.7% (range, 2.5–7.0%), which is similar to that reported above for British mines. Roof bolters had the highest exposures to quartz. Roof bolting involves drilling into the roof rock strata, which is often sandstone or other siliceous rock.

Table 7. Quartz percentages in dust for various underground occupations in United States mines, 1985–92

Occupation	Number of samples	Average quartz content (%)[a]
Roof bolter	6 061	6.97
Roof bolter (DA)[b]	3 508	6.77
Continuous-miner operator	10 793	5.54
Continuous-miner helper	1 386	5.48
Shuttle car operator	1 883	4.33
Scoop car operator	721	4.27
Longwall shearer operator	762	4.02
Jacksetter	815	3.98
Coal drill operator	395	3.29
Cutting machine operator	1 067	2.47

From Tomb *et al.* (1995)

[a] Values quoted are the intercept values from regressions of percentage quartz against time; they thus probably reflect conditions relevant to the start of the period, 1985–92

[b] Data available only since 1986; DA, designated area (area sample)

Leiteritz *et al.* (1971) analysed fine dust from underground coal mines in Germany. **Table 8** presents the mean percentages of quartz, ash, kaolinite and sericite/illitte for three broad coal types; quartz levels ranged from about 2.4% to 5%, with the lower levels associated with the higher coal rank regions. For coal-winning jobs, the quartz level averaged about 3% (**Table 9**). Other information from German mines (**Tables 10** and **11**) gives a similar picture. These findings from German mines indicate that quartz levels were similar to, though slightly lower than, those measured in British and United States mines.

Cram and Glover (1995) reported on quartz samples taken from underground coal mines in New South Wales, Australia, between 1984 and 1995; about 1.7% of these samples exceeded the respirable quartz limit of 0.15 mg/m^3. However, this is unlikely to be a representative figure. The samples analysed were not chosen randomly from all dust samples, but tended to represent locations where high quartz levels are expected. Indeed,

high quartz levels were typically found when tunnelling, when cutting rock or in certain coal seams with a high quartz content.

Table 8. Mean percentages of ash, quartz, kaolinite and sericite/illite in the dust of German coal mines

Type of coal	Ash (%) mean ± SD	Quartz (%) mean ± SD	Kaolinite (%) mean ± SD	Sericite/illite (%) mean ± SD
Fine dust < 5 μm				
Anthracite to steam coal	19.1 ± 8.8	2.4 ± 1.4	3.8 ± 1.0	11.2 ± 6.8
Bituminous coal	21.0 ± 9.0	2.6 ± 1.1	4.7 ± 2.0	12.5 ± 6.1
Gas coal to long-flaming coal	32.8 ± 17.8	5.0 ± 3.6	7.1 ± 2.8	20.2 ± 10.0
Fine dust < 3 μm				
Anthracite to steam coal	20.9 ± 10.9	2.5 ± 1.5	3.9 ± 1.7	13.0 ± 8.1
Bituminous coal	18.0 ± 9.8	1.8 ± 0.9	4.4 ± 2.2	10.6 ± 6.1
Gas coal to long-flaming coal	37.0 ± 18.6	4.2 ± 2.5	10.3 ± 5.6	19.6 ± 11.2

From Leiteritz *et al.* (1971)

Table 9. Mean quartz content in airborne dust generated during coal winning in German mines

Particle size	Number of measurements	Quartz content (% by weight) mean ± SD[a]
Total dust	165	4.1 ± 3.3
Fine dust < 7 μm	165	4.3 ± 3.0
Fine dust < 5 μm	123	2.9 ± 1.9
Fine dust < 3 μm	159	2.2 ± 1.6

From Leiteritz *et al.* (1971)
[a]SD, standard deviation

Houbrechts (1960a) found that the free silica content in an underground coal mine in Belgium prior to 1959 varied from 4.2% for coal-winning jobs up to 14% for workers involved with roof control. Houbrechts (1960b) reported that mean levels were 4.6% for coal-winning and 8.9% for roof control.

Investigators of the bioavailability of silica in coal mine dust have examined the surface properties of the particles using various techniques (Bolsaitis & Wallace, 1996). Recently, Wallace *et al.* (1996) employed electron microscopy, using beams of increasing energy coupled with energy dispersive X-ray analysis, to explore the composition of particles progressively through the particle surface to the core. These authors found that decreasing coal rank was associated with increasing proportions of clay-occluded silica particles. This finding is consistent with the finding that dusts from lower

coal rank mines are less fibrogenic, despite the apparent presence of more silica in those dusts.

Table 10. Quartz percentage and concentration in the return air of coal-faces in different coal seams in the Ruhr, Germany in 1955 and in 1963–71

Location and survey period	Quartz content in respirable dust (%)	Respirable quartz concentration[a] (mg/m^3)
Low-rank coal		
Dorsten, Horst, Essen		
1955	3.3	0.23
1963–67	3.7	0.21
Bochum		
1955	2.2	0.37
1963–67	2.1	0.21
High-rank coal		
Witten, Sprockhövel		
1955	1.5	0.35
1963–67	1.9	0.22

From Reisner *et al.* (1982)
[a]Converted from particle counts

Table 11. Mean and maximal respirable quartz concentrations for miners in three German underground mines, 1974–91

Quartz concentrations (mg/m^3)	Heinrich Robert (high rank)	Walsum (low rank)	Saar (special low rank)[a]
Mean	0.05	0.10	0.21
Maximum	0.13	0.21	0.81

From Morfeld *et al.* (1997)
[a]Period is 1980–91 for this mine

Recent research (Fubini *et al.*, 1995; Vallyathan *et al.*, 1995) indicates that knowledge of the age of dust in terms of the length of time since it was originally fractured may also be important in understanding the biological role and activity of silica and, hence, coal dust (see the monograph on silica in this volume).

1.1.4 *Particle size distribution*

The particle size distribution of dust in the underground mine environment includes respirable, thoracic and inhalable particulate mass fractions. These fractions are defined as those that have the aerodynamic characteristics that result in deposition in the

following regions of the human respiratory tract: the gas-exchange region (respirable dust), the lung airways and gas-exchange region (thoracic dust), and anywhere within the respiratory tract (inhalable dust) (United States National Institute for Occupational Safety and Health, 1995).

A recent intensive study of particle size-specific fractions of dust in underground coal mines (Seixas *et al.*, 1995) came to the conclusion that particle size distributions may differ across mines, but were similar across different occupations within a mine. Overall, thoracic particulate mass was about four times greater than the respirable mass (as defined by the American Conference of Governmental Industrial Hygienists (ACGIH), 1985), while the alveolar deposition fraction was about 60% of the respirable mass.

A much older German study by Leiteritz *et al.* (1971) used various instruments and techniques to determine underground dust concentrations in the following four size ranges: total dust; fine dust < 7 μm; fine dust < 5 μm; and fine dust < 3 μm. The results, which are shown in **Table 12**, indicate a fivefold factor for the ratio of total dust to dust < 5 μm at coal-winning sites. This figure appears broadly similar to some data obtained from coalface workers in British mines (see **Table 13**) (Dodgson *et al.*, 1975); direct comparison between these datasets is impossible because of the different sampling techniques used.

Table 12. Mean dust concentrations in airborne dust generated during coal winning in German mines

Particle size	Number of measurements	Dust concentration (mg/m^3) mean ± SD
Total dust	165	53.1 ± 29.4
Fine dust < 7 μm	165	25.3 ± 13.0
Fine dust < 5 μm	123	9.2 ± 7.9
Fine dust < 3 μm	159	2.1 ± 1.6

From Leiteritz *et al.* (1971)

Two studies, one examining total dust concentrations in underground mines (Cowie *et al.*, 1981) and the other inspirable dust (Mark *et al.*, 1988), concluded that the respective dust fractions were related linearly to measurements of respirable dust.

1.1.5 *Analysis*

Three types of environmental monitoring are generally used for sampling airborne coal dust. These include personal sampling, breathing zone sampling and area sampling. For personal sampling, a device is attached to the worker and is worn continuously for all work and rest periods during the shift. For breathing zone sampling, a device is placed in the breathing zone of the worker; a second individual may be required to hold the device in this location. For area sampling, the sampler is placed in a fixed location in the workplace. When the purpose of the environmental monitoring is to determine worker

exposures, personal or breathing zone sampling should be used. To determine worker exposures by means of area sampling requires a site-specific demonstration that such samples are analogous to worker exposures (United States National Institute for Occupational Safety and Health, 1995).

Table 13. Total and respirable dust concentrations in British mines prior to 1970

Colliery[a]	Coalface samples			Samples from elsewhere underground		
	No. of samples	Mean respirable dust (mg/m^3)	Mean total dust (mg/m^3)	No. of samples	Mean respirable dust (mg/m^3)	Mean total dust (mg/m^3)
NT1	28	4.40	22.65	7	1.91	12.36
W1	22	4.40	27.67	–	–	–
SC1	11	4.30	21.90	1	1.68	12.89
Y2	14	8.23	58.76	–	–	–
SWB1	41	4.60	42.74	9	1.46	20.10
SWS3	24	6.70	82.85	5	5.54	76.63
SWA1	18	3.29	33.92	10	1.49	21.72

From Dodgson *et al.* (1975)
[a] See also Table 6

The concentration of respirable coal mine dust in the mine atmosphere is determined gravimetrically. In the United States, such respirable coal dust is sampled with a coal mine dust personal sampler unit. Respirable dust, passing through the unit, is collected on a 5 μm polyvinyl chloride (PVC) filter. The respirable dust concentration in the mine atmosphere is then determined from the mass of dust collected and the volume of air sampled (United States National Institute for Occupational Safety and Health, 1995).

In the United States, sampling and analysis for respirable crystalline silica should be performed in accordance with United States National Institute for Occupational Safety and Health Method 7500 or 7602 or a demonstrated equivalent. Sampling devices that may be used for Method 7500 or 7602 include the following: the coal mine dust personal sampler unit (CPSU) (with a 0.8 μm or 5 μm PVC or mixed cellulose ester membrane filter) operated at a flow rate of 1.7 L/min; the Higgins-Dewell sampler operated at 2.2 L/min; or an equivalent sampler. The presence of kaolinite and calcite in the dust sample may interfere with analysis by Method 7602. If these minerals are present, correction procedures should be used. When respirable coal mine dust is to be analysed in the same sample, mixed cellulose ester membrane filters should not be used because of their high weight variability. A pre-weighed PVC filter should be used and a final weight should be taken before ashing when Method 7602 is used to analyse crystalline silica in coal mine dust. In Method 7500, neither kaolinite nor calcite interfere with the method if the samples are ashed in a low-temperature asher or if they are suspended in tetrahydrofuran (United States National Institute for Occupational Safety and Health, 1995).

The current analytical method used by the United States Mine Safety and Health Administration (known as MSHA P-7) differs from United States National Institute for Occupational Safety and Health Method 7602 in the sample preparation procedures. The uneven deposition of ash that has been observed in the filtration step of MSHA P-7 can adversely affect the quantification of the quartz. United States National Institute for Occupational Safety and Health Method 7603 is similar to MSHA P-7 both in its use of the same filtration technique and in its specification of a 2.0 L/min flow rate for sample collection. Both methods are designed specifically to analyse respirable crystalline silica in coal mine dust and thus may reduce some of the interferences that can occur in samples collected in the mining environment. However, United States National Institute for Occupational Safety and Health Method 7602 is the preferred infrared method because it avoids the uneven deposition of ash and has the more appropriate sample collection flow rate of 1.7 L/min. In lieu of either United States National Institute for Occupational Safety and Health Method 7603 or MSHA P-7, United States National Institute for Occupational Safety and Health Method 7602 is recommended for the analysis of respirable crystalline silica (United States National Institute for Occupational Safety and Health, 1995).

1.2 Production and use

Coal has been burned in China for thousands of years, and its use in Europe goes back at least 2000 years (Schobert, 1987). By the thirteenth century, coal was in wide use in Europe, and air pollution was becoming a problem in some cities. A major increase in usage came with the Industrial Revolution and the invention of the steam engine. Subsequently, there was a rapid increase in coal mine employment and production, and this continued until the early part of the twentieth century. Employment in coal mining peaked in 1923 in the United States, at which time over 800 000 miners were employed (United States Bureau of the Census, 1975). However, after decades of the declining use of coal for transportation and steel-making, coal mining employment in the United States in 1993 stood at about 100 000 (United States Energy Information Administration, 1996). About two-thirds of these miners worked underground (United States National Institute of Occupational Safety and Health, 1995). A similar trend in coal mine employment has occurred in Europe, particularly in recent years. From 1980 to 1991, coal mining employment in the European Union halved, from 583 000 to 260 000 miners. Less than 10% of European coal is extracted from surface mines (European Commission, 1993).

Coal is found on all continents. However, no coal is mined in Antarctica, and production is low in South America and Africa relative to the other continents. Coal is mined in about 70 different countries, there being a very wide range in production, from countries producing just a few thousand tonnes per year, to a single country, China, with a production of over 1×10^9 tonnes. The top five coal-producing countries were reported to be China, the United States, Russia, Germany and Australia in 1992 (United States Bureau of Mines, 1992). Production figures for these and for further major producers are

shown in **Table 14**. Note that the division between production of lignite and harder coals differs markedly among countries.

Table 14. Coal production reported in 1992 in major coal producing countries (million tonnes)

Country	Lignite	Bituminous coal and anthracite
China	–	1 110
United States	82	821
Russia	60	275
Germany	242	66
Australia	50	205
India	15	210
Poland	67	132
South Africa	–	174
Ukraine	7	127
Kazakhstan	–	127
Former Czechoslovakia	82	19
Republic of Korea	21	70
United Kingdom	–	87
Canada	10	55
Turkey	50	5
Greece	54	–
Romania	35	5
Spain	19	15
Chile	–	132

From United States Bureau of Mines (1992)

To understand why dust exposures vary both in extent and in composition, it is necessary to understand the coal mining process. Coal is mined by surface or underground methods. In the former, the strata overlying the coal are removed, usually by drilling, blasting and use of bulldozers or dragline excavators. The overburden consists of various rock types, including limestone, sandstone, clays and shales. The uncovered coal is loaded into trains or trucks for delivery to the user. Reclamation of the land sometimes follows coal removal.

In underground mining, shafts are sunk vertically, or slopes or drifts cut at an angle or horizontally, in order to reach the coal seams. Bituminous coal has been, and still is, cut in various ways. Originally, manual labour was used. Later, this was followed by the technique called conventional mining, in which a machine is used to remove a thin slice of coal from the lower part of the coal seam. Explosives then bring down the upper part of the seam. Though this system remains in use, a technical advance on it was the continuous miner, which is a machine with a rotating cutter on a boom. In the conventional and continuous miner methods of mining, the roof is usually supported by pillars of coal,

leading to the terms 'room-and-pillar' or 'bord-and-pillar' mining. Roof bolts are often used to prevent falls of rock from the ceiling strata.

The most recently adopted method is the longwall face. The longwall method permits much higher productivity, although it often incurs much higher dust levels. In longwall mining, a machine removes a strip of coal from the coal-face, the roof being supported by jacks. As the face moves forward, the mined-out area is left to collapse. Other methods that are less frequently used include shortwall mining and auger mining.

Anthracite mines often pose special difficulties. The seams of coal are folded and typically incline, sometimes at extreme angles. In these cases, the pitch mining technique is employed, in which the miners work upwards through the seam, the work being slow and strenuous.

Various geological features impinge greatly on the underground mining of coal and can have major effects on the degree and type of dust exposures. Among these are coal seam splits and dirt bands in the coal. Often, with modern techniques, there is no option but to mine these non-coal layers together with the coal, the resulting coal mixture being cleaned of spurious material at the surface. Faults, in which the coal and adjacent rock strata are displaced, can be problematic for the mining engineer. Rock may need to be cut in order to move the face back into the coal seam. In addition, it is sometimes necessary to cut into the floor or roof in order to remove unstable or soft material, or, in the case of thin seams of coal, to provide sufficient room in which to work. Roof bolting involves drilling into the ceiling rock.

The principal use for coal is for power generation, which accounted for 88% of total consumption in 1993 in the United States and 66% of total consumption in 1991 in the European Union. Coke production in each location accounted for 3% and 20%, respectively. Other industrial and domestic uses accounted for the remainder of consumption (European Commission, 1993; United States Energy Information Administration, 1996).

1.3 Occurrence and exposure

This section presents information on occupational exposure to airborne coal dust (see also Sections 1.1.3 and 1.1.4). Nearly all of the available data are for coal mining operations. When assessing and comparing this information, it must be borne in mind that the data were collected by a variety of techniques and for different purposes. Some of the data were obtained in order to undertake research into health risks, while other data were collected for regulatory purposes. Sampling instruments differ considerably; the data range from converted particle counts to direct gravimetric measurements. Some of the sample measurements are from static samples, while others are from personal or quasi-personal sampling. These fundamental differences make direct comparisons difficult. Unless otherwise stated, all dust concentrations are for the respirable mixed dust fraction (approximately 50% of particles selected at 3.5 μm, the exact form of size cut-off with particle size depending on sampling instrument and technique).

1.3.1 *Underground mines*

Dust levels in underground mines vary considerably according to location within the mine. In general, workers at the coalface receive the highest exposures, while those working progressively further away experience lower exposures. In addition, those employed in locations receiving intake (clean) air are exposed to lower dust levels than those who have to breathe returning air, which has passed the coalface. Most surface workers at underground mines experience lower dust exposures than their colleagues underground. However, some jobs, such as tipple and coal cleaning, involve dust exposures equivalent to some underground occupations.

Table 15 shows how dust concentrations differed among occupations in 29 underground mines in the United States between 1968 and 1969 (Attfield & Morring, 1992). Workers at the coalface (e.g. cutting machine operators, continuous miner operators) were experiencing average dust concentrations of about 6–10 mg/m^3. Other workers, employed away from the face (e.g. supply men, brattice men, motormen), were exposed to much lower levels of about 1–2 mg/m^3. Surface jobs at underground mines involved lower exposures, in general, most being less than 1.5 mg/m^3 (see **Table 16**) (Parobeck & Tomb, 1974).

Although ventilation and production play a major role in affecting dust levels, the mining method is also a critical factor. In general, the longwall method of mining, with its high productivity in what is often a confined space, has higher dust concentrations than jobs associated with room-and-pillar mining. For example, Watts and Niewiadomski (1990) reported that dust levels for one longwall face occupation were about twice as great as the most exposed job on continuous miner face sections. Parobeck and Jankowski (1979) collected data in coal mines in the United States between 1970 and 1977. Auger and conventional mining led to the lowest dust levels, with continuous miner faces producing slightly more. Longwall faces, introduced in 1975, were associated with by far the most dust.

Industrial hygiene information collected at 20 British mines prior to 1969 as part of a research study showed that dust levels were comparable to those in United States underground mines at about the same time (Jacobsen *et al.*, 1971). The average concentration over all collieries was 4.1 mg/m^3. This average conceals a wide range of inter-mine variation, from 1.2 to 8.2 mg/m^3 (**Table 17**). In general, the dust level was correlated with coal rank, the concentrations being the greatest where the higher-rank coal was mined. Further information on dust level by mine, collected for the purpose of compliance with regulations, is provided for 274 British collieries for 1970–75 (Crawford *et al.*, 1982). This showed that mean dust concentrations in the face air return lay between 3 and 9 mg/m^3 in most cases, with a maximum of about 12 mg/m^3, respectively.

Dust levels in western German mines appear to have been similar to those seen in the United States and the United Kingdom (Leiteritz *et al.*, 1971). Data from 11 collieries gave a mean of 9.2 mg/m^3 (< 5 μm particle size) and 2.1 mg/m^3 (< 3 μm particle size) for coal-winning occupations (**Table 12**). Other German information (Breuer & Reisner, 1988), on all miners in 10 collieries in the Ruhr from 1954 to 1973, gave a mean level of

3.9 mg/m^3, with a trend downwards from 5.7 mg/m^3 in 1954–58 to 2.6 mg/m^3 in 1969–73. **Table 18** gives dust concentrations converted to the respirable fraction for three mining areas of western Germany (Reisner *et al.*, 1982); levels of between 7 and 23 mg/m^3 were current around 1955 and those of 6–12 mg/m^3 from 1963 to 1967. Finally, more recent information is given in **Table 19**, which shows respirable dust concentrations in three German mines prior to 1991. Based on over 10 000 gravimetric measurements at fixed locations converted to personal exposures, these data indicate dust levels of between 1.6 and 2.9 mg/m^3 on average, with maximum values about twice the mean.

Table 15. Mean respirable coal dust concentrations by occupation in 29 United States mines between 1968 and 1989

Occupation	Number of samples	Mean concentration (mg/m^3)
Roof bolter helper	30	8.4
Jack setter (longwall)	25	7.7
Continuous miner operator	486	6.8
Rock duster	15	6.6
Cutting machine helper	68	6.4
Coal drill operator	127	5.7
Auger jack setter (intake)	73	5.7
Continuous miner helper	165	5.4
Cutting machine operator	363	5.1
Blaster	134	4.8
Loading machine operator	225	4.7
Loading machine helper	44	4.5
Roof bolter	603	3.0
Face beltmen, conveyor men	75	3.0
Labourer	19	3.0
Non-face beltmen, convevor men	60	2.8
Hand loaders	93	2.6
Brattice men	34	2.4
Section foremen	339	2.2
Shuttle car operator	632	2.1
Supply men	20	2.1
Utility men	26	2.0
Motormen	19	1.8
Face mechanics	171	1.7
Electrician	11	0.9

From Attfield & Morring (1992)

The Dutch Technical Research Institute carried out limited dust measurements in 1963 in a sample of 159 workplaces selected to represent the general exposure situation in coal pits in the Netherlands. The mean total gravimetric dust concentration was 27.3 mg/m^3; the mean proportion of quartz was 5.3%. Differences existed in the dust

concentrations between the pits and seams; however, the underground exposure to mine dust was generally high and usually above 20 mg/m^3 (Meijers *et al.*, 1991).

Table 16. Dust levels for surface jobs at United States underground mines

Occupation	Average (mg/m^3)	Range (mg/m^3)	Number of samples	Percentage of samples ≤ 2.0 mg/m^3
Clean-up man	1.5	0.1–10.8	853	79
Scalper screen operator	1.3	0.1–9.5	514	76
Cleaning plant operator	1.3	0.1–10.4	1 568	81
Welder	1.2	0.1–14.8	4 176	84
Tipple operator	1.1	0.1–10.6	2 269	85
Labourer	0.9	0.1–12.3	6 108	89
Mechanic	0.8	0.1–11.0	7 839	90
Refuse truck driver	0.7	0.1–9.3	967	92
Car dropper	0.7	0.1–12.0	1 733	93
Highlift operator	0.7	0.1–10.9	2 584	94
Electrician	0.6	0.1–9.9	1 923	94
Shopman	0.6	0.1–9.6	498	95
Coal truck operator	0.6	0.1–9.5	4 472	95
Oiler/greaser	0.5	0.1–9.8	2 505	96
Outside foreman	0.5	0.1–11.1	1 079	97
Lampman	0.4	0.1–8.1	504	98

From Parobeck and Tomb (1974)

Goldstein and Webster (1972) reported some gravimetric dust samples taken in South African mines prior to 1970. Dust levels were in the range 3.9–12.5 mg/m^3, the highest concentrations occurring during coal cutting and the lowest at the surface. Person-weighted dust concentrations, converted from photoelectric measurement to gravimetric, lay in the range 2.5–3.0 mg/m^3.

Huhrina and Tkachev (1968) measured dust concentrations in two coalfields in the former USSR in 1965. In the Kuzneck coalfield, these authors reported total dust concentrations of 60–70 mg/m^3 for mechanized mining. In another mine at the Doneck coalfield, the highest average concentrations of 2.2–2.8 g/m^3 were found during mechanical extraction without water spraying and the lowest concentrations of 22 mg/m^3 were found for support work.

In 1981–82, Elez *et al.* (1985) measured total dust concentrations in the working zones of miners engaged in underground transport in two mines in the Doneck coalfield in the former USSR (**Table 20**). Mean dust concentrations for various occupations ranged 6.4 to 79 mg/m^3. A maximal concentration of 113 mg/m^3 was measured during the transportation of non-humidified coal.

Huhrina and Tkachev (1968) reported lower total dust concentrations in mines in the Moscow coalfield, where high concentrations of water are found in the coal. Average concentrations on cutter–loader and heading machines without water spraying devices

did not exceed 50 mg/m^3, and 40% of the samples were found to contain less than the maximal allowable concentration (10 mg/m^3); 80–85% of the samples were also below 10 mg/m^3 during loading.

Table 17. Dust concentration data from British coal mines prior to 1969[a]

Coalfield	Colliery	Dust concentration (mg/m^3)
Scottish	SC1	1.60
	SC2	1.60
	SC4	1.20
	SC5	3.40
Northumberland	NH1	1.60
Cumberland	C1	4.40
Durham	D1	5.00
	D2	4.80
Yorkshire	Y1	2.60
	Y2	4.50
Lancashire	L1	7.20
North Wales	NW1	5.90
Nottinghamshire	NT1	5.90
Warwick	W1	2.50
South Wales (anthracite)	SWA1	5.00
	SWA2	4.45
South Wales (steam coal)	SWS1	3.60
	SWS3	8.20
South Wales (bituminous)	SWB1	5.10
Kent	K1	4.20
All Collieries		4.14

From Jacobsen *et al.* (1971)
[a] See also Table 6

Cram and Glover (1995) examined dust sampling data from New South Wales, Australia. During the period 1984–1995, 8% of the 8449 samples from longwall faces exceeded 3 mg/m^3; closer examination of the data by the authors revealed that although 10–20% of the samples exceeded 3 mg/m^3 in the 1980s, only 3–5% did so in the early 1990s. The pattern was similar for continuous miner faces; overall, 1.5% of samples exceeded this threshold value, with a trend from about 3% in the 1980s to less than 1% in the 1990s. Four mines in Queensland, Australia, which also used longwall methods, had mean dust concentrations for coalface work ranging from 1.6 to 3.5 mg/m^3 in 1992–94 (Bofinger *et al.*, 1995).

Dust levels have been reduced in the last 20 or so years in some countries following regulatory action. For instance, exposures in the United States prior to regulatory action (survey data 1968–69) were more than twice as great as those immediately following regulatory action in 1970, when the dust limit was provisionally set at 3 mg/m^3.

Furthermore, in 1977, four years after the dust limit had been set to 2 mg/m^3, exposures had dropped to a fifth of the levels experienced in 1968–69 (Parobeck & Jankowski, 1979). More recent information, for 1978–92, reveals that the progress made in reducing the level of dust was apparently maintained (Watts & Niewiadomski, 1990; United States National Institute for Occupational Safety and Health, 1995). For example, the mean dust concentration for continuous mine operators (workers at the coalface) was 6.8 mg/m^3 prior to 1969 (Parobeck & Jankowski, 1979) and 1.3 mg/m^3 from 1988 to 1992 (United States National Institute of Occupational Safety and Health, 1995). Similarly, in Germany, data from different time periods indicate a continuing trend to lower dust levels (see **Table 18**). Soutar *et al.* (1993) reported some data from three British mines that show a similar tendency, with dust levels prior to 1970 being about 3.0, 3.5, and 5.0 mg/m^3 for the three mines but less than 2 mg/m^3 for each thereafter.

Table 18. Respirable dust concentrations in the return air of coal-faces in different coal seams in the Ruhr, Germany, in 1955 and in 1963–1971

Location and survey period	Dust concentration[a] (mg/m^3)
Dorsten, Horst, Essen	
1955	7
1963–67	5.6
Bochum	
1955	17
1963–67	10.1
Witten, Sprockhovel	
1955	23
1963–67	11.8

From Reisner *et al.* (1982)
[a]Converted from particle counts

Table 19. Mean and maximal respirable dust concentrations for miners in three German underground mines, 1974–91

Dust level (mg/m^3)	Heinrich Robert (high rank)	Walsum (low rank)	Saar (special low rank)[a]
Mean	2.9	2.3	1.6
Maximum	5.0	5.1	3.7

From Morfeld *et al.* (1997)
[a]Time period is 1980–91 for this mine

Table 20. Airborne total dust levels in the working zone of miners engaged in underground coal transport in the former USSR

Mine	Occupation	No. of samples	Dust concentration (mg/m^3)	
			Range	Mean
Ayutinskaja	Operators of underground reloaders on slopes	48	9.5–69.7	37.0
	Underground machine operators	16	4.1–13.8	6.4
	Electric locomotive drivers	13	9.5–64.6	26.0
	Operators of tipping equipment	36	6.4–83	37.0
	Miners engaged in belt-conveyor cleaning	12	17.5–113	43.7
Krasnyj Partizen	Operators of underground equipment on belt inclines	145	6.7–111	79.0
	Electric locomotive drivers	6	–	46.0

From Elez *et al.* (1985)

In underground mines in the United States, compliance samples are collected by mine operators. These are then forwarded to the responsible government agency for weighing and processing. Bias has long been suspected in these samples (Boden & Gold, 1984), and has been investigated (Seixas *et al.*, 1990). Recently, following the discovery of samples that appeared to have suffered from operator tampering, a special sampling study was undertaken (Mine Safety and Health Administration, 1992). This revealed evidence of underestimation of dust levels in small mines but not in large mines. Attfield and Hearl (1996) investigated the implications of this previously unknown bias for epidemiological studies; these authors suggested that the bias may not have impinged greatly on the epidemiological findings, since the studies had involved larger coal mines.

1.3.2 *Surface mines*

Although dust levels in surface mines are generally lower than those at underground mines, there are several jobs that put workers at risk for silica exposure and silicosis. **Table 21** presents the mean mixed dust levels for the 10 dustiest jobs at surface mines in the United States for the period 1981–86 (Piacitelli *et al.*, 1990). Workers involved in drilling received respirable quartz exposures of approximately 0.33 mg/m^3, which was about three times the average for all workers.

Piacitelli *et al.* (1990) also calculated average mixed dust concentrations at surface coal mines in the United States between 1982 and 1986. When preparation plants and miscellaneous jobs were included, these averaged about 0.7 mg/m^3. Quartz concentrations for the same time period and jobs had a mean of 0.11 mg/m^3.

Data from a study of British surface (opencast) coal miners (nine sites) concur fairly well with those from the United States (Love *et al.*, 1992). The mixed respirable dust samples had geometric mean exposures of less than 1 mg/m^3 for all jobs. Respirable concentrations of quartz were less than 0.1 mg/m^3 (geometric mean). Drill operators had

the highest mixed dust concentration (0.96 mg/m^3 geometric mean) and the highest quartz exposure (0.1 mg/m^3 geometric mean).

Table 21. The ten dustiest (respirable quartz) jobs at surface coal mines in the United States (1982–86)

Job	No. of samples	Average (mg/m^3)	Standard deviation
Highwall drill helper	53	0.36	0.94
Highwall drill operator	683	0.32	0.47
Rock drill operator	21	0.29	0.22
Bulldozer operator	608	0.17	0.25
Pan scraper operator	71	0.11	0.14
Refuse truck driver	329	0.07	0.07
Coal truck driver	33	0.06	0.06
Crusher attendant	34	0.06	0.18
Highlift operator	304	0.05	0.07
Coal sampler	44	0.04	0.04

From Piacitelli *et al.* (1990)

Borisenkova *et al.* (1984) took 162 air samples at the Kansk-Achinsk opencast mine in the former USSR. The mean dust concentrations in operators' cabins were 0.2–4.8 mg/m^3 (average, 2.2 mg/m^3), and 1.6–11.9 mg/m^3 (average, 8.8 mg/m^3) on the platform of the transport belt excavator. The total dust (19–36% respirable fraction) contained 3.5% free crystalline silica.

In some Hungarian surface mines, all dust samples were greater than 1 mg/m^3, with 70% > 8 mg/m^3 (Kohegyi & Karpati, 1986). Mixed respirable dust levels in some surface mines in Yugoslavia (Ivanovic *et al.*, 1988) ranged from about 1 mg/m^3 in winter to > 6 mg/m^3 in summer.

1.3.3 *Other exposures*

Other than in mining, exposure to coal dust can also occur during bulk coal transfer and at sites where coal is used. These sites include power stations, steel and coke works and plants where coal is refined to produce chemicals or liquid fuels. The domestic use of coal for heating is another potential source of exposure to coal dust. However, information on these other exposures to coal dust is limited.

In a study of lignite mining and handling, Lazarus (1983) found the highest respirable dust concentrations in enclosed coal handling areas (mean, 0.7 mg/m^3; range, 0.15–1.17 mg/m^3 across 13 sites). In relatively open areas in the power station, the average respirable dust level was 0.3 mg/m^3 (0.12–0.54 mg/m^3 across three sites.

A study on coal trimmers (loaders of cleaned coal into ships) by Collis and Gilchrist (1928) showed that cleaned coal has fibrogenic properties. These authors were instrumental in showing that coal workers' pneumoconiosis was a different disease from silicosis, since silica exposures among these coal trimmers were minimal.

1.3.4 *Bioaccumulation*

Coal mine dust exposures are typically sufficient to cause substantial dust deposition in the lungs of miners. This dust is captured by macrophages and transported to regions around the small airways, where it is deposited in the form of coal maculae. The dust persists in the lungs for an extensive period of time. In some miners, tissue reactions occur, and coal and/or silicotic nodules develop. In severe cases, progressive massive fibrosis can occur, leading to disability and premature death in some miners.

The pathological appearances of coal miners' lungs have been studied extensively. Most of these studies have concentrated on the relationship between pathological abnormalities and lung dust, the association between pathological abnormalities and radiographic abnormalities, or the relationship between radiographic abnormalities and lung dust. Lack of airborne exposure data in most studies has prevented the comparison of lung dust extent and composition with dust exposures during life, and thus led to limited information on bioaccumulation.

King *et al.* (1956) estimated the lung dust weights for five occupational groups (**Table 22**). Of the five groups, coal miners (coalface workers) had the highest total lung dust weight, this being about eight times greater than that for tin and granite miners. Most of the difference in lung dust weights between occupations was related to the presence of coal dust, the quantity of which varied widely. In contrast, lung quartz, lung kaolin and mica, and lung total silica levels varied little across the occupations, with the exception of rock workers.

Table 22. Lung dust weights (% dry lung) for different occupations

Occupation	No. of men	Mean dust exposure (years)[a]	Total dust (g)	Coal (g)	Quartz (g)	Kaolin plus mica (g)	Total silica (g)
Tin miners	15	23	4.0 ± 0.4[b]	1.8 ± 0.3	0.7 ± 0.1	1.5 ± 0.2[c]	1.4 ± 0.2
Rock workers	9	37	20.2 ± 4.9	11.1 ± 2.4	2.5 ± 0.6	6.6 ± 2.0	5.7 ± 1.6
Hauliers, etc.	10	38	10.6 ± 2.5	7.0 ± 1.6	1.3 ± 0.7	2.3 ± 0.9	2.2 ± 1.0
Unclassified	18	34	17.4 ± 2.4	13.5 ± 1.8	1.1 ± 0.2	2.8 ± 0.6	2.4 ± 0.5
Coal miners	28	33	34.7 ± 6.2	31.1 ± 5.8	0.9 ± 0.1	2.6 ± 0.4	2.2 ± 0.3

From King *et al.* (1956)
[a] Years worked in underground jobs
[b] Standard error of mean
[c] Contains also feldspar

Bergman and Casswell (1972) tabulated the lung dust composition of coal miners with the rank of the coal in which the miners had worked. As shown in **Table 23**, they found that the percentage of coal in the lung increased with coal rank, while the percentage of quartz in total dust and in non-coal dust decreased with coal rank. However, as noted earlier, the same relationships apply to airborne dust exposures. It is therefore not clear to what extent these observations reflect different patterns of depo-

sition and retention for the various components, or whether they are just a reflection of the underlying dust composition in the inhaled air.

Table 23. Average lung dust composition in different regions of the United Kingdom

Coalfield	Number of lungs	Rank factor (% carbon)[a]	Lung dust composition		
			Coal in total dust (%)	Quartz in total dust (%)	Quartz in non-coal dust (%)
South Wales (high rank)	37	92.4	84.3	2.02	13.2
South Wales (low rank)	27	90.2	77.1	3.20	14.0
Northumberland and Durham	16	88.2	83.9	2.51	16.1
Yorkshire	12	85.9	56.9	7.05	17.3
North Western	13	84.5	60.5	7.20	16.8
Scotland	19	83.4	85.5	2.13	14.1
West Midlands	14	83.1	57.9	7.67	19.8
East Midlands	15	83.1	37.0	12.78	20.1

From Bergman and Casswell (1972)
[a] Percentage carbon in dry mineral-matter-free coal

Only one study exists that has both measured airborne exposures and retained lung dusts (Douglas et al., 1986). Linear regression analysis, based on 430 cases, relating retained lung dust to respirable dust exposure (gh/m3) showed that miners with increasing severity of pneumoconiosis had apparently retained progressively more dust per unit of exposure. The same was true for the ash component of the dust. There were no obviously consistent trends across coal rank groups. Mean ratios of percentage lung dust to percentage exposure for ash and quartz are shown in **Table 24** by pneumoconiosis severity and coal rank group. It can be seen that there is a tendency for both the ash and quartz ratios to increase with coal rank and with pneumoconiosis status. It is therefore apparent that the findings of Bergman and Casswell (1972) reflect both the innate composition of the airborne dust together with a tendency for greater deposition and/or retention of ash and quartz in the lower rank coals.

1.4 Regulations and guidelines

Occupational exposure limits and guidelines for some countries are presented in **Table 25**. Exposure limits cannot be compared directly from country to country because of differences in measurement strategies. The World Health Organization (WHO) (1986) has recommended a 'tentative health-based exposure limit' for respirable coal mine dust (with < 7% respirable quartz) ranging from 0.5 to 4.0 mg/m^3. WHO recommended that this limit be based on (i) the risk factors (i.e. coal rank or carbon content, proportion of respirable quartz and other minerals, and particle size distribution of the coal dust) for

Table 24. Mean values for the ratio of percentage lung dust component to the percentage of the same component in respirable coal mine dust divided by pathological and coal-rank groups

Component	Pathological group	Coal rank group[a]			
		A	B	C	D
Ash[b]	M	0.80	0.92	0.79	0.93
Quartz		0.99	1.23	1.44	1.45
Ash	F	0.82	1.06	1.24[c]	1.10
Quartz		1.16	1.46	2.16[c]	1.66
Ash	PMF	0.87	1.09	1.21[c]	1.33[d]
Quartz		1.27	1.47	2.08[c]	2.35[d]
Residual mean squares	Ash = 0.14 Quartz = 0.59	(418 degrees of freedom)			

M, minimal evidence of fibrosis; F, fibrotic dusted lesions 1–9 mm in diameter; PMF, progressive massive fibrosis (fibrotic dusted lesions ≥ 10 mm in diameter)
From Douglas *et al.* (1986)

[a] A, 91.4–94.0% carbon; B, 88.8–90.6% carbon; C, 85.2–87.0% carbon; D, 81.1–85.5% carbon

[b] Ash is the non-coal mineral portion of the exposure dust of which quartz is a component

[c] Difference from next M group $p < 0.05$

[d] Difference from F group $p < 0.05$

coal workers' pneumoconiosis category 1 that are determined at each mine, and (ii) the assumption that the risk of progressive massive fibrosis over a working lifetime (56 000 h) will not exceed 2/1000. Based on the WHO approach, the risk of disease would be determined separately for each individual mine or group of mines, and the exposure limit would vary from mine to mine (United States National Institute for Occupational Safety and Health, 1995).

United States coal mine operators are required to take bimonthly samples of airborne respirable dust in the active workings of a coal mine with an approved device. The measured concentration is multiplied by a conversion factor of 1.38 to adjust for differences in sampling devices used in the United States (a 10 mm nylon cyclone) and the United Kingdom (a horizontal elutriator developed by the British Mining Research Establishment). The respirable particulate size fraction is defined by the British Medical Research Council criterion for particle-size selective dust samples as '100% efficiency at 1 micron or below, 50% at 5 microns, and zero efficiency for particles of 7 microns and upward' (United States National Institute for Occupational Safety and Health, 1995).

Table 25. Occupational exposure limits and guidelines for respirable coal mine dust in various countries[a]

Country	Recommended value (gravimetric) (mg/m^3)	Comment	Interpretation
Australia	3	Coal dust with ≤ 5% respirable free silica	TWA
Belgium	10 /(% respirable quartz + 2)		TWA
Brazil	8/(% respirable quartz + 2)		TWA
Canada			TWA
Québec[b]	2	< 5% crystalline silica	
Ontario[c]	4	total dust	
	2	respirable dust	
Finland	2.0	Coal dust	MAK
	0.2	Quartz (fine dust < 5 μm)	
	0.1	Silica: cristobalite, tridymite	
France[d]	5 (alveolar)	Coal dust without silica	VLns
	10 (inhalable dust)	Coal dust without silica	
Germany	0.15	Quartz (including cristobalite and tridymite)	MAK
	4.0	Fine dust containing quartz (≥ 1% quartz by weight)	
Italy	3.33	Coal dust with < 1% quartz	TWA
	10/(q + 3) where q = % of quartz (mass)	Coal dust with > 1% quartz	TWA
Netherlands	2	Coal dust (less than 5% respirable quartz)	TWA
	0.075	Silica: cristobalite, tridymite	
Sweden	0.05	Silica: cristobalite, tridymite	TWA
United Kingdom	3.8	Coal mine dust (average concentration at the coalface)	TWA
United States			
MSHA	2.0	Coal dust with < 5% silica	
	10/(% SiO_2)	Coal dust with > 5% silica	
	10/(% respirable quartz + 2)	Silica: quartz	
	Half of the value for quartz	Silica: cristobalite, tridymite	
ACGIH[e] (TLV)	2	Respirable fraction of particulate matter containing < 5% crystallline silica	TWA

Table 25 (contd)

Country	Recommended value (gravimetric) (mg/m^3)	Comment	Interpretation
United States (contd)			
OSHA[f] (PEL)	2.4/(% silica + 2) 10/(% silica + 2)	Respirable fraction < 5% silica Respirable fraction > 5% silica	TWA
NIOSH[e] (REL)	1		TWA

TWA, time-weighted average; MAK, maximum workplace concentration; VLns, limit value, dust with no specific effect; MSHA, United States Mine Safety and Health Administration; ACGIH, American Conference of Governmental Industrial Hygienists; TLV, threshold limit value; OSHA, United States Occupational Safety and Health Administration; PEL, permissible exposure limit; NIOSH, United States National Institute for Occupational Safety and Health; REL, recommended exposure limit;
[a]From United States National Institute for Occupational Safety and Health (1995) except where specified. See also the monograph on silica in this volume.
[b]From Anon. (1995)
[c]Anon. (1994)
[d]From Ministère du Travail et des Affaires Sociales (1996)
[e]From American Conference of Governmental Industrial Hygienists (ACGIH) (1995)
[f]From United States Occupational Safety and Health Administration (OSHA) (1995)

2. Studies of Cancer in Humans

The Working group reviewed numerous epidemiological reports of cancer risks among persons exposed to coal dust. These studies were predominantly cohort mortality studies among coal miners throughout the world. Also considered, although given less emphasis by the Working Group, were case series, autopsy studies, and community based case–control studies where coal dust exposure was not a principal focus. The majority of evidence pertained to cancers of the lung and stomach. Several studies provided information on the possible roles of pulmonary fibrosis and impaired function as risk indicators.

2.1 Case reports and descriptive studies

Autopsy studies of the prevalence of lung cancer among coal miners have not indicated an association with coal mine dust. James (1955) reported a lower prevalence of lung cancer at autopsy among 1827 coal miners (3.3%) compared to a sample of 1531 non-mining men (5.4%) in South Wales, United Kingdom. Moreover, lung cancer was less prevalent among the subset of 860 coal miners with massive pulmonary fibrosis (1.4%) than among 967 cases of simple pneumoconiosis (5.1%). Goldstein and Webster (1972) reported the prevalence of lung neoplasms at autopsy in 3100 Bantu and 222 white South African coal miners. Coal dust exposures averaged 3.9 mg/m^3 at the surface and 12.5 mg/m^3 at cutting operations; intermediate level exposures occurred in drilling,

loading and other miscellaneous operations. Among 562 Bantu coal miners with dust lesions at autopsy consistent with pneumoconiosis, four (0.7%) had lung cancers compared with six (0.2%) of 2538 Bantu coal miners without dust lesions. The corresponding numbers of lung cancers in white coal miners were 3/64 (4.7%) with dust lesions and 6/158 (3.8%) without dust lesions.

Several descriptive population surveys of cancer mortality in coal mines have been conducted in England and Wales (United Kingdom) and the United States. Kennaway and Kennaway (1953) reported lower mortality rates among coal miners during 1921–38 for lung cancer (rate ratios, 0.44–0.72) and laryngeal cancer (rate ratios, 0.44–0.73) compared to national rates for men aged 20 years and older in England and Wales; coal miners had experienced similar secular trends as the national population. Stocks (1962) found consistently elevated stomach cancer rates among miners aged 20–64 compared to non-miners in an analysis of mortality data among men in nine counties in England and Wales during 1949–53. In this study, average annual age-adjusted mortality rate excesses among miners, expressed as rate differences, ranged from 65 to 226 per million. Acheson *et al.* (1981) found a statistically significant excess of nasal cancer incidence among miners and quarrymen in England and Wales during 1963–67. The standardized incidence ratio (SIR) for coal miners was 1.60 (48 observed; [95% confidence interval (CI), 1.18–2.12]), with the highest risk detected for coalface workers (22 observed; SIR, 4.30; [95% CI, 2.69–6.5]) and a smaller, non-significant excess among underground workers (30 observed; SIR, 1.32; [95% CI, 0.89–1.88]).

Using data on deaths in 1950 in working men in the United States aged 20–64, Enterline (1964) estimated cause-specific standardized mortality ratios (SMRs) among coal miners. Mortality from all causes in coal miners was approximately twice that of other employed men. A large excess of deaths was reported from non-malignant respiratory disease, which included 321 deaths from pneumoconiosis (487 observed; SMR, 4.91 [95% CI, 4.99–5.38]). The SMR for all cancers was elevated (764 observed; SMR, 1.79; [95% CI, 1.66–1.92]). In addition, mortality excesses were observed for numerous site-specific cancers, including lung (161 observed; SMR, 1.92; [95% CI, 1.63–2.24]), stomach (146 observed; SMR, 2.75; [95% CI, 2.33–3.24]), buccal cavity and pharynx (21 observed; SMR, 1.31; [95% CI, 0.81–2.01]), intestine and rectum (78 observed; SMR, 1.32; [95% CI, 1.04–1.65]), prostate (35 observed; SMR, 2.06; [95% CI, 1.43–2.86]), kidney (22 observed; SMR, 2.00; [95% CI, 1.25–3.03]), urinary bladder (24 observed; SMR, 1.71; [95% CI, 1.1–2.55]), leukaemia and aleukaemia (30 observed; SMR, 1.50; [95% CI, 1.01–2.14]) and lymphosarcoma (47 observed; SMR, 1.68; [95% CI, 1.23–2.23]). When the analysis was restricted to ages 20–59, the SMRs remained elevated but were slightly lower; the SMRs for lung cancer and stomach cancer for this age group were 1.64 and 2.36, respectively [observed numbers not given].

A proportionate mortality ratio (PMR) analysis of death certificates from England and Wales during 1970–72 showed an increased risk for coal miners of stomach cancer (252 deaths; PMR, 1.71 [95% CI, 1.51–1.93]) and lung cancer (843 deaths; PMR, 1.15 [95% CI, 1.07–1.23]) (Office of Population Censuses and Surveys, 1978). In a similar analysis of 1979–80 and 1982–90 death certificates, Coggon *et al.* (1995) reported decreased

mortality from lung cancer among coal miners (4610 deaths; PMR, 0.92; 95% CI, 0.89–0.94). Morality from stomach cancer was not significantly different from expected [detailed results not presented for stomach cancer].

In a cohort study of approximately 300 000 United States veterans followed during 1954–80, Hrubec *et al.* (1995) recorded nine stomach cancer deaths among 777 coal miners; the smoking-adjusted relative risk was 1.9 (90% CI, 1.10–3.32). The corresponding relative risk for respiratory cancer was 1.3 (26 deaths; 90% CI, 0.91–1.74). In this study, industry and occupation were determined by questionnaire in 1954.

Several ecological studies have not lead to clear conclusions about stomach cancer mortality and exposure to coal dust and employment in the coal mining industry (e.g. Matolo *et al.*, 1972; Creagan *et al.*, 1974; Klauber & Lyon, 1978).

2.2 Cohort studies

Goldman (1965) presented data from a mortality survey of miners and ex-miners employed by the National Coal Board in the United Kingdom. For men aged 20–65 in 1955, the SMRs (relative to rates in England and Wales) among underground workers were 0.70 (216 observed; [95% CI, 0.61–0.80]) for lung cancer and 1.02 (459 observed; [95% CI, 0.93–1.12]) for all other neoplasms; among surface workers, the SMR for lung cancer was 0.92 (54 observed; [95% CI, 0.69–1.19]) and the SMR for other neoplasms was 1.13 (93 observed; [95% CI, 0.92–1.39]). For all coal miners, the SMR for lung cancer was 0.74 (270 observed; [95% CI, 0.65-0.83]). Geographical analyses of the SMRs for lung cancer revealed higher rates in the North than in the South-West, with SMRs ranging from 0.63 to 1.47. SMRs for all neoplasms ranged from 0.61 in Kent to 1.29 in the North.

As part of the same study, Goldman (1965) also reported on lung cancer mortality among 5096 male coal miners and ex-miners aged ≥ 35 years from the Rhondda Fach area in Glamorgan. A lower than expected lung cancer mortality risk was found for the period 1951–56 (30 observed; SMR, 0.81 [95% CI, 0.55–1.16]). Analyses were performed separately for various radiographic categories of pneumoconiosis: the SMR for lung cancer for miners with grade 0 was 0.87 (16 observed; [95% CI, 0.50–1.41]); the SMR for miners with grades 1–3 pneumoconiosis was 0.57 (6 observed; [95% CI, 0.21–1.24]); and the SMR for miners with progressive massive fibrosis was 1.00 (8 observed; [95% CI, 0.43–1.96]).

Boyd *et al.* (1970) reported on a proportionate mortality study of lung cancer, for the years 1948–67, in coal miners aged 15 years and older in Cumberland, United Kingdom. Compared with local non-mining mortality distributions, the authors detected a deficit of lung cancer mortality in the cohort of underground miners (28 observed; PMR, 0.79 [95% CI, 0.53–1.15]); no such deficit was found for surface workers (11 observed; PMR, 0.99 [95% CI, 0.49–1.77]). For the combined group of coal miners, the PMR for lung cancer was 0.84 (39 observed [95% CI, 0.60–1.15]). The PMRs for all other cancers were 1.04 (117 observed) for underground miners and 0.98 (33 observed) for surface workers. All of these PMRs were slightly lowered when comparisons were made based on national mortality distributions.

Rooke *et al.* (1979) presented proportionate mortality findings for lung cancer among 1003 deaths that occurred among coal miners in North-West England, United Kingdom, during 1974–76. The PMR for lung cancer was 1.17 (114 observed; $p > 0.05$) for the entire group. Separate results were given for coal miners without pneumoconiosis (62 observed; PMR, 1.29; $p < 0.05$), for those with simple pneumoconiosis (24 observed; PMR, 1.25; $p > 0.05$) and miners with complicated pneumoconiosis (28 observed ; PMR, 0.92; $p > 0.05$).

Enterline (1972) followed a cohort of 533 male coal miners in West Virginia, in 1937. Follow-up was from 1938 to 1966 and mortality comparisons were made against rates for men in the United States. Overall, there were 140 deaths in this cohort during this time (SMR, 1.58; [95% CI, 1.33–1.86]). The author reported SMRs for all malignant neoplasms (15 observed; SMR, 1.22; [95% CI, 0.68–2.01]), digestive system cancers (8 observed; SMR, 2.10; [95% CI, 0.91–4.25]), respiratory system cancers (4 observed; SMR, 1.11; [95% CI, 0.3–2.85]) and all other cancers (3 observed; SMR, 0.61; [95% CI, 0.13–1.79]).

Liddell (1973) reported 5362 deaths in 1961 among coal miners aged 20–64 whose employment history was identified by the National Coal Board in the United Kingdom. There was a lower percentage of deaths from lung cancer (8.8%) among coal miners than among men nationally (13.2%). The percentage of deaths due to lung cancer increased from 2.4% in coal miners who were last employed before 1950 to 10.4% in coal miners who were last employed in 1960–61. Among 3239 deaths who were last employed in 1961, there were deficits, relative to national rates, in lung cancer in face workers (SMR, 0.49), other underground workers (SMR, 0.53) and surface workers (SMR, 0.82). The SMRs for stomach cancer among these subsets of coal miners were 1.01, 1.28 and 0.32, respectively. The lung cancer deficits were not counterbalanced by excesses in mortality from neoplasms other than lung and stomach cancers; the SMRs for other neoplasms, by worker subgroup, were 0.69, 0.72 and 1.01, respectively. Mortality from pneumoconiosis was consistently elevated, whereas mortality from cardiovascular diseases was lower than national rates [95% CI cannot be calculated].

Ortmeyer *et al.* (1974) conducted a mortality follow-up of 2549 miners employed in 1963–65 and 1177 ex-miners from the Appalachian region of the United States. All were men who had been randomly selected to participate in a pneumoconiosis survey by the United States Public Health Service. Mortality was determined for 1963–71; vital status was ascertained for 95% of employed miners and 99% of ex-miners. Compared to United States mortality rates, the SMR for all causes for employed miners was 0.93 (225 observed; [95% CI, 0.81–1.06]) and for ex-miners 1.19 (308 observed; [95% CI, 1.11–1.39]). The largest excesses were found among miners with complicated pneumoconiosis. Among employed miners within this subcohort, the SMR for all causes was 1.32 (15 observed; [95% CI, 0.73–2.17]); among ex-miners the SMR was 1.59 (39 observed; [95% CI, 1.13–2.17]). Among miners with complicated pneumoconiosis, years underground was only related to increased mortality in employed miners. Among ex-miners the largest excess was found with 29 years or less underground mining (14 observed; SMR, 2.21; $p < 0.05$) compared with mining for more than 30 years (25 observed; SMR,

1.38; $p < 0.05$). In a separate analysis of lung cancer in the same cohort, Costello *et al.* (1974) found a deficit of lung cancer mortality (24 observed; SMR, 0.67; [95% CI, 0.43–0.99]) compared to national rates.

Rockette (1977) performed a cohort mortality study of 23 232 United States coal miners who represented a 10% sample of men covered by the United Mine Workers Health and Retirement Funds as of 1959. Follow-up was conducted for the years 1959–71. Vital status was determined for over 99% of the cohort and death certificates were obtained for all 7741 deaths. Mortality comparisons were made against United States rates. Mortality from all causes in this cohort was nearly identical to national rates (7741 observed; SMR, 1.02 [95% CI, 0.998–1.04]) as was mortality from all cancers (1243 observed; SMR, 0.99; [95% CI, 0.94–1.05]). The authors detected mortality excesses for the non-malignant respiratory diseases category (752 observed; SMR, 1.59; [95% CI, 1.48–1.70]), especially pneumoconiosis (188 observed; SMR, 9.26; [95% CI, 7.98–10.68]), and for ill-defined causes (164 observed; SMR, 1.79; [95% CI, 1.52–2.08]). Mortality was also in excess among coal miners for stomach cancer (129 observed; SMR, 1.40; [95% CI, 1.17–1.66]) and lung cancer (352 observed; SMR, 1.13; [95% CI, 1.02–1.26]). The stomach cancer excess was larger among coal miners who were pensioners at the beginning of follow-up (85 observed; SMR, 1.56; [95% CI, 1.24–1.93]) than among non-pensioners (44 observed; SMR, 1.17; [95% CI, 0.85–1.56]); the lung cancer SMRs were nearly identical for these subcohorts.

Cochrane *et al.* (1979) conducted a mortality follow-up study among residents of the Rhondda Fach coal mining community in Wales, United Kingdom. The population was examined in 1950–51 and was composed of 6212 male miners and ex-miners and 2138 male non-miners, aged ≥ 20 years. Follow-up was carried out for the period 1950–70, and mortality comparisons were made against rates for England and Wales. Among miners and ex-miners combined, there were elevations of mortality from all causes; SMRs ranged from 1.16 to 1.95 among miner and ex-miner groups classified by radiographic category of pneumoconiosis (1953 International Labour Office (ILO) classification: four categories of simple pneumoconiosis 0, 1, 2, 3 and categorized large shadows according to the size (A, B, C)), with the largest excess (467 observed; SMR, 1.95) occurring among those with category B,C. In this later group, an approximately twofold excess of mortality from all causes occurred both in miners (66 observed; SMR, 2.10) and ex-miners (401 observed; SMR, 1.93). In contrast, mortality from all causes in non-miners was not elevated (357 observed; SMR, 0.99). No excesses were found for lung cancer in either miners or non-miners: SMRs for non-miners and miners with radiographic categories 0, 1–3 and A–C were 0.66 (21 observed), 0.70 (57 observed), 0.68 (33 observed) and 0.80 (23 observed), respectively. Stomach cancer mortality was elevated in all groups; the SMRs for the aforementioned groups were 1.13 (13 observed), 1.60 (52 observed), 1.08 (21 observed) and 1.84 (23 observed), respectively. Mortality from all other malignancies combined was lower than national rates for miners and non-miners.

An extended follow-up of the Rhondda Fach population through 1980 yielded generally similar results (Atuhaire *et al*, 1985, 1986). Mortality from all causes was not elevated in non-miners (637 observed; SMR, 0.99), whereas miners experienced

excesses, especially those with radiographic category B,C (567 observed; SMR, 1.98; 95% CI, 1.82–2.15). The SMRs for lung cancer for non-miners and miners with radiographic categories 0, 1–3, A and B,C were 0.70 (43 observed), 0.77 (100 observed), 0.77 (60 observed), 0.69 (12 observed) and 0.91 (19 observed), respectively. The corresponding SMRs for stomach cancer were 1.31 (24 observed), 1.52 (69 observed; $p < 0.05$), 1.23 (33 observed), 2.17 (14 observed; $p < 0.05$) and 1.51 (13 observed). A case–control analysis of 37 stomach cancer deaths among ex-miners and 148 age-matched ex-miner controls did not reveal any differences in years worked at the coalface (mean for cases 14.7 years, mean for controls 14.5 years; $p > 0.50$) or years worked underground (22.1 versus 21.3; $p > 0.50$) (Atuhaire *et al.*, 1986).

Armstrong *et al.* (1979) conducted a mortality study of 213 male coal miners in Western Australia during the years 1961–75. Follow-up was not possible for 318 additional coal miners whose records had been lost. All but eight (3%) of the 213 coal miners worked underground, and 99.5% had at least 10 years of mining experience. Vital status was determined for 210 of 213 (99%) coal miners. Smoking habits were also determined; 20.7% had never smoked and 17.4% smoked 25 or more cigarettes per day. Compared to rates in Western Australian men, miners had an excess of mortality from all causes (54 observed; SMR, 1.24; 95% CI, 0.93–1.62). There was a deficit of lung cancer mortality (1 observed, SMR, 0.2). However, an excess of non-respiratory cancer mortality was noted (17 observed; [SMR, 3.04; 95% CI, 1.77–4.86]), due mainly to stomach cancer (2 observed; [SMR, 2.22; 95% CI, 0.27–8.03]), colorectal cancer (3 observed; [SMR, 3.0; 95% CI, 0.62–8.77]), pancreatic cancer (2 observed; [SMR, 3.33; 95% CI, 0.41–12.04]) and melanoma of the skin (3 observed, [SMR, 15; 95% CI, 0.31–43.83]).

A series of nested case–control studies among United States coal miners addressed associations of coal dust exposure and cigarette smoking with cancers of the lung and stomach. Ames and Gamble (1983) conducted a nested case–control study of 46 stomach cancers and 46 age-matched lung cancers identified from among approximately 20 000 coal miners constructed from four United States cohorts. Controls consisted of 92 coal miners matched on age and year of death who had died from cancers other than of the lung or stomach or from other causes except cancer and accidents. Employment for 25 years or longer as an underground coal miner was associated with elevated risks for stomach cancer (odds ratio, 1.55; 95% CI, 0.76–3.17) and lung cancer (odds ratio, 1.42; 95% CI, 0.70–2.89). These associations were both restricted to workers with ≥ 30 years history of smoking (3.52; 1.11–11.7) and (2.25; 0.92–5.49) respectively. The stomach cancer risk related to years underground was confined to workers with functional evidence of airways obstruction (forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) < 70% predicted; odds ratio, 3.64; 95% CI, 0.62–21.4). In contrast, the association of years worked underground with lung cancer was not modified by pulmonary function. A further analysis of these data (Ames, 1983) indicated a negative association of stomach cancer with radiographic evidence of coal workers' pneumoconiosis (odds ratio, 0.43; 90% CI, 0.18–1.05). [The Working Group noted that the study base and overlap of cases within and between the cohorts is unkown. The Working Group also noted that the number of cases and controls excluded is unknown and therefore the representativeness of the sample is unknown.]

In a larger nested case–control study of lung cancer, Ames *et al.* (1983) compared employment history and smoking habits between 317 white lung cancer death cases and two control groups. The control groups consisted of one-to-one matched coal miners who died from conditions other than cancer or accidents, matched to cases on age and year of birth and two-to-one matched deaths other than cancer and accidents who were further matched to cigarette smoking status. Compared to the first control group, the odds ratio for ≥ 25 years of underground mining was 1.18 (95% CI, 0.86–1.62); the corresponding odds ratio relative to exposures in the second control series was 0.89 for ≥ 25 years of underground mining (95% CI, 0.66–1.20). The effect estimates for years underground did not differ significantly when the data were stratified by years as a smoker.

A mortality follow-up study of 26 363 male coal miners from 20 collieries in England and Wales, United Kingdom, was conducted by Miller and Jacobsen (1985). Dust-exposure reconstruction permitted mortality to be analysed in relation to cumulative exposure (gh/m^3) for 19 550 (74%) members of the cohort. Workers were classified by radiographic categories of pneumoconiosis using the 1953 ILO system, and vital status during 1953–79 was ascertained for 24 736 (94%) miners. Overall, mortality from all causes was lower than national rates, with individual coal mine SMRs ranging from 0.74 to 0.99. However, there was an upward trend in relative mortality from 1953–72 to 1973–79. Excluding violent deaths, 22-year survival estimates in miners aged 25–64 were considerably lower among those with progressive massive fibrosis (PMF) (categories A–C) than miners with simple pneumoconiosis (categories 1–3) or no radiographic abnormality (category 0). Mortality from cancers of the digestive organs and peritoneum (318 in total, of which 274 were stomach cancers) was generally unrelated to cumulative exposure. Among men aged 35–64 at entry, lung cancer mortality rates were 18% and 26% lower in men with simple pneumoconiosis and for those with PMF, respectively, than among miners with category 0. [The Working Group noted the absence of site-specific cancer mortality data, which limited the interpretation of the results.]

Meijers *et al.* (1991) conducted a mortality follow-up study of 334 coal workers' pneumoconiosis cases diagnosed in the Netherlands during 1956–60. Follow-up was through to 1983. Compared to national rates, mortality from all causes was elevated (165 observed; SMR, 1.53; $p < 0.05$), as was mortality from all cancers combined (56 observed; SMR, 1.63; $p < 0.05$) and from non-malignant respiratory disease (31 observed; SMR, 4.26; $p < 0.05$). A large excess was detected for cancer of the stomach and small intestine (16 observed; SMR, 4.01; [95% CI, 2.29–6.51]), whereas only a small nonsignificant elevation was found for lung cancer (19 observed; SMR, 1.31; [95% CI, 0.79–2.05).

In a larger study in the Netherlands of 3790 coal miners, Swaen *et al.* (1995) followed workers with evidence of some radiographic abnormalities initially detected during the 1950s. Follow-up was performed through to 1992; vital status was determined for 96% of the cohort, and cause of death was ascertained for 99% of deaths. An excess of mortality from all causes (2941 observed; SMR, 1.27; 95% CI, 1.23–1.32) and excesses of mortality from non-malignant respiratory diseases (761 observed; SMR, 4.11; 95% CI, 3.82–4.41) and small intestine and stomach cancer (120 observed; SMR, 1.47; 95% CI,

1.22–1.76) were observed. No excesses were detected for all cancers combined (668 observed; SMR, 0.97; 95% CI, 0.90–1.04) or for lung cancer (272 observed; SMR, 1.02; 95% CI, 0.90–1.15). The gastric cancer excess was greatest in workers with ≥ 30 years of underground employment (SMR, 1.54; 95% CI, 1.23–1.91). Gastric cancer was also inversely related to pneumoconiosis grade at the initial survey; the SMRs for workers with pneumoconiosis grades 0–1 (other abnormalities), 2–5 (simple pneumoconiosis) and 6–7 (progressive massive fibrosis) were 2.07 (95% CI, 1.24–3.22), 1.47 (95% CI, 1.19–1.81) and 0.99 (95% CI, 0.49–1.76), respectively. [The extent of overlap, if any, between the studies of Meijers *et al.* (1991) and Swaen *et al.* (1995) was not indicated.]

Kuempel *et al.* (1995) reported exposure–response trends among 8878 United States coal miners who had been examined medically in 1969–71 as part of the National Study of Coal Workers' Pneumoconiosis. Mortality follow-up was through to 1979. Exposure data were based on airborne dust measurements made during 1968–72; however, cumulative exposures could only be estimated for the years prior to 1971 because work history data had not been updated. Mortality from all causes was lower than that expected from national rates (793 observed; SMR, 0.85; [95% CI, 0.79–0.91]), although there was an excess of mortality from the 'pneumoconioses and other respiratory diseases' category (68 observed; SMR, 3.72; 95% CI, 2.89–4.71). Mortality from lung cancer (65 observed; SMR, 0.77; [95% CI, 0.60–0.99]) and stomach cancer (8 observed; SMR, 0.91; [95% CI, 0.39–1.80]) was lower than expected. A negative exposure–response trend was found for lung cancer, based on proportional hazards modelling; the SMR in the highest exposure category (127–234 mg-year/m^3) was 0.54 (9 observed), and the rate ratio was estimated as 0.68 (95% CI, 0.36–1.25) for 90 mg-year/m^3, which corresponds to 45 working years at 2 mg/m^3. The dose–response gradient was slightly positive for stomach cancer, but not statistically significant; the SMR for the highest exposure category was 1.64 (3 observed; [95% CI, 0.34–4.79]), and the rate ratio for 90 mg-year/m^3 was 1.19 (95% CI, 0.30–4.78).

[Mortality studies have been conducted in occupational cohorts with exposure to coal dust in settings other than coal miners. However, the Working Group did not consider that these studies (e.g. Howe *et al.*, 1983; Petrelli *et al.*, 1989) provide sufficiently unconfounded assessments of any link between coal dust and cancer.]

2.3 Case–control studies

Swaen *et al* (1985) reported findings from a case–control study of stomach cancer in the Netherlands. The study included 323 male cases diagnosed during 1973–83 from three pathology departments and 323 hospital controls matched on pathology department and date of birth. Employment in coal mining was determined by linkage with the Central Coal Miners Pension Fund; an odds ratio of 1.14 (95% CI, 0.34–1.73) was estimated for past employment as a coal miner. Mean years of underground coal mining among subjects with a history of coal mine employment was 16.8 for cases as compared with 19.7 for controls.

In a follow-up of the above preliminary report, Swaen *et al.* (1987) identified 683 male cases of gastric cancer. An odds ratio for underground coal mine employment and

gastric cancer was 1.15 (95% CI, 0.89–1.47). There was no increased risk of gastric cancer with increased duration of underground coal mining. The average duration of underground mining was 18.8 years for cases and 18 years for controls. [The authors concluded these data do not support the hypothesis that underground coal mining increases the risk of gastric cancer.]

Weinberg *et al.* (1985) performed a case–control study of stomach cancer in the coal mining region of Pennsylvania, United States. One hundred and seventy-eight stomach cancer deaths that occurred during 1978–80 in four western counties of Pennsylvania were matched with three sets of controls, matched on age, race, sex and county of residence. The controls were deaths from other digestive system cancers, deaths from arteriosclerotic heart disease and living controls chosen from the cases' neighbourhoods. Among men, occupation as a coal miner was related to risk for stomach cancer only when cases were compared with other digestive system cancer controls (odds ratio, 1.55; 95% CI, 0.72–3.30). The relative risks associated with coal mining, based on comparisons with heart disease deaths and neighbourhood controls were, respectively, 0.78 (95% CI, 0.39–1.56) and 0.83 (95% CI, 0.37–1.89). There were no female coal miner cases or controls. [The Working Group noted that the choice of the control groups may have biased the results.]

Coggon *et al.* (1990) conducted an incident case–control study of stomach cancer in the Stoke-on-Trent area, United Kingdom. This district had stomach cancer rates that were 80% higher than the national average. Cases consisted of 95 stomach cancer patients (73 men and 22 women) aged 70 years or younger, who were diagnosed during 1985–87. One hundred and ninety sex- and age-matched controls were chosen from the community. Employment in coal mining was associated with an increased risk of stomach cancer, after allowing for the effects of diet (odds ratio, 1.7; 95% CI, 0.8–3.6). The relative risk estimate increased to 2.0 (95% CI, 0.8–4.8) for coal mining employment of five years or more at least 10 years before the interview. There was no association with coal mining employment for shorter or more recent periods (odds ratio, 1.0; 95% CI, 0.3–3.2). However, the risk was greater for employment in the least-dusty jobs within coal mines (odds ratio, 3.6; 95% CI, 1.1–12.2) than for employment in the high dust exposure jobs (underground coal mines, coal mines) (odds ratio, 1.2; 95% CI, 0.5–2.9).

Siemiatycki (1991) carried out a population-based case–control study of cancer among male residents of Montréal, Canada, aged 35–70. This study included histologically confirmed cases of cancer at 11 major sites, newly diagnosed between 1979 and 1985, in 19 major hospitals. With a response rate of 82%, 3730 cancer patients were successfully interviewed. For each site of cancer analysed, the control group was selected from among cases of cancer at the other sites studied (cancer controls). An interview was designed to obtain detailed lifetime job histories and information on potential confounders. Each job was reviewed by a trained team of chemists and industrial hygienists who translated jobs into occupational exposures, using a checklist of 293 common occupational substances. Cumulative exposure indices were created for each substance, on the basis of duration, concentration, frequency and the degree of certainty in the exposure assessment itself, and these were analysed at two levels: 'any' and

'substantial' exposure; the latter was a subset of 'any'. Of the entire study population, 6% had been exposed to coal dust at some time (i.e. lifetime exposure prevalence). The main occupations in which coal dust was attributed in this study were stationary engineers, truck drivers (coal delivery) and coal miners. The odds ratios for stomach cancer were 0.9 (12 exposed cases; 90% CI, 0.5–1.5) for any exposure and 1.5 (8 cases; 90% CI, 0.8–2.8) for substantial exposure. Corresponding odds ratios for lung cancer were 1.3 (63 cases; 90% CI, 1.0–1.9) and 1.1 (27 cases; 90% CI, 0.7–1.7).

There have been other population-based case–control studies in which associations with coal dust exposure have been explored, although none has been as explicit as the studies by Swaen *et al.* (1985), Weinberg *et al.* (1985), Coggon *et al.* (1990) or Siemiatycki (1991) in examining the potential carcinogenicity of coal dust.

Gonzalez *et al.* (1991), in a study from Spain, reported a relative risk for stomach cancer of 11.8 (95% CI, 1.36–103) for ever having been employed in coal mining or coke production. Morabia *et al.* (1992) carried out a hospital-based case–control study in nine metropolitan areas of the United States. A gradient of relative risk for lung cancer was found in relation to years of exposure to coal dust; odds ratios, adjusted for smoking, age, geographical area and asbestos exposure, were 1.3 (95% CI, 0.8–2.0) for < 10 years' exposure and 1.7 (95% CI, 1.1–2.7) for ≥ 10 years' exposure, respectively, compared to never exposed to coal dust. Wu-Williams *et al.* (1993) reported an odds ratio for lung cancer of 1.4 (95% CI, 1.0–1.9) associated with occupational exposure to coal dust among Chinese women.

Cohort, proportionate mortality studies and case–control studies of exposure to coal dust are summarized in **Table 26**.

3. Studies of Cancer in Experimental Animals

3.1 Inhalation exposure

Rat: Two groups of female Sprague-Dawley rats [age and initial numbers unspecified] were exposed by inhalation in chambers to air containing 200 mg/m^3 coal dust [origin of dust and particle size unspecified] or a mixture of coal dust and quartz dust [origin unspecified] (quartz content ensuring that the dust present in the lungs contained about 10% quartz). The duration of exposure was 5 h per day for five days a week, on alternate weeks, for 12, 18 or 24 months. Control rats inhaled air without any added particulate material (room air). Histological examination was performed on the lungs and tumours of the lungs. After 18–24 months, no lung tumours were observed in the 485 controls; after coal dust exposure, the incidence of lung tumours was 4/36 (epidermoid tumours and adenocarcinomas), whereas after combined exposure to coal dust and quartz, the number of lung tumours (epidermoid tumours and adenocarcinomas) was 32/72 (Martin *et al.*, 1977). [The Working Group noted the high dose of coal dust used, the limited reporting concerning the initial number of animals and that a control group using quartz alone was not available.]

Table 26. Cohort, proportionate mortality and case–control studies of exposure to coal dust

Reference/ country	Study base/follow-up	Cancer site/subgroup	Relative risk, PMR, SMR, OR (cases; 95% CI)	Comments
Cohort and proportionate mortality studies				
Goldman (1965) United Kingdom	Miners and ex-miners employed by the National Coal Board, aged 20–65 in 1955	Lung cancer	SMR	
		Underground workers	0.70 (216; [0.61–0.80])	
		Surface workers	0.92 (54; [0.69–1.19])	
	5096 male coal miners ≥ 35 years in Glamorgan, 1951–56	Lung cancer	0.81 (30; [0.55–1.16])	
		Lung cancer occurrence in pneumoconiosis cases by		
		Grade = 0	0.87 (16; [0.50–1.41])	
		Grades = 1–3	0.57 (6; [0.21–1.24])	
Boyd *et al.* (1970) United Kingdom	Coal miners in Cumberland, England, between 1948–67, aged ≥ 15	Lung cancer	PMR	
		Underground workers	0.79 (28; [0.53–1.15])	
		Surface workers	0.99 (11; [0.49–1.77])	
		Combined	0.84 (39; [0.60–1.15])	
Rooke *et al.* (1979) United Kingdom	1003 deaths in coal miners in North-West England, 1974–76	Lung cancer	PMR 1.17 (114; [0.96–1.41])	
		Without pneumoconiosis	1.29 (62; [0.60–1.15])	
		With simple pneumoconiosis	1.25 (24; [0.80–1.86])	
		With complicated pneumoconiosis	0.92 (28; [0.61–1.33])	
Enterline (1972) West Virginia, USA	553 male coal miners in 1937; follow-up 1938–66	All cancers	SMR 1.22 (15; [0.68–2.01])	
		Digestive system	2.10 (8; [0.91–4.25])	
		Respiratory system	1.11 (4; [0.3–2.85])	
Liddell (1973) United Kingdom	3239 deaths in 1961 among coal miners aged 20–64 identified by the National Coal Board	Lung cancer	SMR	There are no observed values reported by cancer type. 95% CI cannot be calculated.
		Face workers	0.49	
		Underground workers	0.53	
		Surface workers	0.82	
		Stomach cancer		
		Face workers	1.01	
		Underground workers	1.28	
		Surface workers	0.32	

Table 26 (contd)

Reference/ country	Study base/follow-up	Cancer site/subgroup	Relative risk, PMR, SMR, OR (cases; 95% CI)	Comments
Cohort and proportionate mortality studies (contd)				
Costello *et al.* (1974) USA	2549 employed miners, 1962–63, 1177 ex-miners from the Appalachian region; follow-up to 1 January 1972	Lung cancer	SMR, 0.67 (24; [0.43–0.99])	
Rockette (1977) USA	23 232 coal miners covered by the United Mine Workers Health and Retirement Funds in 1959; follow-up, 1959–71	All cancers Lung cancer Stomach cancer	SMR 0.99 (1243; [0.94–1.05]) 1.13 (352; [1.02–1.26] 1.40 (129; [1.17–1.66]	
Cochrane *et al.* (1979) Wales, United Kingdom	6212 miners and ex-miners, 2138 non-miners aged ≥ 20 years; follow-up through 1950–70	Lung cancer by radiographic category Non-miners 0 1–3 A–C	SMR 0.66 (21; [0.41–1.00]) 0.70 (57; [0.53–0.91]) 0.68 (33; [0.48–0.98]) 0.80 (23; [0.51–1.2])	
		Stomach cancer by radiographic category Non-miners 0 1–3 A–C	SMR 1.13 (13; [0.60–1.93]) 1.60 (52; [1.19–2.09]) 1.08 (21; [0.67–1.66]) 1.84 (23; [1.17–2.76])	
Atuhaire *et al.* (1985, 1986) Wales, United Kingdom	Extended follow-up of Cochrane *et al.* (1979)	Lung cancer by radiographic category Non-miners 0 1–3 A B,C	SMR 0.70 (43; [0.51–0.94]) 0.77 (100; [0.63–0.94]) 0.77 (60; [0.59–0.99]) 0.69 (12; [0.34–1.20]) 0.91 (19; [0.54–1.41])	

Table 26 (contd)

Reference/ country	Study base/follow-up	Cancer site/subgroup	Relative risk, PMR, SMR, OR (cases; 95% CI)	Comments
Cohort and proportionate mortality studies (contd)				
Atuhaire *et al.* (1985, 1986) (contd)		Stomach cancer by radiographic category	SMR	
		Non-miners	1.31 (24; [0.84–1.95])	
		0	1.52 (69; [1.18–1.92])	
		1–3	1.23 (33; [0.85–1.73])	
		A	2.17 (14; [1.18–3.64])	
		B, C	1.51 (13; [0.81–2.59])	
Armstrong *et al.* (1979) Western Australia	213 male coal miners during 1961–75	Respiratory cancer Stomach cancer	SMR, 0.2 (1) [2.2] (2; [0.27–8.03])	
Ames & Gamble (1983) USA	Four cohorts composed of approximately 20 000 coal miners provided cases of lung and stomach cancer	Lung cancer ≥ 30 years smoking Stomach cancer ≥ 30 years smoking	OR, 1.42 (0.70–2.89) 2.25 (0.92–5.49) 1.55 (0.76–3.17) 3.52 (1.11–11.7)	> 25 years undergound mining (Nested case–control study)
Miller & Jacobsen (1985) England and Wales	26 363 coal miners from 20 collieries, follow-up through 1953–79	Lung cancer smokers *vs* nonsmokers Digestive cancer and cumulative dust exposure	SMR, 5.5 $\chi^2 = 4.07$	
Meijers *et al.* (1991) The Netherlands	334 coal miners' pneumoconiosis diagnosed between 1956–60; follow-up through to 1983	Lung cancer Stomach and small intestine cancer	SMR, 1.31 (19; [0.79–2.05]) 4.01 (16; [2.29–6.51])	
Swaen *et al.* (1995) The Netherlands	3790 coal miners; follow-up through to 1992	Lung cancer Stomach cancer	SMR, 1.02 (272; 0.90–1.15) 1.47 (120; 1.22–1.76)	

Table 26 (contd)

Reference/ country	Study base/follow-up	Cancer site/subgroup	Relative risk, PMR, SMR, OR (cases; 95% CI)	Comments
Cohort and proportionate mortality studies (contd)				
Kuempel *et al.* (1995) USA	8878 coal miners medically examined 1969–71; follow-up through 1979	Lung cancer Stomach cancer	SMR, 0.77 (65; [0.60–0.9]) 0.91 (8; [0.39–1.80])	Exposure–response analysis for lung cancer was negative while the exposure–response gradient for stomach cancer was slightly positive.
Case–control studies				
Swaen *et al.* (1985) The Netherlands	323 male stomach cancer cases; 323 hospital controls	Stomach cancer	OR 1.14 (0.34–1.73)	Matched on pathology department and date of birth
Weinberg *et al.* (1985) USA	178 cancer deaths between 1978 and 1980 in four western Pennsylvania counties; controls were other digestive system cancer deaths	Stomach cancer	OR 1.55 (0.72–3.30)	Matched on age, race, sex and county of residence
Coggon *et al.* (1990) United Kingdom	95 newly diagnosed stomach cancer patients; 190 controls	Stomach cancer > 5 years' coal mining	OR 1.7 (26; 0.8–3.6) 2.0 (19; 0.8–4.8)	Matched on age and sex and adjusted for diet
Siemiatycki (1991) Canada	3730 male cancer patients resident in Montréal, aged 35–70. Six percent exposed to coal dust. 'Substantial' exposure a subset of 'any' exposures	Stomach cancer Any exposure Substantial exposure Lung cancer Any exposure Substantial exposure	OR 0.9 (12; 0.5–1.5) 1.5 (8; 0.8–2.8) 1.3 (63; 1.0–1.9) 1.1 (27; 0.7–1.7)	 90% CI 90% CI 90% CI 90% CI

PMR, proportionate mortality ratio; SMR, standardized mortality ratio; OR, odds ratio; CI, confidence interval

Male Wistar rats [initial numbers unspecified], 18 weeks old, were exposed in chambers to coal dust and diesel-engine exhaust particle aerosols either separately or combined for 6 h per day on five days a week for up to 20 months. The coal dust sample was in the form of micronized bituminous coal obtained from Cambria, PA, United States. Respirability was approximately 50% for coal dust (mass-median aerodynamic diameter (MMAD), 2.1 μm)) and 95% for the diesel exhaust soot (MMAD, 0.71 μm). The groups of rats were exposed to the following: diesel-engine exhaust particles alone (8.3 ± 2.0 mg/m^3); diesel-engine exhaust plus a low concentration of coal dust (8.3 ± 2.0 mg/m^3 diesel particles and 5.8 ± 3.5 mg/m^3 coal dust particles); a low concentration of coal dust (6.6 ± 1.9 mg/m^3 dust particles); and a high concentration of coal dust (14.9 ± 6.2 mg/m^3 dust particles). Control animals inhaled room air. Six rats per group were killed after four, eight, 16 and 20 months of exposure. All macroscopic lesions and selected organs (respiratory tract, lymph nodes, stomach, oesophagus) were studied histologically. Exposure to coal dust and diesel soot either singly or in combination had no significant effect on body weight or on mortality patterns of exposed animals. Neoplasms were first observed after 16 months of exposure: one subcutaneous fibrosarcoma in a control and one fibrosarcoma of the heart in a rat exposed to diesel exhaust only. After 20 months, one mammary fibroadenoma and one bronchiolar adenoma were observed in six animals exposed to diesel exhaust; one bronchiolar adenoma and one basal-cell tumour of a hind leg were observed in six animals exposed to diesel exhaust and a low concentration of coal dust; one systemic lymphoma, one subcutaneous fibroma and one malignant histiocytoma were observed in six animals exposed to the high concentration of coal dust; one systemic lymphoma and one adrenal phaeochromocytoma were observed in six animals exposed to the low concentration of coal dust; and one subcutaneous lymphoma and one renal lymphoma were observed in six controls (Karagianes *et al.*, 1981). [The Working Group noted the short study duration and the small number of animals examined at the end of the 20-month exposure.]

Groups of 144 male and 72 female Fischer 344 weanling rats were exposed by inhalation in chambers to bituminous coal dust alone (respirable coal dust concentration, 2 mg/m^3), diesel engine particles alone (diesel particle concentration was 2 mg/m^3) or coal dust and diesel engine particles combined (coal dust and diesel engine particle concentrations, both 1 mg/m^3) for 7 h per day on five days a week for 24 months. The coal came from a high-prevalence pneumoconiosis coal seam [source and particle size unspecified]. Control animals inhaled filtered air in the chambers. There was no difference in survival across treatment groups or sexes. In each of the four groups, 120–121 males and 70–71 females were necropsied. The incidence of tumours did not differ statistically (Fisher's exact test) between the three exposure groups and filtered air controls for the fifty tissues examined and was similar to that reported for control groups in other studies (Lewis *et al.*, 1986). [The Working Group noted the lack of specific details regarding histopathological findings in the lungs.]

3.2 Intrapleural administration

Rat: Groups of 16 SPF Wistar rats [sex unspecified] of an average age of 39 days received a single intrapleural injection of 20 mg/animal coal dust (respirable) [source unspecified] or 20 mg carbon black (pelican black ink without shellac) in 0.4 mL saline. A group of 20 controls was treated with saline. Mean survival rate was 690 days (coal dust), 618 days (carbon black) and 720 days (in controls). Thymomas/lymphosarcomas were detected in 1/16 rats treated with coal dust, in 2/16 rats treated with carbon black and in 1/15 controls (Wagner, 1976).

4. Other Data Relevant to an Evaluation of Carcinogenicity and its Mechanisms

4.1 Deposition, distribution, persistence and biodegradability

4.1.1 *Humans*

Coal workers' pneumoconiosis and progressive massive fibrosis are highly correlated to (estimates of) cumulative dust exposure and dust (components) remaining in the lung (Rossiter *et al.*, 1967; Hurley *et al.*, 1982; Ruckley *et al.*, 1984; Attfield & Seixas, 1995). The amount of dust remaining in the lung is the net result of deposited dose minus (long-term) clearance. Love *et al.* (1970) found no difference in the deposition of an experimental 1 μm aerosol between two groups of coal workers, one with simple coal workers' pneumoconiosis and an age and occupation history matched group with normal chest X-rays. The presence of coal dust in the lungs does not increase deposition rate; however, Bergman and Casswell (1972) did show that the rate of accumulation was higher among workers in high-rank coal mines and in subjects with progressive massive fibrosis. Several post-mortem studies have been carried out in which the whole lung was digested or ashed and the total or specific dust in the lung was measured (Nagelschmidt *et al.*, 1963; Bergman & Casswell, 1972; Douglas *et al.*, 1986). These studies showed that, in coal workers, 40–60 g total dust may be found in the lungs, and that both the total amount retained (as part of estimated cumulative exposure) and the ash fraction are higher in miners with coal workers' pneumoconiosis or progressive massive fibrosis than in reference miners. These data suggest that the lung dust burden is not simply a reflection of (cumulative) exposure, but that individual differences in deposition and/or clearance might be factors explaining disease susceptibility. In studies of animals subjected to the same dose of asbestos, those animals that developed asbestosis were found to have retained significantly more fibres in their lungs, and this was found to be related both to differences in deposition (longer fibres) and individual clearance. The available human studies do not allow a distinction to be made between these two mechanisms. Chapman and Ruckley (1985) noted that quartz dust is usually found more in low-rank coal dust exposure, and is 'concentrated' in lymph nodes. This phenomenon was not, however, related to the grade of coal workers' pneumoconiosis.

4.1.2 *Experimental systems*

No data were available to the Working Group.

4.2 Toxic effects

Many extensive epidemiological studies (including exposure–response relationships) have demonstrated a causal relationship between coal dust exposure and fibrosis (coal workers' pneumoconiosis, progressive massive fibrosis), lung function decline, bronchitis and (somewhat more controversially) emphysema. However, experimental studies have generated useful information on the toxicity and effects of respirable coal mine dust and its components (free silica, metals, coal rank, diesel exhaust, etc.). Such studies can be divided into experimental studies, including both in-vitro and animal research, and human studies ranging from case studies to carefully designed molecular epidemiological studies (Schulte, 1993). In the past decade, these studies have enhanced our understanding of disease mechanisms by the elucidation of several key-events in particle-induced pulmonary toxicity. More specifically, as the lung burden of particles increases, alveolar macrophages and epithelial cells become activated leading to the release of inflammatory mediators, reactive oxygen species (ROS), enzymes (elastase, proteases, collagenase), cytokines (tumour necrosis factor (TNF), interleukin (IL)-1, IL-8, macrophage inflammatory protein 2 (MIP-2), monocyte chemotactic protein 1 (MCP-1) and growth factors (platelet-derived growth factor (PDGF), transforming growth factor (TGF)) that control and stimulate pathogenic events (Borm, 1994; Janssen *et al.*, 1994; Driscoll *et al.*, 1996). Some of these events will be discussed as markers of toxicity or bioactivity of coal dust in experimental systems.

4.2.1 *Humans*

Diseases caused by coal (mine) dust exposure have been reviewed (Parkes, 1994; Rom, 1992; Heppleston, 1992; Wouters *et al.*, 1994; United States National Institute for Occupational Safety and Health, 1995); apart from simple coal workers' pneumoconiosis, which is characterized by the presence of small opacities (< 10 mm) on a chest X-ray (International Labour Office, 1980), various other diseases have been reported in coal miners and ex-coal miners and in some occupations other than mining: complicated coal workers' pneumoconiosis (progressive massive fibrosis), pleural abnormalities, emphysema, chronic bronchitis, accelerated lung function loss, lung cancer and stomach cancer. Most of the above outcomes are highly correlated to estimates of cumulative dust exposure and dust or dust components remaining in the lung (Rossiter *et al.*, 1967; Hurley *et al.*, 1982; Ruckley *et al.*, 1984; Attfield & Seixas, 1995). However, no such generalization can be made about the effects of quartz content and coal rank in the induction of fibrotic endpoints (for a review, see Heppleston, 1988). Particle deposition, dust clearance and biological factors are considered important in the susceptibility to these outcomes (Borm, 1994).

In pathological terms, coal workers' pneumoconiosis should be considered as a variable entity, the exact pattern of which depends on the amount and the composition of

the dust retained in the lung (Davis *et al.*, 1983). The various components of coal workers' pneumoconiosis include primarily the coal dust macula, silicotic nodule, chronic bronchitis, several types of emphysema and secondary manifestations in the lung. Diagnosis and classification are generally based on working history and chest X-ray findings (International Labour Office, 1980) although high-resolution computed tomography (HRCT) can be used to detect early changes (e.g. < 0/1, 1/0) and parenchymal fibrosis or emphysematous changes (Remy-Jardin *et al.*, 1990). The main determinant of coal workers' pneumoconiosis is cumulative dust exposure; prevalence estimates vary between different countries, but show that the level of no coal workers' pneumoconiosis is between 50 and 100 mg/m^3 per year, which conforms to a lifetime exposure of 2 mg/m^3 coal dust limit in a number of countries (i.e. United States, Germany).

Progressive massive fibrosis can be diagnosed when large opacities (> 1 cm) are observed in chest X-rays. Progressive massive fibrosis is usually associated with significant decreases in lung function, breathlessness, chronic bronchitis and recurrent infections. The main determinants are cumulative dust exposure and the presence of simple coal workers' pneumoconiosis, although it may also develop in miners without previous coal workers' pneumoconiosis. The difference in both the prevalence (2–20%) and the incidence of progressive massive fibrosis varied by a factor 20 or more between different mining countries and also between regions and coal mines within regions (Hurley *et al.*, 1987), a finding that could not be related to the quartz content of the coals. However, progressive massive fibrosis risk is consistently higher in high-rank coal mines (MacLaren *et al.*, 1989; Attfield & Seixas, 1995). Biological factors that probably play a role in individual susceptibility to progression of coal workers' pneumoconiosis to progressive massive fibrosis include the extent of release of TNF (Lassalle *et al.*, 1990; Schins & Borm, 1995) and growth factors such as TGF-β from alveolar macrophages (Vanhée *et al.*, 1994). In a five-year follow-up study of 104 ex-coal miners, Schins and Borm (1995) showed that progression of coal workers' pneumoconiosis was more frequent (relative risk, 8.1) in those with an abnormally high coal mine dust-induced monocyte TNF-release, compared to a relative risk of 3.7 for cumulative exposure to respirable coal mine dust. Porcher *et al.* (1994) found that TNF-release from monocytes was also consistently higher in ex-miners with progressive massive fibrosis compared to controls. Interestingly, immunogenetic studies in subjects with silicosis and coal workers' pneumoconiosis (Honda *et al.*, 1993; Rihs *et al.*, 1994) have revealed 'susceptible' HLA-regions. In addition to TNF, Vanhée *et al.* (1994) found that the release of active TGF-β (which is anti-fibrotic) was decreased in alveolar macrophages of miners with progressive massive fibrosis compared with those with simple coal workers' pneumoconiosis. Thus, the balance of pro- and anti-fibrogenic cytokines is a better indicator of susceptibility (Vanhée *et al.*, 1995). It should be noted, however, that TGF-β can also be released by fibroblasts and blood platelets, whereas TNF is only released by macrophages/monocytes.

Based on the mild alveolitis occurring in coal workers' pneumoconiosis, several research groups formulated the hypothesis that an increased release of oxidants in the lung was important and have investigated the adaptive anti-oxidant response as a back-

ground for markers of disease or exposure. P.J.A. Borm and co-workers described an initial decrease in red blood cell glutathione (GSH) and GSH-S-transferase in early-stage coal workers' pneumoconiosis, while an increase was seen in progressed stages (Borm *et al.*, 1987; Engelen *et al.*, 1990; Evelo *et al.*, 1993). Other studies have demonstrated that superoxide dismutase (SOD), and more specifically MnSOD-induction is associated with exposure to cristobalite (Janssen *et al.*, 1994) and coal mine dust (Perrin-Nadif *et al.*, 1996).

Focal emphysema is a characteristic though controversial component of simple dust lesions; this topic has been reviewed by Heppleston (1972). The precise diagnosis and distinction of the morphological forms of focal emphysema depend on pathology and HRCT (Remy-Jardin *et al.*, 1990). Post-mortem analyses of coal miners' lungs have demonstrated an association between focal emphysema and both dust exposure (Ruckley *et al.*, 1984) and dust content (Leigh *et al.*, 1994), but these studies have failed to reveal the role of crystalline silica and pre-existing dust-related fibrosis. Nevertheless, a basic mechanism has been suggested and this involves a protease–antiprotease imbalance in which activated neutrophils (in response to coal mine dust) release oxidants that inactivate α1-antitrypsin and release elastases/proteases (Rom, 1990; Huang *et al.*, 1993). Coal mine dust exposure does cause a mild alveolitis, while the absorbed ferrous sulfate in the coal mine dust is responsible for the ROS production that inactivates α1-antitrypsin *in vitro* (Huang *et al.*, 1993). However, levels of this anti-protease detected by bronchoalveolar lavage were not altered in coal miners with emphysema (Rom, 1990), and these findings are supported by experimental findings in animal studies (Martin *et al.*, 1980). Other studies have found that the post-mortem lung iron content also correlated well with coal workers' pneumoconiosis-score (Rossiter, 1972) and hydroxy-proline (Ghio & Quigley, 1994) as markers of fibrosis in coal miners.

Chronic bronchitis and airflow obstruction have been described in coal miners (reviewed in Wouters *et al.*, 1994) and are common effects of inorganic dust exposure in the workplace (reviewed in Oxman *et al.*, 1993). The extra loss of lung function has been estimated from both cross-sectional and longitudinal studies and lies between –0.5 and –1.2 mL FEV_1 per gh/m^3 of exposure, which is equivalent to 40–100 mL at current standards of 2 mg/m^3. Chronic bronchitis is also increased among smoking and non-smoking coal miners (Marine & Gurr, 1988) and is associated with a greater loss of FEV_1 (Rogan *et al.*, 1973). Swaen *et al.* (1995) showed that, in miners with low FEV_1 (< 70 %) or FVC (< 80 %), mortality for gastric cancer was significantly lower than in those with 'normal' lung function (FEV_1 > 70 %, FVC > 80 %). The impaired pulmonary clearance in those with airway obstruction may deliver less coal dust to the gastrointestinal tract.

4.2.2 *Experimental systems*

(*a*) *In-vivo studies: long-term effects of coal dust*

(i) *Fibrosis, intratracheal administration*

Ray *et al.* (1951a,b) determined the effect of coal mine dust and supplemented quartz (2–40%) in rats after intratracheal doses of 100 mg of each dust. They observed fibrotic lesions and concluded that anthracite coal mine dust had no inhibitory action on quartz-

induced fibrosis. Later studies, using intratracheal administration of 50 mg coal dust in rats, confirmed that coal dust was less fibrogenic than quartz or hard rock dust, but did suggest an attenuating effect of coal mine dust on the quartz-induced effect (Martin *et al.*, 1972; Rosmanith *et al.*, 1982; Szymczykiewicz, 1982; Sahu *et al.*, 1988). An intratracheal dose of 50 mg coal dust containing 4, 7 or 18% quartz induced significant fibrosis from 3 to 18 months after exposure; the dusts high in quartz content (7 and 18%) always led to more fibrosis (Martin *et al.*, 1972). Rosmanith *et al.* (1982) injected 50 mg of 30 different coal mine dusts into rats: 5 of these dusts caused focal or diffuse fibrosis in parenchyma and lymph nodes 6 and 12 months after administration. The fibrogenic samples were characterized by the highest dust and ash content in the lymph nodes of exposed animals. An intratracheal dose (50 mg) of coal dust supplemented with quartz up to 10% of the total mixture caused an increase in the numbers of cells in the tracheobronchial lymph nodes of the rats after 90 days. The same dose in combination with a sugar cane extract (gur, or jaggery) in drinking-water caused lymphadenopathy (Sahu *et al.*, 1988).

(ii) *Fibrosis, inhalation exposure*

SPF-Wistar rats exposed for 20 months (6 h/day, 5 days/week) at levels of 6.6 and 14.9 mg/m^3 coal dust from a mine developed lesions similar to simple coal workers' pneumoconiosis in humans. No advanced lesion such as micro- or macronodules or infective granulomas were observed in these animals, but focal bronchiolization occurred after exposure for 20 months (Busch *et al.*, 1981). The importance of quartz in coal dust fibrogenicity was demonstrated by Ross *et al.* (1962) and Martin *et al.* (1972) who exposed rats to different coal–quartz mixtures. Martin *et al.* (1972) found that fibrosis developed in all groups exposed to coal dust (300 mg/m^3, 6 h/day, 5 days/week, 3 months) supplemented with quartz, but only at 18 months for the lowest concentration of quartz (4%). At higher quartz concentrations (7 and 18%), collagen formation was already increased at six months; above 10% quartz, nodules developed and collagen production was five times greater than with coal alone. Ross *et al.* (1962) carried out similar experiments in which rats were exposed to dust levels of 60 mg/m^3 (16 h/day, 10 months) and quartz concentrations from 5 to 40%. The experimental animals showed little fibrosis after exposure to mixtures with 5 and 10% quartz. However, rats exposed to 20 and 40% quartz–coal mixtures had fibrosis and increased collagen content at the end of exposure. Both parameters appeared to be correlated with the total quartz remaining in the lung 100 days after exposure.

(iii) *Effects on immune system and inflammatory cells*

Most studies of the effects on the immune system in experimental animals exposed to coal dust alone or with crystalline silica have described an increase in the number of alveolar macrophages and neutrophils (Bingham *et al.*, 1975; Brown & Donaldson, 1989; Brown *et al.*, 1992; Terzidis-Trabelsi *et al.*, 1992; Mack *et al.*, 1995). The persistence of this inflammation has been found to be strongly dependent on exposure route, regimen and total dose. In rats exposed by inhalation to 10 mg/m^3 coal dust (7 h/day, 5 days/week, 32 days), the number of neutrophils and lymphocytes was still increased (15 versus

0.5%) 64 days after recovery, whereas the total cell number had returned to normal (Brown & Donaldson, 1989; Donaldson *et al.*, 1990). In a similar inhalation experiment, quartz (Sykron-F600) caused a marked progression of the inflammatory response after cessation of exposure. On the other hand, after a single intratracheal instillation (Adamson & Bowden, 1978), alveolar macrophage yield increased for the first six days and returned to control levels by 28 days, while neutrophils increased after one day and returned to normal after three days. The United States National Institute for Occupational Safety and Health conducted a long-term study of inhalation exposure to coal dust and/or diesel. In rats, exposure to coal dust (2 mg/m^3 for 7 h/day, 5 days/week, over 2 years) resulted in a chronic elevation of alveolar macrophages (Castranova *et al.*, 1985). Coal dust was shown to have no effects on influenza infection in mice (Hahon *et al.*, 1985), on immunocompetence (Mentnech *et al.*, 1984) or on biotransformation enzymes (Rabovsky *et al.*, 1984). Bingham *et al.* (1975) found that the phagocytic and bactericidal functions of alveolar macrophages were depressed in rats after inhalation exposure to two coal dust types (from Utah and Pennsylvania, United States) at levels of 2 mg/m^3 (6 h/day, 5 days/week, 4 months). In mice, Singh *et al.* (1982) found that immune responses were inhibited by intraperitoneal administration of coal mine dust. In guinea-pigs, a selective depression of the lysosomal enzyme sialidase in alveolar macrophages was caused by sub-chronic coal dust exposure for four months (6 h/day, 5 days/week) to 300 mg/m^3 coal mine dust (Terzidis-Trabelsi *et al.*, 1992).

Activation of macrophages has also been described after in-vivo exposure to coal dust, as indicated by increased cytokine release (Bruch & Rehn, 1994). Inhalation of coal mine dust was associated with increased release of connective tissue proteases by the bronchoalveolar leukocytes (Brown & Donaldson, 1989). Kusuka *et al.* (1990) found that bronchoalveolar lavage cells from SPF-PVG rats treated with 1 mg of coal dust or TiO_2 showed significantly less inhibition to lymphocyte mitogenesis compared to normal alveolar macrophages. In fact, the mitogenic index was linearly related to the polymorphonuclear neutrophil content in bronchoalveolar macrophages and is probably regulated by cytokines, including IL-1. Brightwell and Heppleston (1971) conducted an inhalation study in mice (400 h over 4 weeks) using low- (13 mg/m^3) and high-rank (22 mg/m^3) coal mine dust from Wales. These experiments demonstrated a depression of mitotic indices in tissue areas with deposited coal dust; similar effects were seen in quartz-exposed mice at exposure levels of 12 and 28 mg/m^3.

(iv) *Interaction with diesel emissions*

Vallyathan *et al.* (1986) exposed rats and monkeys to the four following regimens: coal dust (2 mg/m^3), diesel exhaust (2 mg/m^3), coal dust plus diesel exhaust (1 mg/m^3 each) and filtered air (controls). Except for dust-laden macrophages in alveolar spaces and focal accumulations of dust-laden macrophages near the respiratory bronchioles that were associated with hyperplasia of type II cells, few pathological changes were demonstrated in any group. No major immunological, inflammatory or biotransformation enzyme changes occurred in the mixed diesel and coal dust group compared to control or coal dust-exposed animals (Mentnech *et al.*, 1984; Rabovsky *et al.*, 1984; Castranova *et al.*, 1985; Hahon *et al.*, 1985).

(*b*) *In-vitro studies: acute, short-term effects*

(i) *Haemolysis*

Gormley *et al.* (1979) tested haemolysis by coal mine dust from low coal rank and high coal rank mines in the United Kingdom; haemolysis by the former did not correlate with the total or individual components of the coal mine dust, while lysis by dust from high-rank pits increased with the amount of non-coal minerals and quartz (but not with kaolin or mica levels). Moreover, haemolysis was poorly correlated to results of cytotoxicity in a macrophage cell line. In addition, cytotoxicity was poorly correlated with various measurements of pneumoconiosis risk in different studies and was therefore judged to be too simplistic a model (Robock & Reisner, 1982).

(ii) *Cytotoxicity to alveolar macrophages or macrophage cell-lines*

Freshly-derived macrophages from different animal species (rat, guinea pig, rabbit) and a permanent tumour cell line of macrophage-like cells (P388D1) have both been used in cytotoxicity assays of various coal mine dusts that used proper positive (e.g. quartz) and negative (e.g. TiO_2) controls. Typical concentrations in these experiments ranged between 50 and 100 μg/mL for coal mine dust and 20 and 40 μg/mL for quartz and TiO_2. Gormley *et al.* (1979) measured viability in P388D1 cells by trypan blue exclusion and several biochemical indices of cytotoxicity such as release of lactate dehydrogenase, glucosaminidase, lactic acid or total protein. No correlation was observed between the quartz content of the coal mine dust and cytotoxicity. However, the study did show that the rank and non-coal mineral content was more important. These results were confirmed by data from other studies (Reisner & Robock, 1977; Robock & Reisner, 1982; Bruch & Rehn, 1994; Massé *et al.*, 1994).

(iii) *Surface properties and formation of radicals*

The adverse effects of radicals, including ROS, in the lung may include the following: (i) damage to cell membranes through lipid peroxidation; (ii) oxidation of proteins; and (iii) DNA damage (Fubini *et al.*, 1995). Oxidative DNA damage, most probably occurring via hydroxyl-radicals formed in Fenton-like reactions (Arumoa *et al.*, 1989; Schraufstätter & Cochrane, 1991), may lead to cell death or to cell/tissue proliferation and may play a role in carcinogenesis (Janssen *et al.*, 1993). ROS may also be involved in the pathogenesis of emphysema (Huang *et al.*, 1993). Several mechanisms by which radicals play a role in mineral dust-induced effects have been demonstrated. Direct damage has been attributed to the intrinsic properties of particles such as silanol groups on the surface of silica (Nash *et al.*, 1966), surface charge properties (Brown & Donaldson, 1989) and the iron content of asbestos fibres (Zalma *et al.*, 1987). Mechanical processes, such as the grinding and cleavage of dust, including coal dust, are believed to cause the generation of radicals on 'fresh' surfaces (Vallyathan *et al.*, 1988; Dalal *et al.*, 1989).

Dalal *et al.* (1991) detected long-lived coal dust radicals in coal dust recovered from coal miners' lungs and lymph nodes. Furthermore, an increase in disease severity was accompanied by a progressive increase in coal dust radical concentration. Also, Kuhn and Demers (1992) suggested that these stable coal dust radicals may induce macrophage

eicosanoid production. By analogy to its role in asbestos toxicity, iron content may also play an important role in the toxicity of coal dust (Tourmann & Kaufmann, 1994) since the Fenton-reaction type formation of hydroxyl radicals was found to be positively related to the iron content of coal dust (Dalal *et al.*, 1995).

An indirect toxicity of particles may result from the formation of free radicals by the oxidative burst of macrophages and/or neutrophils during particle phagocytosis and inflammation. Both rat and human alveolar macrophages produce considerable amounts of oxygen radicals, including superoxide anion and hydrogen peroxide. Both the shape and the chemical properties of particles were found to be related to the generation of ROS from phagocytic cells (Hansen & Mossman, 1987). Evidence for the excessive production of ROS in coal dust-induced disorders is derived from bronchoalveolar lavage fluid of coal miners compared to non-exposed subjects (Voisin *et al.*, 1985; Rom *et al.*, 1987; Wallaert *et al.*, 1990). The oxidant-generating capacity of macrophages or neutrophils isolated from bronchoalveolar lavage fluid was higher in coal miners and was related to the severity of coal workers' pneumoconiosis (Wallaert *et al.*, 1990).

(iv) *Release of inflammatory mediators, growth factors and cytokines*

Heppleston and Styles (1967) and Heppleston *et al.* (1984) carried out the first studies on cytokines and mineral dust. In these studies, the investigators measured the release of the 'macrophage fibrogenic factor' by adding the supernatant of macrophage culture medium and (coal mine) dusts to cultured fibroblasts. A number of cytokines and related factors are now known to affect fibroblast growth, cell proliferation, chemotaxis and collagen production. These factors include the following: TNF-α, IL-1, TGF-β, PDGF, interferon-γ (IFN), insulin-like growth factor (IGF-1), fibronectin (FN), prostaglandin E_2 (PGE_2), insulin, retinoic acid thromboxane A_2 (TBA_2) and glucocorticosteroids. **Table 27** shows in a simplified form which of these factors were found *in vitro* or *ex vivo* in studies with macrophages or monocytes where silica, asbestos or coal dust was used to stimulate the macrophages or monocytes.

Release of TNF-α and IL-1 by monocytes/macrophages has been observed in response to several mineral dusts. Stimulation with coal mine dust particles results in an enhanced expression of TNF-α mRNA as well as release of active protein in a dose–response manner (Borm *et al.*, 1988; Lassalle *et al.*, 1990; Gosset *et al.*, 1991). The last study showed that coal mine dust, in comparison to crystalline silica, had a much greater effect on macrophage release of TNF-α; interestingly, no IL-6 release was induced by silica or TiO_2, but only by coal mine dust (Gosset *et al.*, 1991). Freshly ground coal dust also induced the production of PGE_2 and TBA_2 by rat alveolar macrophages *in vitro* (Kuhn & Demers, 1992). Release of leukotriene-B4 (LTB4) from rat alveolar macrophages was induced after in-vivo exposure of rats to coal mine dust (Kuhn *et al.*, 1990). Several growth factors including PDGF, IGF-1 and TGF-β were also increased after incubation of alveolar macrophages from healthy subjects with coal dust (1 mg/mL) compared to TiO_2 (Vanhée *et al.*, 1994). Coal dust was also reported to release platelet activating factors (PAF) (Kang *et al.*, 1992) at dust concentrations of 10 mg/mL and IL-1 at dust levels as low as 50 μg/mL from alveolar macrophages, although this release was

much lower than that induced by crystalline silica (Schmidt *et al.*, 1984; Leroy Lapp & Castranova, 1993).

Table 27. Factors released by monocyte/macrophage upon in-vitro incubation with coal dust, asbestos or silica

Cell/source	Dust	Factor	Reference
Macrophage/murine	Quartz (45 μm)	IL-1	Gery *et al.* (1981)
Macrophage/murine	Quartz	IL-1	Oghiso & Kubota (1987)
Monocyte/human	Quartz	IL-1	Schmidt *et al.* (1984)
Monocyte/human	Coal, Min-U-Sil	TNF-α	Borm *et al.* (1988)
Macrophage/murine	Asbestos, Min-U-Sil	TNF-α	Bissonnette *et al.* (1989)
Macrophage/human	Asbestos, Min-U-Sil	TNF-α, LTB4	Dubois *et al.* (1989)
Macrophage/murine	DQ 12, asbestos	FN	Davies *et al.* (1989)
Macrophage/murine	Min-U-Sil, asbestos	TNF-α, LTB4	Driscoll *et al.* (1990)
Macrophage/human	Coal, quartz Coal	TNF-α IL-6	Gosset *et al.* (1991)
Macrophage/murine	Coal, Min-U-Sil	PGE_2, TXA_2	Kuhn *et al.* (1992)
Macrophage/human	Coal, silica (unknown)	PAF	Leroy Lapp *et al.* (1993)
Macrophage/human	Asbestos	TNF-α	Perkins *et al.* (1993)
Macrophage/human	Coal, Silica	PDGF, TGF-β, IFG-1	Vanhée *et al.* (1995)

IL-1, interleukin 1; TNF-α, tumour necrosis factor-α; LTB4, leukotriene-B4; FN, fibronectin; IL-6, interleukin-6; PGE_2, prostaglandin-E2; TXA_2, thromboxane-A_2; PAF, platelet activating factor; PDGF, platelet-derived growth factor; TGF-β, transforming growth factor-β; IGF-1, insuline-like growth factor-1

Extracellular matrix synthesis by cultured type II epithelial cells was increased by various coal and mine dusts at levels between 300 and 750 μg/mL. Among the four dusts screened, no effect of the quartz fraction was apparent (Lee *et al.*, 1994). In-vitro studies of tracheal epithelial cells have shown that the TGF-β system is important in regulating proliferation (Nettesheim, 1995). Release of active TGF-β found to be decreased in alveolar macrophages isolated from miners with progressive massive fibrosis compared to those with simple coal workers' pneumoconiosis (Vanhée *et al.*, 1994).

4.3 Reproductive and developmental effects

No data were available to the Working Group.

4.4 Genetic and related effects (see also **Table 28** and Appendices 1 and 2)

4.4.1 *Humans*

Four groups of 23–31 men and women were studied in the soft coal opencast mining industry in Czechoslovakia. One group was employed in stripping operations 20–50 m from the mine surface, another group in digging operations 50–80 m from the mine surface, another in a coal cleaning plant and the final group had no known occupational exposure to known chemical mutagens. Peripheral blood lymphocytes stimulated with phytohaemagglutinin were scored for chromatid or chromosome breaks and exchanges. The frequency of aberrant cells was elevated only in the workers employed in digging operations. Exposure to fumes and fires leading to formation of polycyclic aromatic hydrocarbons in the soft coal opencast mining operation was considered to be responsible for increased chromosomal aberrations in this group (Šrám *et al.*, 1985).

Schins *et al.* (1995) measured the 7-hydro-8-oxo-2′-deoxyguanosine (8-oxodG) to deoxyguanosine (dG) ratio as a marker for oxidative DNA damage in peripheral blood lymphocytes of 38 retired coal miners (30 healthy and 8 with coal miners' pneumoconiosis) and 24 age-matched non-exposed controls. This ratio was significantly higher in miners than in the control group. Neither age nor smoking status was related to the extent of oxidative DNA damage. Among the miners, no difference was observed between those with or without pneumoconiosis. No relationship was observed between oxidative DNA damage and calculated cumulative dust exposure, total years of exposure and time since first exposure. The increased oxidative DNA damage in peripheral blood lymphocytes can be explained by increased oxidative stress induced by coal dust in the lungs and/or the presence of stable coal dust radicals in the lymph nodes (Dalal *et al.*, 1991).

4.4.2 *Experimental systems*

Five studies investigated mutagenicity of a variety of coal dust extracts in the pre-incubation variant of the Ames assay using several strains of *Salmonella typhimurium*, with and without exogenous activation. Non-nitrosated extracts were negative or borderline positive in this assay, while nitrosated extracts of bituminous or sub-bituminous coal dusts and lignite were positive. Nitrosated extracts of peat and anthracite were negative. Nitrosation of coal dusts at acidic pH may contribute to the development of gastric cancer in coal miners (Green *et al.*, 1983; Whong *et al.*, 1983; Krishna *et al.*, 1987; Hahon *et al.*, 1988; Stamm *et al.*, 1994).

There are conflicting results on the ability of coal dusts to transform mammalian cells: Yi *et al.* (1991) found that coal dust from Jiayang, China, did not induce foci in Syrian hamster embryo cells, whereas Wu *et al.* (1990) found that extracts of non-nitrosated and nitrosated sub-bituminous coal dust from New Mexico, USA, did transform BALB/c-3T3 cells.

Tucker *et al.* (1984) investigated mutagenicity at the *tk* locus of mouse lymphoma cells and sister chromatid exchange in Chinese hamster ovary cells. Nitrosated extracts of sub-bituminous coal dust were positive in these assays. Extracts of nitrosated sub-

Table 28. Genetic and related effects of coal dust

Test system	Result[a]		Dose[b] (LED/HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Non-nitrosated extracts				
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	2 830[c]	Green *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	2 830[c]	Green *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	15 600[d]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	7 800[e]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	15 600[f]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	45 000[g]	Krishna *et al.* (1987)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	(+)	138[h]	Hahon *et al.* (1988)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	15 600[i]	Stamm *et al.* (1994)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	31 250[j]	Stamm *et al.* (1994)
SAS, *Salmonella typhimurium* YG1024, reverse mutation	–	–	15 600[i]	Stamm *et al.* (1994)
SAS, *Salmonella typhimurium* YG1024, reverse mutation	–	–	31 250[j]	Stamm *et al.* (1994)
TBM, Cell transformation, BALB/c-3T3 cells	+	NT	2 080[k]	Wu *et al.* (1990)
TFS, Cell transformation, Syrian hamster embryo cells, focus assay	–	NT	10[l]	Yi *et al.* (1991)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	50 000[k]	Tucker *et al.* (1984)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	500[f]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	500[e]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	5 000[d]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	5 000[i]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	–	NT	50 000[j]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	15 000[m]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	–	NT	50 000[n]	Tucker & Ong (1985)

Table 28 (contd)

Test system	Result[a]		Dose[b] (LED/HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Non-nitrosated extracts (contd)				
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	15 000[o]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	15 000[p]	Tucker & Ong (1985)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	–	NT	50 000[q]	Tucker & Ong (1985)
CHL, Chromosomal aberrations, human lymphocytes *in vitro*	+	NT	16 650[k]	Tucker *et al.* (1984)
BFA, Body fluids from animals (urine from rats), microbial mutagenicity	–	–	0.5 inh 7 h/d; 5 d/wk × 24 m[r]	Green *et al.* (1983)
SVA, Sister chromatid exchange, rat peripheral lymphocytes *in vivo*	–		0.5 inh 7 h/d; 5 d/wk × 3 m[r]	Ong *et al.* (1985)
SVA, Sister chromatid exchange, mouse bone marrow *in vivo*	–		20 000 po × 2[s]	Krishna *et al.* (1987)
MVM, Micronucleus test, mice *in vivo*	–		25 000 po × 2[k]	Tucker *et al.* (1984)
MVM, Micronucleus test, mice *in vivo*	–		0.8 inh; 7 h/d; 5 d/wk × 6 m[r]	Ong *et al.* (1985)
MVR, Micronucleus test, rats bone marrow *in vivo*	–		0.5 inh × 24 m[r]	Ong *et al.* (1985)
DVH, DNA damage (7-hydroxy-8-oxo-2′-deoxyguanosine), human lymphocytes *in vivo*	+		NG	Schins *et al.* (1995)
CLH, Chromosomal aberrations, human lymphocytes *in vivo*	?		NG	Šrám *et al.* (1985)
Nitrosated extracts				
SA0, *Salmonella typhimurium* TA100, reverse mutation	(+)	(+)	NG[e]	Whong *et al.* (1983)
SA0, *Salmonella typhimurium* TA100, reverse mutation	(+)	(+)	NG[f]	Whong *et al.* (1983)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	NG[e]	Whong *et al.* (1983)

Table 28 (contd)

Test system	Result[a]		Dose[b] (LED/HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Nitrosated extracts (contd)				
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	NG[f]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	+	15 600[d]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	+	950[e]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	+	1 170[f]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	NG[g]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	NG[f]	Whong *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	+	5 500[g]	Krishna *et al.* (1987)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	+	18[h]	Hahon *et al.* (1988)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	+	925[i]	Stamm *et al.* (1994)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	+	925[j]	Stamm *et al.* (1994)
SAS, *Salmonella typhimurium* YG1024, reverse mutation	+	+	925[i]	Stamm *et al.* (1994)
SAS, *Salmonella typhimurium* YG1024, reverse mutation	+	+	925[j]	Stamm *et al.* (1994)
G5T, Gene mutation, mouse lymphoma L5178Y cells, *tk* locus *in vitro*	+	+	5 000[k]	Tucker *et al.* (1984)
SIC, Sister chromatid exchange, Chinese hamster ovary cells *in vitro*	+	+	5 000[k]	Tucker *et al.* (1984)
MIA, Micronucleus test, BALB/c-3T3 mouse cells *in vitro*	+	NT	3 750[k]	Gu *et al.* (1992)
TBM, Cell transformation, BALB/c-3T3 cells	+	NT	1 040[k]	Wu *et al.* (1990)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	+	NT	1 670[k]	Tucker *et al.* (1984)
CHL, Chromosomal aberrations, human lymphocytes *in vitro*	+	NT	1 670[k]	Tucker *et al.* (1984)
SVA, Sister chromatid exchange, mouse bone marrow *in vivo*	(+)		20 000 po × 2[g]	Krishna *et al.* (1987)

Table 28 (contd)

Test system	Result[a]		Dose[b] (LED/HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Nitrosated extracts (contd)				
MVM, Micronucleus test, mice *in vivo*	–		75 000 po × 2[k]	Tucker *et al.* (1984)

[a] +, positive; (+), weak positive; –, negative; NT, not tested; ?, inconclusive
[b] LED, lowest effective dose; HID, highest ineffective dose; in-vitro tests, µg/mL (coal dust equivalent mass/vol); in-vivo tests, mg/kg bw/day (coal dust equivalent mass/bw); NG, not given
[c] Bituminous coal dust from Pittsburgh, PA, United States
[d] Lignite
[e] Sub-bituminous coal dust
[f] Bituminous coal dust
[g] Sub-bituminous coal dust from Wyoming, United States
[h] Bituminous coal dust from New Mexico, United States
[i] Coal dust from West Virginia, United States
[j] Coal dust from New Mexico, United States
[k] Sub-bituminous coal dust from New Mexico, United States
[l] Coal dusts from Jiayang, China
[m] Water solvent extract of bituminous coal dusts
[n] Water solvent extract of sub-bituminous coal dusts
[o] Water solvent extract of lignite coal dusts
[p] Water solvent extract of peat coal dusts
[q] Water solvent extract of anthracite coal dusts
[r] Bituminous coal dust particulate from Pittsburgh, United States
[s] Peat
[t] Anthracite

bituminous coal dust also induced micronuclei in BALB/c-3T3 cells (Gu *et al.*, 1992). Non-nitrosated extracts were not tested in these studies.

One study explored whether inhalation of bituminous coal dust at 2 mg/m^3 by rats and mice for 6–24 months induced micronuclei in bone-marrow cells or mutagenic activity in urine. No mutagenic activity was evident after inhalation exposure (Green *et al.*, 1983; Ong *et al.*, 1985). [The Working Group noted that bone-marrow cells are not an appropriate target cell for inhalation exposure of coal dust.]

Two studies examined the induction of sister chromatid exchange in normal human peripheral blood lymphocytes exposed to a variety of coal dust extracts *in vitro*. Organic solvent extracts of sub-bituminous coal dust induced chromosomal aberrations that were increased by exposure to extracts from nitrosated coal dust. Organic solvent extracts of bituminous or subbituminous coal dusts, lignite and peat induced sister chromatid exchange; anthracite extracts were negative. In contrast, water solvent extracts of bituminous coal dust, lignite and peat were positive in this assay while water solvent extracts of sub-bituminous coal dust and anthracite were negative (Tucker *et al.*, 1984; Tucker & Ong, 1985).

Neither micronuclei nor sister chromatid exchange were induced in bone marrow cells of mice treated orally with extracts of two samples of sub-bituminous coal (Tucker *et al.*, 1984; Krishna *et al.*, 1987).

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Coal is a generic term for a heterogeneous, carbonaceous rock of varying composition and characteristics. It is mined in over 70 different countries around the world, and utilized in many more for electricity generation, heating, steel making and chemical processes. It varies in type from the soft and friable lignite to the hard and brittle anthracite. The term 'rank', which reflects the percentage carbon content, is used conventionally for its classification.

Coal typically contains variable but substantial amounts of mineral matter, of which quartz is an important component. The major exposures to coal dust occur during mining and processing of coal. In these operations the exposure includes dusts generated not only from the coal but also from adjacent rock strata and other sources. These may increase the quartz component of the airborne dust to about 10% of the total mixed dust, or to even greater levels if significant rock cutting is being undertaken.

Before 1970, in Germany, the United Kingdom and the United States, levels of respirable mixed dust in underground mines were typically 12 mg/m^3 or less, depending on occupation and mine. More recently, regulations in some countries have brought these levels down to 3 mg/m^3 or less. Dust concentrations in surface (strip, opencast) coal mines are generally lower than those found in underground mining. However, owing to

the need to disturb overlying rock strata in surface mining, quartz exposures can be significant in some jobs, e.g. in rock drilling.

Exposure to coal dust also occurs during bulk loading and transfer, and at sites where coal is stored and used, such as power stations, steel and coke works, chemical plants, and during domestic use.

5.2 Human carcinogenicity data

There have been no epidemiological investigations on cancer risks in relation to coal dust *per se*. There is, however, a large body of published literature concerning cancer risks potentially associated with employment as a coal miner, including a small number of exposure–response associations with coal mine dust.

Cancers of the lung and stomach have been investigated most intensively among coal miners, with sporadic reports for other sites, such as urinary bladder. The absence of information on levels of the specific components of coal mine dust (e.g. coal, quartz, metals) further hindered interpretation of the epidemiological literature.

The evidence from occupational cohort studies for an association between coal mine dust and lung cancer has not been consistent; some studies revealed excess risks, whereas others indicated cohort-wide lung cancer deficits. There is no consistent evidence supporting an exposure–response relation for lung cancer with any of the customary dose surrogates, including duration of exposure, cumulative exposure or radiographic evidence of pneumoconiosis.

In contrast to the lung cancer findings, there have been reasonably consistent indications of stomach cancer excess among coal miners, detected both in occupational cohort studies and in community-based case–control studies. However, there is no consistent evidence supporting an exposure–response gradient for coal mine dust and stomach cancer.

5.3 Animal carcinogenicity data

Coal dust was tested for carcinogenicity both separately and in combination with diesel particle aerosols by inhalation in one adequate experiment in rats. The incidence of tumours was not increased compared to controls.

In one study in rats, single intrapleural injection of coal dust did not increase the incidence of thoracic tumours.

5.4 Other relevant data

The biological effects of coal mine dust in coal miners include simple coal workers' pneumoconiosis, progressive massive fibrosis, emphysema, chronic bronchitis and accelerated loss of lung function. Fibrotic endpoints in animals are attributable either to its quartz, clay or ash content; the age and dimensions of the particles probably also play a role. Human studies suggest that coal dust contains stable radicals and is able to induce reactive oxygen species that may cause DNA damage. Coal mine dust can cause cyto-

toxicity and induce the release of mediators from inflammatory cells; however, these effects are not predictable from its quartz content alone. *In vitro*, the cytotoxicity of quartz is clearly inhibited by the presence of coal dust, while the inflammatory activity is dependent on yet unidentified parameters. The release of cytokines and growth factors most probably contributes to pneumoconiosis development. Reactive oxygen species also can inactivate α-1-antitrypsin and bronchoalveolar leukocytes from rats inhaling coal mine dust had increased secretion of connective tissue proteases, leading to the development of emphysema.

Non-nitrosated extracts of a variety of coal dust samples were not mutagenic to *Salmonella typhimurium*. Non-nitrosated extracts of sub-bituminous coal dust induced mammalian cell transformation in one study; these extracts also induced chromosomal aberrations and sister chromatid exchange in human lymphocyte cultures. These extracts also induced sister chromatid exchange in Chinese hamster ovary cells.

Exposure of rodents to coal dust by inhalation or oral gavage did not produce any evidence of mutagenicity.

5.5 Evaluation[1]

There is *inadequate evidence* in humans for the carcinogenicity of coal dust.

There is *inadequate evidence* in experimental animals for the carcinogenicity of coal dust.

Overall evaluation

Coal dust *cannot be classified as to its carcinogenicity to humans (Group 3).*

6. References

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[1] For definition of the italicized terms, see Preamble, pp. 24–27

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