3. Biological Data Relevant to the Evaluation of Carcinogenic Risk to Humans

3.1 Carcinogenicity studies in animals

No data were available to the Working Group.

3.2 Other relevant data in humans

(a) *Toxic effects*

(i) *Skin and eyes*

Workers in the paint manufacturing industry (Pirilä, 1947; Ulfvarson, 1977) and painters (Pirilä, 1947; Högberg & Wahlberg, 1980; Winchester & Madjar, 1986) are at a considerable risk of developing an occupational dermatosis. In one study of Swedish paint industry workers, the prevalence of occupational dermatoses was about 40%, 26% of which were on the hands and arms (Ulfvarson, 1977). Among Swedish house painters, the prevalence of occupational skin disease was 4–6%, mainly affecting the hands. More than half of the dermatoses were nonallergic contact eczemas, probably mostly induced by organic solvents, mainly in atopic subjects. Allergic contact eczemas involved hypersensitivity towards chromium, nickel, epoxy resin components and formaldehyde. Several cases were seen of allergy to chloracetamide, which was widely used as a biocide in water-based paints and glues. Sensitivity to turpentine, which was formerly prevalent among painters, is now rare (Högberg & Wahlberg, 1980).

The wide variety of skin sensitizing agents in paints include some of the monomer residues from resins (e.g., phenol/formaldehyde resins, carbamide resin, melamine resin, epoxy compounds, acrylates). In addition, natural resins, such as colophony, may contain sensitizing agents. Some hardeners, such as acid anhydrides and *para*-toluenesulfonic acid, may cause sensitization, as may some metals used as pigments and driers in paints, e.g., cobalt and zirconium. Chromate sensitivity is rare in the painting trades, due to the low solubility of the salts used. Of other additives, several biocides (e.g., formaldehyde, chlorophenols and isothiazolinones) may have this effect. Of the solvents, only turpentine and dipentene (limonene) are known to be sensitizers (Fregert, 1981; Hansen *et al.*, 1987).

Some organic solvents (e.g. some ketones and esters) are irritants, as are some resin monomers (e.g., butyl acrylate) and additives (e.g., amines, ammonia and organic peroxides; Hansen *et al.*, 1987).

Corneal changes have been described in workers exposed to spray paints containing xylene (Matthäus, 1964). Changes in the lens of the eye have been recorded in car painters exposed to a mixture of solvents (Raitta *et al.*, 1976; Elofsson *et al.*, 1980). However, no ocular effect was noted in industrial spray painters occasionally exposed to toluene at up to 4125 mg/m^3 (Greenburg *et al.*, 1942). Water-based paints may contain triethylamine (Hansen *et al.*, 1987), which can cause corneal oedema (Åkesson *et al.*, 1985, 1986).

(ii) *Respiratory tract*

Complaints of irritation in the upper airways were reported among paint factory workers (Winchester & Madjar, 1986) and among painters occupationally exposed to white spirits and other solvents (Cohen, 1974; Seppäläinen & Lindström, 1982; Lindström & Wickström, 1983; Pham *et al.*, 1985; White & Baker, 1988). Hyposmia has sometimes been associated with exposure of painters to solvents (Lindström & Wickström, 1983). Histological changes of the nasal mucosa were reported among industrial spray painters (Hellquist *et al.*, 1983).

Some painters suffer from lower airway symptoms (Schwartz & Baker, 1988; White & Baker, 1988), and there is a high prevalence of chronic phlegm bronchitis among spray painters (White & Baker, 1988) and lacquerers (Sabroe & Olsen, 1979). An obstructive ventilatory pattern was recorded after testing lung function in people who abused spray paint by inhalation (Reyes de la Rocha *et al.*, 1987). A decrease in expiratory flow rates was noted in a few workers in a printing paint factory, probably due to irritant effects, but not among car painters (Beving *et al.*, 1984a). Other studies of painters have also indicated bronchial obstruction (Pham *et al.*, 1985; Schwartz & Baker, 1988; White & Baker, 1988), and small airways disease has been noted in car painters exposed to isocyanates (Alexandersson *et al.*, 1987). In contrast, no disturbance of lung function was reported among house painters using solvent-based (Hane *et al.*, 1977; Askergren *et al.*, 1988) and water-based (Askergren *et al.*, 1988) paints. Danish painters were reported to have a high rate of disability pensions due to respiratory disease (Mikkelsen, 1980).

Painting may also entail exposure to compounds that cause allergic reactions in the airways. Isocyanates can cause both asthma and pneumonitis in painters (Nielsen *et al.*, 1985; Hagmar *et al.*, 1987). Exposure in the painting trade to isocyanates and polyisocyanates may induce antibody formation (Welinder *et al.*, 1988). Acid anhydrides (e.g., trimellitic anhydride, phthalic anhydride and its derivatives, and maleic anhydride) caused sensitization in workers producing alkyd binders (Wernfors *et al.*, 1986; Hagmar *et al.*, 1987; Nielsen *et al.*, 1988). Moreover, paints sometimes contain asthma-inducing amines (Hagmar *et al.*, 1987).

Exposure to aluminium dust and iron oxide during paint production may cause fibrosis, and exposure to iron oxide can cause pneumoconiosis (Maintz & Werner, 1988).

(iii) *Nervous system*

The neurotoxic effects of exposures to solvents have been reviewed (World Health Organization, 1985; Cranmer & Golberg, 1986; National Institute for Occupational Safety and Health, 1987). Such effects have been determined by means of questionnaires about subjective symptoms, neuropsychological testing and neurophysiological examination of central and peripheral nervous system function (Table 20), as well as in epidemiological studies of neuropsychiatric diseases.

Subjective symptoms, e.g., a feeling of intoxication, fatigue, poor concentration, emotional instability, short-term memory problems and headache, have been recorded in a series of cross-sectional studies of workers in the paint manufacturing industry, of house painters, of car and industry painters and of shipyard painters. Some of the symptoms are short or mid-term, others are persistent. However, no such symptom was recorded in house painters using mainly water-based paints (Askergren *et al.*, 1988). Neuropsychological tests have documented impairment of psychomotor performance, memory and other intellectual functions, as well as changes of mood (Table 20).

Electroencephalographic changes and a slight decrease in cerebral blood flow were recorded in paint industry workers (Ørbaek *et al.*, 1985). Electroencephalographic abnormalities have also been seen in car and industry painters (Seppäläinen *et al.*, 1978; Elofsson *et al.*, 1980). Other studies of solvent-exposed painters have failed to identify such effects (Seppäläinen & Lindström, 1982; Triebig *et al.*, 1988), and no effect on auditory-provoked

Table 20. Symptoms and neurobehavioural effects in studies of workers in the painting trade[a]

Population	Symptoms	Psychomotor performance	Short-term memory	Other intellectual functions	Mood	Reference
Paint industry workers	+	+	+			Anshelm Olson (1982)
	++	–	–	[+]	++	Ørbaek *et al.* (1985)
	[+]	[+]				Winchester & Madjar (1986)
House painters	[+]	+	+	+		Hane *et al.* (1977)
	+	++	+	–	–	Lindström & Wickström (1983)
	[++]	[–]	[–]	[–]	[–]	Fidler *et al.* (1987)
	–	–	–	–	+	Triebig *et al.* (1988)
	++	[++]	[++]	[++]	++	Baker *et al.* (1988)
		++	[+]	[++]		Mikkelsen *et al.* (1988)
Car/industry painters		+	+	+	+	Hänninen *et al.* (1976)
	+					Husman (1980)
	+	+	+	+	+	Elofsson *et al.* (1980)
	+				+	Struwe *et al.* (1980)
		[–]	[+]	[–]		Maizlish *et al.* (1985)
Shipyard painters	+	[+]	–	–		Cherry *et al.* (1985)
	[+]			+		Valciukas *et al.* (1985)

[a] +, exposed group differed statistically significantly from a control group; ++, there was a dose-response relationship; –, there was no statistically significant difference; [], the Working Group considered that the evidence was limited because the effect was weak or inconsistent and/or the duration and/or intensity of the exposure was low.

potential was seen in painters exposed to water-based paints (Askergren *et al.*, 1988). In one group of house painters (Mikkelsen *et al.*, 1988) and in a study of car and industrial painters (Elofsson *et al.*, 1980), signs of slight atrophy were found by computed brain tomography, but another study showed no such effect (Triebig *et al.*, 1988).

Occasional cases of clinical polyneuropathy have been described in spray painters exposed to methyl-*n*-butyl ketone (Mallov, 1976). In a few cross-sectional studies of car and industry painters (Elofsson *et al.*, 1980; Husman, 1980; Maizlish *et al.*, 1985), signs of slight neurological impairment were observed during physical examinations. Neurophysiological studies of house painters (Askergren *et al.*, 1988) and of car and industrial painters (Seppäläinen *et al.*, 1978) have indicated slight toxic effects on the peripheral nervous system, but other studies have not (Seppäläinen & Lindström, 1982; Cherry *et al.*, 1985; Ørbaek *et al.*, 1985;

Triebig *et al.*, 1988). Formerly, painters exposed to lead sometimes showed clinical effects on the peripheral nervous system, including palsy (mainly affecting the extensor muscles of the forearm) and drop (affecting the wrist; Rosen, 1953). No effect on the peripheral nervous system was observed in painters who used mainly water-based paints (Askergren *et al.*, 1988).

In a cohort of Danish painters, statistically significant two- to three-fold increases in the relative risk of being granted a disability pension due to neuropsychiatric disease was found (Mikkelsen, 1980). Similarly increased risks were observed in case-control studies of applicants for disability pensions due to neuropyschiatric disease and for nursing home accommodation due to encephalopathy, in which the occupation of 'painter or other solvent-exposed trade' was used as an indicator of exposure (Axelson *et al.*, 1976; Olsen & Sabroe, 1980; Lindström *et al.*, 1984; Rasmussen *et al.*, 1985). Significant increases in risk were not, however, seen in other case-control studies, using subjects granted a disability pension (van Vliet *et al.*, 1987), subjects who had consulted general practitioners because of minor psychiatric illness (Cherry & Waldron, 1984) and deaths from presenile dementia (O'Flynn *et al.*, 1987). [The Working Group noted that the confidence intervals were wide and that the results of the latter studies could thus be considered non-positive rather than negative.] Painters were overrepresented among cases of psychomotor epilepsy (Littorin *et al.*, 1988).

[The Working Group noted that reasons for the variable outcome include differences in exposures, i.e., identity of chemicals, intensity and duration. Also, selection bias may have occurred; and the examination methods varied, some possibly being influenced by recent rather than chronic exposures. Finally, the control groups used may have not been appropriate, so that the effects of confounders cannot be ruled out.]

(iv) *Kidneys*

At the beginning of the century, it was claimed that exposure of painters to turpentine caused glomerulonephritis; this association was not firmly established (Chapman, 1941), although the suspicion that a toxic effect of solvents caused clinical disease of the glomeruli remained. Goodpasture's syndrome has been associated with exposure to paint solvents (Klavis & Drommer, 1970; Beirne & Brennan, 1972).

Case studies of glomerulonephritis indicated a possible association with exposure to various solvents, including those in paints (Zimmerman *et al.*, 1975; Ehrenreich *et al.*, 1977; Lagrue *et al.*, 1977; Ravnskov *et al.*, 1979; Finn *et al.*, 1980), although one study that showed a relative risk (RR) of 1.1 (95% confidence interval (CI), 0.4-3.1) did not (van der Laan, 1980).

Most studies on kidney disease in the painting trade have concentrated on solvents. Several solvents are nephrotoxic (Lauwerys *et al.*, 1985). In a study of industrial spray painters exposed to toluene-containing paints, no indication of kidney disease was observed (Greenburg *et al.*, 1942). Later cross-sectional studies using more sophisticated methods revealed only minor effects. Among paint industry workers who were exposed to toluene and xylene, slight haematuria and albuminuria were observed but no effect on concentrating ability or glomerular filtration rate (Askergren, 1981; Askergren *et al.*, 1981a,b,c). These results were interpreted as being a minor effect on the glomeruli. In another study of painters exposed to toluene and xylene, indications of very slight tubular effects were reported (Franchini *et al.*, 1983). In a third study of car painters exposed to low levels of white spirits and

toluene, no such effect was observed (Lauwerys *et al.*, 1985); however, a minor increase in urinary albumin excretion was reported among house painters using mainly water-based paints (Askergren *et al.*, 1988).

Kidney disease may be caused by exposure to lead in paints (Skerfving, 1987; see also IARC, 1980a).

(v) *Liver and gastrointestinal tract*

Slight effects on serum liver enzymes were noted in early studies of groups of industrial spray painters (Greenburg *et al.*, 1942). In other studies of paint industry workers (Lundberg & Håkansson, 1985), of car painters (Kurppa & Husman, 1982), of house painters (Hane *et al.*, 1977) and of subjects with suspected organic solvent poisoning (e.g., car painters; Milling Pedersen & Melchior Rasmussen, 1982), no consistent change in levels of serum liver enzymes was observed.

Lead may cause colic ('painter's colic'), and solvents and arsenic have also been claimed to cause gastrointestinal symptoms among painters and varnishers (Rosen, 1953).

(vi) *Blood and haematopoietic system*

Results obtained from haematological studies of workers in the painting trade vary. In painters who used gasoline as a solvent, a reduction in blood haemoglobin level was observed. Typical levels of aromatic hydrocarbons (one–fifth to one–tenth of the total hydrocarbon content) were 300–800 ppm (Sterner, 1941). Similarly, in later studies of car spray painters exposed mainly to xylene at rather low levels (Angerer & Wulf, 1985) and of house painters who had been exposed to various solvents (Hane *et al.*, 1977), slight decreases in haemoglobin levels were reported. In contrast, in one study of car and industrial spray painters, increased levels of haemoglobin were reported (Elofsson *et al.*, 1980).

In early studies of car spray painters, a slight decrease in white cell counts was observed, with relative lymphocytosis (Lind, 1939). This was probably due to a myelotoxic effect of benzene which was a contaminant of toluene and xylene before 1950–60. However, in one later study of patients with suspected solvent poisoning (mostly house, industrial and car painters), a slight decrease in white cell counts was reported (Milling Pedersen & Melchior Rasmussen, 1982), and in a study of car spray painters, lymphocytosis was observed (Angerer & Wulf, 1985). In contrast, another study of house painters showed no change in white cell counts (Elofsson *et al.*, 1980).

In more recent studies of paint industry workers (Beving *et al.*, 1984b; Lam *et al.*, 1985; Ørbaek *et al.*, 1985) and car painters (Beving *et al.*, 1983), slight decreases in thrombocyte counts were observed; in paint industry workers, the fatty acid composition of platelet membrane was altered (Beving *et al.*, 1988). In a further study of patients with chronic poisoning suspected to be induced by solvents (e.g., car painting), no change in thrombocyte counts was reported (Milling Pedersen & Melchior Rasmussen, 1982).

In painters, lead affects the formation of haemoglobin and red cells in bone marrow and causes haemolysis in peripheral blood (Skerfving, 1987).

(vii) *Other organs*

Some indication has been found that solvents affect muscles (raised serum creatine kinase levels), as seen during short-term exposure of volunteers to white spirits (Milling Pedersen & Cohr, 1984), in workers (e.g., house painters; Milling Pedersen & Melchior Rasmussen, 1982) and in patients with poisoning suspected to be due to solvents (mostly house, industrial and car painters; Milling Pedersen *et al.*, 1980). In the latter study, an increase in the activity of lactic dehydrogenase was observed in muscle biopsy specimens.

Case histories have been reported of subjects who suffered myocardial infarction after exposure to dichloromethane in paint removers (Stewart & Hake, 1976). However, cohort studies of paint industry workers have not indicated an increased risk for cardiovascular disease (Morgan *et al.*, 1981, 1985; Lundberg, 1986).

(viii) *Mortality from conditions other than cancer*

Many of the papers mentioned below are discussed in greater detail in section 3.3. Only statistically significant results are given here.

In one study of US paint industry workers, no increase in the total deaths from diseases of the nervous system was observed over that expected (Morgan *et al.*, 1981), and in a further study a significant decrease was observed (Matanoski *et al.*, 1986). In a study of Swedish painters, there was increased mortality from suicide (Engholm & Englund, 1982; Engholm *et al.*, 1987).

A cohort of Swedish painters showed an increase in mortality from chronic obstructive respiratory disease (Engholm & Englund, 1982), but no such increase was seen in studies of US painters (Matanoski *et al.*, 1986), of US automobile painters (Chiazze *et al.*, 1980) or of US aeroplane spray painters (Dalager *et al.*, 1980) or in two studies of workers in the paint industry (Morgan *et al.*, 1981; Lundberg, 1986).

In two further studies of paint industry workers, no increase in the total number of deaths from diseases of the genitourinary system was observed (Morgan *et al.*, 1981; Lundberg, 1986), although in one of the studies three deaths from infectious urinary tract disease were observed among cleaners in paint manufacture who had been heavily exposed to solvents, while only 0.2 were expected (Lundberg, 1986).

In two studies of paint industry workers, no increase in the total number of deaths from diseases of the gastrointestinal tract was observed (Morgan *et al.*, 1981; Lundberg, 1986). In another study of US painters, a significant decrease in the number of deaths from gastrointestinal disease was observed (Matanoski *et al.*, 1986). Increased mortality from diseases of the oesophagus and stomach has been reported in painters (Engholm & Englund, 1982). There was an indication of an increased rate of liver cirrhosis in one study (Lundberg, 1986), and, in automobile (Chiazze *et al.*, 1980) and aeroplane (Dalager *et al.*, 1980) spray painters, proportionate mortality from liver cirrhosis also appeared to be increased. Similar findings were reported in Swedish house painters (Engholm *et al.*, 1987). Danish house painters did not display an increase in the incidence of cirrhosis (Mikkelsen, 1980). [The Working Group noted that, in interpreting effects on the liver and gastrointestinal tract, the possibility that workers in the painting trade have a higher alcohol consumption than the general population must be considered.]

In one study of paint industry workers, no increase in the total number of deaths from diseases of the blood or blood-forming organs was found (Morgan *et al.*, 1981).

Studies of paint industry workers have not indicated an increased risk for cardiovascular disease (Chiazze *et al.*, 1980; Morgan *et al.*, 1981; Engholm & Englund, 1982; Morgan *et al.*, 1985; Lundberg, 1986; Matanoski *et al.*, 1986). In one study of Danish painters (Mikkelsen, 1980), deaths from diseases of the circulatory system were increased 30% as compared to the general population, but not as compared to a control group of bricklayers. Spray painters in automobile factories showed increased proportionate mortality from hypertensive heart disease (Chiazze *et al.*, 1980).

An increased number of deaths from cerebrovascular disease was observed in paint factory workers (Morgan *et al.*, 1981, 1985). Data on cerebrovascular mortality among aeroplane painters are in accordance with these results but are not significant (Dalager *et al.*, 1980). In a study of US painters, a significant decrease in the number of deaths from cerebrovascular disease was observed (Matanoski *et al.*, 1986).

[The Working Group noted that cohorts of workers in the painting trades may be subject to selection, which may bias the results of mortality studies. Also, in mortality studies, the occupational and disease categories used are broad, decreasing the specificity of the observations.]

(*b*) *Effects on fertility and on pregnancy outcome*

(i) *Fertility*

McDowall (1985) analysed a 10% sample of 601 526 births within marriage registered in England and Wales in 1980–82 for which the occupation of the father was recorded on the birth certificate. The standardized fertility ratio for men in each of 350 occupational units, defined by the Office of Population Censuses and Surveys, was calculated, taking the value for all occupational groups combined to be 100. Table 21 summarizes the findings in the five occupational groups in which paternal exposure to paint is likely: artists and commercial artists; coach painters; other spray painters; painters and decorators not elsewhere classified, and french polishers; and painters, assemblers and related occupations. Men with occupations classified as 'other spray painters' and 'painters and decorators, and french polishers' had significantly more children than expected on the basis of national rates (standardized fertility ratios, 129 and 141, respectively, based on 694 and 2871 births).

Rachootin and Olsen (1983) carried out a case–control study of 1069 infertile couples and 4305 fertile control couples attending Odense University Hospital, Denmark, in 1977–80. The RRs associated with occupational exposure to 'lacquer, paint or glue' were 1.2 (95% CI, 0.9–1.7) for men with sperm abnormalities, 1.1 (0.7–1.7) for women with hormonal disturbances, 1.4 (0.8–2.6) for women with idiopathic infertility and 1.1 (0.7–1.8) for men with idiopathic infertility.

Bjerrehuus and Detlefsen (1986) reported on a postal survey of 3251 male painters in Copenhagen, Denmark, and 1397 construction labourers. Approximately half responded, and 18% of the painters reported failure to conceive after two years of trials, compared with 10% of the construction workers. Telephone interview with a sample of the painters who had not responded to the postal questionnaire yielded a similar infertility rate.

Table 21. Standardized fertility ratios, sex ratio, percentage of births with birthweights of less than 2500 g, stillbirths, perinatal mortality and infant mortality, according to father's occupation, in occupational units in which exposure to paint is likely; England and Wales, 1981–82[a]

Occupational title (Office of Population Census and Surveys, 1970)	No. of births[b]	Standardized fertility ratio	Sex ratio (M:F births)	Births with birthweight < 2500 g (%)	Stillbirths		Perinatal mortality		Infant mortality	
					SMR	No.	SMR	No.	SMR	No.
Artists; commercial artists	373	105	0.884	4.8	75	19	65	20	72	19
Coach painters	20	89	1.000	10.0	73	1	56	1	119	2
Other spray painters	694	129*	1.224*	8.1	112	52	120	71	111	62
Painters and decorators not elsewhere specified; french polishers	2871	141*	1.049	6.6	99	191	99	242	98	224
Painters, assemblers and related occupations	341	100	1.018	7.9	131	30	131	38	144*	38
All occupations	601 526	100	1.061	6.6	100		100		100	

[a]From McDowall (1985); SMR, standardized mortality ratio

[b]10% sample, except for 'all occupations'

*Differs significantly from all occupations ($p < 0.05$)

(ii) *Perinatal toxicity*

Olsen and Rachootin (1983) reported in a letter to the Editor data on 2259 couples who had had a healthy child in 1978–79 at the Odense University Hospital, Denmark. Occupational exposure to various substances was assessed prior to delivery. Exposure to 'lacquer, paint or glue' was reported by 217 mothers and 1512 of their spouses. For maternal exposures, mean birth weights were 64 g less than the average; the authors reported that, after adjustment for maternal age, smoking and drinking habits and time to conception, birth weights were 51 g less than the average ($p = 0.12$). For paternal exposures, the adjusted birth weight of the babies was 14 g above average ($p = 0.56$).

Heidam (1984a,b) carried out a postal survey of the reproductive history of women living in Funen county, Denmark. Female painters were recruited from the local divisions of the trade union, and 76 of 81 (94%) to whom questionnaires were sent replied; among these, 38 pregnancies were reported (0.5 per woman), of which five (13%) were reported to be spontaneous abortions. A 91% response rate was obtained from a reference group of 1571 employed women; among these, 843 pregnancies were reported (0.5 per woman), of which 84 (10%) were reported to be spontaneous abortions. The corresponding RR was 2.9 (95% CI, 1.0–8.8) for painters after controlling for gravidity, pregnancy order and age. When, however, the analysis was done using separate data on births and spontaneous abortions registered in hospital, the corresponding RR was 1.1 (0.4–2.9). The authors suggested that there may have been some reporting bias among the painters.

McDowall (1985) also presented data relevant to perinatal toxicity, including the sex ratio of offspring, percentage of low birth weights and standardized mortality ratios (SMRs) for stillbirths and perinatal and infant deaths, for births in England and Wales in 1980–82 (Table 21). 'Other spray painters' had an elevated ratio of male:female births; but in each of the four other occupational units with exposure to paint, the sex ratio was below average. The infant mortality rate for the offspring of 'painters, assemblers and related occupations' was higher than that of all occupations. Workers in this group would generally be classified in social class IV, in which the infant mortality rate is 115.

Daniell and Vaughan (1988) used records of live births in Washington State, USA, from 1980–83 to compare the outcome of pregnancy in various occupational groups. Among the 1299 live births for which the occupation of father of the child was described as 'painter', the sex distribution and Apgar score at 1 min and 5 min were similar to that found in the 2529 live births for whom the occupation of the father was described as 'electrician' and in 1469 'general controls'. The RR for low birth weight (< 2500 g) among the offspring of painters was 1.1 (95% CI, 0.7–1.5) compared to 'electricians' and 1.4 (0.9–2.1) when compared to 'general controls'.

(iii) *Malformations*

McDowall (1985) also reported on malformations in England and Wales in 1980–82, according to maternal and paternal occupation (Table 22). Overall, there was no excess of malformations, except in the offspring of men in occupations classified as 'painters, assemblers and related occupations'. When specific malformations were considered, there was an excess of polydactyly in the children of men and women with occupations classified as 'paint-

Table 22. Standardized malformation ratios for specified malformations according to occupation, England and Wales, 1980–82[b]

Malformation	Artists; commercial artists				Coach painters				Other spray painters				Painters and decorators not elsewhere specified; french polishers				Painters, assemblers and related occupations			
	Father		Mother		Father		Mother		Father		Mother		Father		Mother		Father		Mother	
	Ratio	No.	Ratio	No.	Ratio	No.	Ratio	No.	Ratio	No.	Ratio	No.	Ratio	No.	Ratio	No.	Ratio	No.	Ratio	No.
All malformations	91	46	100	14	102	3	–	0	97	97	100	4	89*	369	100	4	241*	117	100	140
Anencephalus	381	3	–	0	–	0	–	0	120	2	–	0	58	4	–	0	126	1	187	5
Spina bifida	–	0	–	0	–	0	–	0	64	3	–	0	99	19	495	1	314*	7	177	12
Spina bifida and/or anencephalus	102	3	–	0	–	0	–	0	82	5	–	0	84	2	372	1	275*	8	187*	17
Cleft palate and/or cleft lip	93	3	–	0	–	0	–	0	111	7	–	0	77	20	–	0	163	5	111	10
Hiatus hernia and/or diaphragmatic hernia	–	0	–	0	–	0	–	0	–	10	–	0	101	3	–	0	–	0	94	1
Tracheo–oesophageal fistula, oesophageal atresia and stenosis	–	0	–	0	–	0	–	0	138	1	–	0	99	3	–	0	–	0	92	1
Rectal and anal atresia and stenosis	–	0	559	1	–	0	–	0	80	1	–	0	97	5	–	0	166	1	55	1
Malformations of the heart and circulatory system	156	5	–	0	550	1	–	0	80	5	–	0	79	20	–	0	166	5	58	5
Hypospadias, epispadias	54	2	288	3	–	0	–	0	119	9	–	0	78	24	–	0	194	7	83	9
Polydactyly	49	1	–	0	–	0	–	0	164	7	–	0	176*	31	1081*	2	392	8	144	9
Syndactyly	105	2	–	0	–	0	–	0	57	2	–	0	150	22	763	1	459*	8	213*	10
Reduction deformities	–	0	–	0	–	0	–	0	47	1	–	0	82	7	–	0	399*	4	138	4
Exomphalos, omphalocele	–	0	692	1	–	0	–	0	–	0	–	0	39	2	–	0	357	2	223	4
Down's syndrome	90	2	170	1	–	0	–	0	35	1	505	1	60	8	–	0	123	2	72	3

[a]For fathers, standardized malformation ratios are calculated, taking all occupations as 100. For mothers, standardized *proportionate* mortality ratios are calculated, taking all malformations in each occupational group as 100.

[b]From McDowall (1985)

*Differs significantly from all occupations ($p < 0.05$)

ers and decorators not elsewhere specified, and french polishers' and in children of men whose occupations were described as 'painters, assemblers and related occupations'. Syndactyly was in excess in the offspring of men and women whose occupation was 'painters, assemblers and related occupations'. Reduction deformities were also in excess for paternal exposure but not for maternal exposure. Spina bifida and/or anencephalus were in excess in the offspring of men and women described as 'painters, assemblers, and related occupations'.

Olsen (1983) reported data from the Register for Congenital Malformations in the county of Funen, Denmark, and took details of parental occupation from birth certificates. The authors reported a relative prevalence ratio of 4.9 (95% CI, 1.4–17.1) for congenital malformations of the central nervous system in the group in which the children's fathers were entered as painters in comparison with all other occupations; the ratio for mothers in this category was 0.

(c) *Genetic and related effects*

Haglund *et al.* (1980) studied chromosomal aberrations and sister chromatid exchanges in the lymphocytes of 17 male paint industry workers (exposed to organic solvents) who were presumed to have the highest exposure among a group of 47 paint industry workers employed in seven different factories in southern Sweden. For each exposed person, a control was chosen, matched by sex, age, place of residence (rural/urban) and smoking habits. Most of the controls were also factory workers (storeroom personnel, paint grinders, electricians, drivers, carpenters), but presumably unexposed. For analysis of both chromosomal aberrations and sister chromatid exchange, lymphocytes were cultured for 72 h; 20–25 metaphases were studied for sister chromatid exchange (17 subjects) and 100 for chromosomal aberrations (five subjects with the highest combined exposure). No difference was seen in either parameter; a significant difference in the frequency of sister chromatid exchange was observed between smokers and nonsmokers (0.202 and 0.175, respectively; $p = 0.02$). [The Working Group noted the small number of workers studied for chromosomal aberrations.]

Sister chromatid exchange was studied in the peripheral lymphocytes of 106 members of the International Brotherhood of Painters and Allied Tradesmen in two major US cities (Kelsey *et al.*, 1988). Intensity and duration of chronic exposure to solvents were estimated from interviewer-administered questionnaire data. Eight men reported no occupational history of solvent exposure; 13 allied tradesmen (including dry-wall tapers and paperhangers) reported minimal, indirect exposure to solvents and had no history of direct application of solvent-based materials. Cumulative exposure (CEI) to solvents was estimated for the working lifetimes of 85 painters. Fifty cells from each of 91 individuals were scored for sister chromatid exchange; for the remaining 15 persons, a mean of 21.2 cells per individual was examined. Cultures were incubated for 72 h. There was no elevation in the frequency of sister chromatid exchange attributable to cumulative duration of exposure to solvents or to intensity of exposure over the year prior to blood sampling. Smoking was associated with a significant elevation in the level of sister chromatid exchange (6.75 *versus* 5.73 in nonsmokers).

3.3 Epidemiological studies of carcinogenicity in humans

(a) *Occupational mortality and morbidity statistics*

Detailed data from some of the studies described below are given in Table 23.

(i) *National studies*

The occupations recorded on a 10% sample of death certificates in England and Wales were used to calculate SMRs for deaths occurring around the time of the 1951 Census (Office of Population Censuses and Surveys, 1958), of the 1961 Census (Adelstein, 1972; Office of Population Censuses and Surveys, 1972), of the 1971 Census (Office of Population Censuses and Surveys, 1979) and of the 1981 Census (Office of Population Censuses and Surveys, 1986). The SMRs for various cancer sites among painters and decorators are listed in Table 23. SMRs for all cancers were consistently above the average and those for lung cancer consistently 40% above the national average: 149 (909 deaths) in 1949–53, 143 (1502 deaths) in 1959–63, 139 (847 deaths) in 1970–72 and 142 (803 deaths) in 1979–80, 1982–83. The proportion of current smokers among painters and decorators was reported to be slightly higher than that in the total population (smoking ratio, 110, based on a sample of 7566 men, including 153 painters and decorators; Office of Population Censuses and Surveys, 1979).

Guralnick (1963) divided specific causes of death in the USA in 1950 by occupation and industry as reported on death certificates and compared them with the expected causes of deaths of all working men as reported in the census from the same year. There were 6145 deaths among white male painters and plasterers in the age group 20–64, with a SMR of 114; selected SMRs are: all cancers, 126 (1016 cases); buccal cavity and pharynx, 137 (41); oesophagus, 109 (25); stomach, 127 (130); lung, 155 (248); kidney, 120 (24); bladder, 146 (38); brain, 134 (39); and leukaemia, 117 (41) (see Table 23). Proportionate mortality ratios (PMRs) were used to test for significance; only that for lung cancer was significant.

Dunn and Weir (1965) established in 1954 a fixed cohort of 68 153 working men engaged in occupations suspected of engendering a risk for lung cancer and followed them for mortality through to 1962 for this report. In this group, 12 572 men were painters and decorators. Information on smoking and occupation was gathered for this population until 1957. The number of deaths in the eight-year follow-up was compared with that among men in California, USA, 1959–61. Painters and decorators had an SMR of 129 (91 observed) for lung cancer; adjustment for smoking resulted in a decrease in the SMR to 114. The SMR for all other cancers was 94 (153 observed). [The Working Group noted that, since other cancers were treated as a group, it is impossible to determine the risk for those at specific sites.]

Howe and Lindsay (1983) followed a cohort comprising 415 201 Canadian men with known occupational histories in 1965–69, which represented 10% of the Canadian labour force. Cancer mortality in this cohort was monitored by record linkage with a Canadian mortality data base containing all deaths registered in Canada for the years 1965–73. The only significantly elevated SMR (285; based on five observed cases) was found for cancer of the buccal cavity and pharynx, except lip, in the occupational group of construction and maintenance painters, paperhangers and glaziers as compared to the mortality of the entire cohort.

Table 23. Cancer mortality or incidence in studies of national statistics and of large occupational cohorts of painting trades

Type of neoplasm	National statistics										Occupational cohorts					
	UK, 1949–53 (OPCS, 1958); 'other', painters and decorators; males, 20–64		UK, 1959–63 (OPCS, 1972); painters and decorators; males, 15–64		UK, 1970–72 (OPCS, 1979); painters and decorators; males, 15–64		UK, 1979–80, 1972–83 (OPCS, 1986); painters, decorators and french polishers; males, 20–64		USA, 1950 (Guralnick, 1963); painters and plasterers; white males, 20–64		Sweden, 1958–71 (Englund, 1980; Engholm & Englund, 1982); painters' union		Denmark, 1970–79 (Olsen & Jensen, 1987); painters (construction)		USA, 1975–79 (Matanoski *et al.*, 1986); mixed painters	
	SMR[a]	No.	SMR[a]	No.	SMR[a]	No.	SMR[a]	No.	SMR[b]	No.	SIR[c]	No.	SPIR[d]	No.	SMR[b]	No.
All malignant neoplasms	124	2092**	122	2361**	123	1382**	124	1781**	126	1016**	109	647*	NA		110	927**
Buccal and pharynx	114	16[e]	78	12	138	10	145	40*	137	41	NA		61	5	NA	
Oesophagus	84	31	115	53	130	47	106	57	109	25	215	17	148	4	NA	
Stomach	122	360**	120	383**	118	174*	113	132	127	130*	[195	24]**				
Colon	106	120	101	123	98	78	88	74	112	77	[106	80]	94	11	136	50*
Rectum	107	103	103	100	101	57	128	82	112	47	NA		NA		111	93
Liver and gall-bladder	65	11	100	26	103	9[f]	NA		NA		NA		NA		NA	
					60	5[h]					200	12[g]	120	3[f]	156	20[f]
Larynx	91	21	58	14	127	16	141	21	200	28**			107	2[h]		
Nasal cavity	40	2	120	6	54	2	172	5	NA		177	14	NA		NA	
Lung	149	909**	143	1502**	139	847**	142	803**	155	248**	NA		125	1	NA	
											128	81*	149	79**	118	326**
Prostate	105	39	102	43	97	27	109	38	82	28	[127	124]**				
Kidney	86	25	95	39	104	27	NA		120	24	NA		48	8	98	84
Bladder	109	58	118	79	152	66**	116	48	146	38*	NA		61	4	141	27
Non-Hodgkin's lymphoma	109	23	95	38	101	26	NA		NA		NA		112	24	126	40
Hodgkin's disease	113	35	90	37	52	10	144	21	129	22	NA		56	3	NA	
Multiple myeloma	NA		106	19	82	11	NA		NA		NA		106	3	NA	
Leukaemia	111	50	98	65	125	43	81	33	117	41	NA		NA		NA	
											173	13[i]	75	5	116	37

[a]SMR, standardized mortality ratio; expected numbers based on national rates for working men

[b]Expected numbers based on national rates for white males

[c]SIR, standardized incidence ratio; expected numbers based on national rates; in square brackets, SMR

[d]SPIR, standardized proportional incidence ratio; expected numbers based on the proportions of cancers in all persons registered in the Danish Pension Fund

[e]Pharynx

[f]Liver

[g]Intrahepatic bile ducts

[h]Gall-bladder

[i]Lymphatic leukaemia

*Significant at the $p < 0.05$ level

**Significant at the $p < 0.01$ level

NA, not available; OPCS, Office of Population Censuses and Surveys

In cohort studies and national statistics, information on smoking habits is not usually available. A review addressing the effect of smoking as a confounding variable in studies of occupational groups (Simonato *et al.*, 1988) indicates that smoking has a limited effect on the association between lung cancer and occupational exposures: the estimates might be increased by 20–25%. The estimates in the studies described above were usually increased to a greater extent.

(ii) *Other studies*

As part of the US Third National Cancer Survey, both occupation and industry were identified for each subject based on main lifetime employment, recent employment and other jobs held (Williams *et al.*, 1977). Interviews were obtained for a total of 7518 men and women, representing a 57% response rate. The RRs for cancers at particular sites were estimated for specific occupations. Painting (which included painters, construction workers, paper-hangers, and pattern and model makers) was the main lifetime occupation for 27 men and was associated with an excess RR for lung cancer: 4.2, based on 12 cases ($p < 0.01$). There were two cases of leukaemia (RR, 4.0).

In a study carried out in California, USA, using the death certificates of about 200 000 white men during the period 1959–61 (Petersen & Milham, 1980), the cause of death and usual occupation as reported on the death certificate were used to calculate proportionate mortality ratios (PMRs), standardized for age and year of death. The total number of deaths from all causes among painters was 3558. An elevated PMR for lung cancer was reported among painters, but figures were not available to the Working Group.

In a similar study, Milham (1983) analysed the death records of 429 926 men and 25 066 women during the period 1950–79 in Washington State, USA. The PMR for cancers of the lung, bronchus and trachea was significantly elevated among painters (mainly construction and maintenance painters): PMR, 121, $p < 0.01$ for all ages, 251 observed; PMR, 112, not significant for ages 20–64, 103 observed; and among auto painters and body/fender repairmen: PMR, 184, $p < 0.01$ for ages 20–64, 29 observed; PMR, 148, $p < 0.05$ for all ages, 39 observed. The PMR for gastric cancer was also elevated among painters and body/fender repairmen. Among paperhangers and decorators (painters), the PMR for lung cancer was elevated (139; 21 observed), but not significantly for ages 20–64; it was significant for all ages (PMR, 140; $p < 0.05$; 50 observed). In persons with this occupation, cancers of the bladder and other urinary organs (PMR, 179, $p < 0.05$ for all ages, 11 observed; PMR, 186, not significant for ages 20–64, two observed) and reticulosarcoma occurred in excess.

Dubrow and Wegman (1984) examined cancer mortality patterns by occupation for white males over 20 years old in Massachusetts for 1971–73. Using age-standardized mortality ratios, 397 occupational categories defined from information on death certificates were assessed for their association with increased risk for 62 malignancies. Increased risks (at $p < 0.05$) were apparent for stomach cancer (23 deaths; SMR, 158) in construction and maintenance painters; for cancer of the trachea, bronchus and lung in grouped painters (110 deaths; SMR, 131) and in shipyard painters (nine deaths; SMR, 261); and for laryngeal cancer (ten deaths; SMR, 205), skin neoplasms except malignant melanoma (four deaths; SMR, 492) and prostatic cancer (36 deaths; SMR, 146) in grouped painters. Grouped painters aged 55–74

years had a statistically significant increase in risk for buccal cavity and pharynx (14 deaths; SMR, 222); a nonsignificant excess of lymphomas was seen for men in the age group 20–64 years (eight deaths; SMR, 192).

Pearce and Howard (1986) compared cancer deaths among males aged 15–64 years in New Zealand in 1974–78, for whom occupation had been listed on the death certificate, with a 10% sample of census data. The RR for leukaemia was 2.3 in association with the occupation of painting (eight cases; 95% CI, 1.0–4.6). When adjusted for social class, the RR fell to 2.0 (95% CI, 0.86–3.9).

(*b*) *Cohort studies or studies within a cohort*

(i) *Painters*

Chiazze *et al.* (1980) studied workers in ten automobile assembly plants in five large companies in the USA. The plants were selected because of large numbers of employees, similar spray-painting operations, geographic dispersion and adequate records. The study was based on 4760 deaths among active and retired workers from 1970 or 1972 through 1976. A total of 4215 decedents were eligible for study, and employee work records were reviewed; for 253, work histories could not be obtained. The analysis was restricted to white males, who comprised about 80% of the decedents; 226 were spray painters. There was no significant excess proportion of deaths from any cause among spray painters, using either external local deaths or internal non-spray painters deaths. Lung cancer (21 deaths), which was the focus of the study, occurred more frequently among spray painters (PMR, 141) than in the local populations but not more frequently than among other automobile assembly workers (PMR, 108). PMRs greater than unity were noted also for leukaemias and lymphomas and for tumours of the brain, prostate, buccal cavity and pharynx. A nested case–control study covered 263 automotive workers who had died from lung cancer; they were matched by age within two years and by plant of employment with 1001 controls who had died of either cardiovascular disease or accidents. Spray painting was associated with a nonsignificant RR of 1.4 for lung cancer, and there was no indication of a dose–response relationship in association with exposure. The RR for those who had first been exposed at least 15 years prior to death was 1.0. The authors noted that individuals who had worked for only a few years may not have been included among the deaths if they had not been identified by an insurance claim in the company beneficiary file.

Englund (1980) and Engholm and Englund (1982) studied a cohort of 30 580 members of the Swedish painters' union from 1966 to 1974 for mortality and to 1971 for cancer morbidity by matching with national registers. The loss to follow-up was 1%. The SMR for all causes among painters was 102 (2740 cases), and the SIR for cancer was 109 ($p = 0.01$; 647 cases). Excesses were seen for cancers of the oesophagus (17 cases; SIR, 215 [95% CI, 124–340]), liver and bile ducts (12 cases; SIR, 200 [103–349]), lung (81 cases; SIR, 128 [106–152]) and larynx (14 cases; 177 [97–297]) and for lymphatic leukaemia (13 cases; 173 [92–296]). In a study based on population-based registries, about 38 000 painters in the 1960 census were linked to the national cancer registry, 1960–73. Among the 2064 cancers in painters, excesses were seen for cancers of the oesophagus (38 cases; SIR, 148) and of the intrahepatic bile duct (eight cases; SIR, 172). There was also a two-fold excess of pleural

tumours based on six cases. The SIR for all cancers was shown to increase with increasing number of years since entry into the union (Engholm & Englund, 1982). The authors suggested in an abstract that smoking habits were no different among painters than among other groups (Engholm *et al.*, 1987).

Dalager *et al.* (1980) examined the risks for cancer among spray painters employed in the aircraft maintenance industry, where there was exposure to zinc chromate primers. Deaths among painters were compared with those expected among US white males using PMRs. The PMRs for all cancers (136, 50 cases) and for lung cancer (184, 21 cases) were significantly raised. The PMRs for cancers at several other sites were increased but not significantly so. The PMR for respiratory cancers increased with duration of employment.

In a study of 2609 male painters belonging to two painters' unions in the Copenhagen area, Mikkelsen (1980) found no increased risk for all cancers combined when the number of cases (82) was compared with those among men in a bricklayers' union (RR, 1.1; 95% CI, 0.8–1.6) or with those among all Copenhagen men (RR, 1.0; 95% CI, 0.8–1.3). Results were not reported for specific sites.

Whorton *et al.* (1983) followed up a group comprising 6424 union members residing in the San Francisco/Oakland Standard Metropolitan Statistical Area, representing six occupations: asbestos workers, bakers, painters, plasterers, plumbers and roofers. Individuals were considered to be members of the cohort if they appeared on union records in July 1976 and 1977. Incident cases of cancer were identified by computer linkage of union rosters to the California Tumor Registry, and the registry's age–, sex– and year–specific incidence rates were used to calculate expected numbers of cancer cases and SIRs. An increased incidence of cancer of the trachea, bronchus, lung and pleura was seen among painters (15 cases; SIR, 199 [95% CI, 112–330]). Relative risks in excess of unity were also observed for leukaemia and for cancers of the prostate and bladder. The authors pointed out that about 15% of all cohort members were of unknown vital status but were assumed to be alive.

In a cohort mortality study of US paint applicators, primarily in new constructions and maintenance, the records of a large international union of painters and allied tradesmen were used (Matanoski *et al.*, 1986). The cohort consisted of 57 175 men who had been born prior to 1940, had had at least one year of union membership, had been members of the union in 1975–79 in four states in different geographical areas, and had died in 1975–79. A total of 1271 (2.2%) individuals were lost to follow-up. Altogether, 5313 deaths occurred (SMR, 88, based on US white male rates). Death certificates were available for all but 288 (5.4%); SMRs were not significantly elevated for cancers at individual sites. Since there was no direct information on individual worker's trades, data from local union chapters were used to define the usual trade of their members; 58% of the cohort belonged to mixed painting locals. Using the US white male population for comparison, significant excess mortality ratios were seen in local chapters for painters for all malignant neoplasms (SMR, 110; 95% CI, 103–117), stomach cancer (136; 101–180) and lung cancer (118; 106–132), and nonsignificant ratios for cancers of the large intestine (111; 90–136), liver (156; 95–241), bladder (126; 90–172) and kidney (141; 93–205) and for leukaemia (116; 82–160) (see also Table 23). When the risks of men in local mixed painting chapters were compared with those of men in special-

ty locals, the mixed painters had significantly higher mortality from all causes, from malignant neoplasms, from lung cancer, from bladder cancer and from leukaemia. [The Working Group noted that the fact that all painters had to have been active dues-paying members at some time during the follow-up period would tend to have enhanced the 'healthy worker' effect in this population.]

A nested case–control study was conducted of lung cancer incidence in the New York unions included in the study described above (Stockwell & Matanoski, 1985). The 124 male lung cancer cases were identified through the New York State Cancer Registry, and 371 controls without cancer were selected randomly from the union membership and stratified by birth date and geographical location of the unions. Responses to questionnaires on work history, work environment and life-style factors were received from 69 (66%) of the cases and 182 (59%) of the controls; of these, 65 (94%) and 55 (33%) were completed by a proxy for cases and controls, respectively. Painting as the reported usual trade was associated with a high risk (RR, 2.8; 95% CI, 1.5–5.2); high risks were also seen for work in allied trades: painter as a union speciality (RR, 3.2; 95% CI, 1.4–7.1) and ever having worked as a painter (RR, 2.6; 95% CI, 1.3–4.9). In the 57 cases for which the information was available, 53 men were reported to have used spackling compounds (probably containing asbestos), compared with 112 of 161 controls (RR for spackling, 5.2; 95% CI, 1.9–14.5). The authors attempted to adjust for several variables, including asbestos exposure (on the basis of use of spackling compounds). The risk for lung cancer among painters who never wore a respirator remained high (5.4; 95% CI, 1.0–29.3). [The Working Group noted that a high proportion of cases reported using spackling compounds and questioned the accuracy of information obtained from a proxy regarding use of painting materials and of respirators.]

All 93 810 incident cases of cancer recorded in 1970–79 at the Danish Cancer Registry were linked with information on longest employment held submitted by the Supplementary Pension Fund (Olsen & Jensen, 1987). The standardized proportionate incidence ratios (SPIRs) for cancer were reported for each cancer site in each industry and occupation on the basis of the expected proportion of that cancer in all industries. Painters in the construction industry had an increased proportion of lung cancers compared to people in other occupations (SPIR, 149; 95% CI, 119–185; based on 79 cases). Workers in the paint, varnish and lacquer manufacturing industries had an increased proportion of cancers of the nasal cavity and sinus, with a SPIR of 620 (95% CI, 155–2480; based on two cases). In a follow-up study of cases registered through 1984 (Olsen, 1988), the SPIR was reduced to 401 (67–1324) based on two cases of sinonasal cancer. Car painters had a SPIR of 1403 (198–9958) for nasal cavity and sinus cancers based on one case. Several other proportions were above one for these three groups, but the excesses were not significant (Olsen & Jensen, 1987).

(ii) *Paint manufacturers*

Bertazzi *et al.* (1981) followed a small cohort of 427 workers employed in paint manufacturing in Italy. The workers had to have been employed for at least six months at any time from 1946 through 1977 to be eligible for inclusion and were followed for 1954–78. The follow-up was 97.7% complete. There was a significant excess of all cancers in this population (18 cases; SMR, 184; 95% CI, 112–285) when national rates were used as the comparison.

Lung cancers occurred at significant excess when either national (eight cases; SMR, 334; 106–434) or local rates (227; 156–633) were used as a standard, and the risk increased with length of exposure and with latency. These workers were exposed to asbestos as well as to chromate pigments.

A similar study of a larger cohort of 16 243 US male workers in the paint and coating manufacturing industry was reported by Morgan *et al.* (1981). These men had been employed for one year or more after January 1946 in 12 large and 20 medium to small companies and were followed through 31 December 1976. Only plants that retained personnel records for at least 15 years were eligible for the study, and out of 47 eligible plants the 32 largest were finally studied. The overall follow-up rate of the cohort was about 94%. Death certificates could not be obtained for 8.2% decedents. There were 2633 deaths in all (SMR, 86). The cohort was divided into seven subgroups on the basis of their exposures as determined from individual job histories; individuals could appear in multiple exposure groups. Deaths from cancers of the colon and rectum occurred at higher rates in the total population than expected on the basis of numbers among US white males (colon: 65 cases; SMR, 138 [95% CI, 107–176]; rectum: 26 cases; SMR, 139 [91–204]). The risk for respiratory cancers, which was a major focus of the study, was not excessive in this population (SMR, 98; 160 cases); information on smoking habits was not available. Deaths from cancer of the liver and biliary passage occurred more frequently than expected in the subgroups of workers potentially exposed to pigments (seven cases; SMR, 273 [108–555]) and lacquer (five cases; SMR, 255 [81–583]). The SMR for leukaemia was 212 (eight cases [92–418]) in the subgroup of workers exposed to lacquer. A further report on this study (Morgan *et al.*, 1985) provided little additional information.

A small cohort of 416 men who had worked for five years or more in the Swedish paint manufacturing industry during the period 1955–75 were followed for mortality in the years 1961–81 (Lundberg, 1986). Reference numbers were taken from national statistics. Subjects were categorized into lower and higher exposure levels according to duration and intensity of exposure. Overall mortality was low (96 cases; SMR, 88), as was mortality from all cancers (22 cases; SMR, 84; 95% CI, 52–127) and from lung cancer (three cases; SMR, 63; 95% CI, 12–184). The SMR for multiple myeloma was 549 (three cases; 95% CI, 113–1606) and that for cancer of lymphatic and haematopoietic tissues 212 (five cases; 95% CI, 68–496). The three cases of multiple myeloma occurred in workers in the higher exposure category.

(*c*) *Case–control studies*

(i) *Cancers at multiple sites*

Cancer cases recorded at a cancer centre in New York State, USA, in 1956–65 were compared with all patients with non-neoplastic lesions in regard to occupations related to inhalation of combustion products or chemicals and to personal characteristics (Viadana *et al.*, 1976; Decouflé *et al.*, 1977; Houten *et al.*, 1977). The information was obtained through an inverview at the time of admission for all patients. Each of the 11 591 white male subjects was included for analysis for each occupation held; specific occupations were compared with those of an unexposed clerical group. Painters were analysed as a subgroup of people with chemical exposures and as a subgroup of those with metal-related occupations. Cancer sites

for which RRs were increased were: lung (42 cases; RR, 1.7; $p = 0.02$), stomach (eight cases; 2.4; $p = 0.05$), oesophagus (seven cases; 3.0; $p = 0.03$), prostate (nine cases; 1.9), bladder (16 cases; 1.6), kidney (four cases; 2.6) and melanoma (two cases; 3.2). The highest RR was seen in the age group below 60 years for stomach cancer (12.6); for oesophageal cancer, the risk was greater for the age group above 60 years (3.8). These two ratios were even higher among painters with five or more years of exposure (16.6 and 6.9, respectively). For lung and prostate cancer, no such dose–response relationship was observed. The elevated lung cancer risk among painters was no longer significant after adjustment for smoking and age (RR, 1.7). The author noted that the risk for stomach cancer was elevated in more than half of the occupations, which might be explained by the eastern European origin of the workers. No adjustment was made for alcohol drinking (see also Tables 24 and 25).

Coggon *et al.* (1986a,b) identified all cases of cancer in three English counties where chemical, metal and vehicle production industries were situated, using hospital and cancer registration records for the period 1975–80. Males aged 18–54 were included in the study. Occupational and smoking histories were obtained either by mailed questionnaires (response rate, 52.1%) or from information in hospital records or on death certificates. A total of 2942 cancer cases were identified, and cases of cancer at 15 specific sites were compared with those at all other sites with regard to occupation. Data were corrected for age, residence, source of history and smoking. Laryngeal cancer was more likely to be associated with painting and decorating (RR, 3.4; 95% CI, 1.3–9.0; six cases) than with other occupations; bronchial cancer was also associated with painting, the RR being 1.3 (20 cases; see also Table 24). A borderline significant association was seen for cancer of the stomach (RR, 2.3; 95% CI, 1.0–5.0); other sites for which the RR was above unity were oral cavity (RR, 1.9; five cases), skin (RR, 1.4; four cases), testes (RR, 1.9; nine cases) and malignant melanoma (RR, 1.6; four cases). The authors commented that five patients with testicular cancer had worked as paint sprayers, which results in a RR of 4.9 (95% CI, 1.3–18.2). A nonsignificant RR of 0.7 was found for bladder cancer (see also Table 25).

In the same area of the UK, Magnani *et al.* (1987) examined occupations associated with cancer at five sites – oesophagus, pancreas, melanoma, kidney and brain. Deaths from these cancers in men aged 18–54 for the period 1959–63 and 1965–79 were matched by year of death, age at death and residence to those among four controls who had died from other causes. Occupation and industry were identified from death certificates. No significant risk for any of the cancers was associated with exposure to painting and decorating; however, the RR for oesophageal cancer was 2.0 (95% CI, 0.8–4.9) and that for brain cancer, 1.4 (95% CI, 0.7–2.8). The investigators also described exposures for each occupation, summed these across occupations, and examined the risks of these substances as they relate to the cancers. In this analysis, paints were associated with only a small increase in RR for three cancers – oesophageal and brain cancers and melanoma; none of the associations is significant. The authors noted that only the most recent full-time job was recorded on the death certificate. No adjustment was made for smoking or alcohol drinking.

Table 24. Case–control and other studies of lung cancer among persons exposed in paint manufacture and painting

Reference	Location, time	Type of controls	Source	Exposure	No. of cases (no. of painters)	RR	95% CI	Comments
Case–control studies								
Wynder & Graham (1951)	USA, NG	[Unclear]	Interview		857 (200 fume-exposed; 11 painters)	NG	NG	
Breslow *et al.* (1954)	USA, 1949–52	Hospital	Interview	Construction and maintenance painters for ≥5 years	518 (22)	1.9	0.93–3.8	Not adjusted
Menck & Henderson (1976)	USA, 1968–70	Estimated population by industry	Death certificates, hospital records	Painter at diagnosis	2161 (45)	SMR, 158	NG	Significant; adjusted for age
Milne *et al.* (1983)	USA, 1958–62	Deaths from other causes (except pancreas, bladder, nasal, kidney, haematopoietic)	Death certificates, occupation	Painter	925 men (24)	1.8	NG	Significant ($p < 0.01$); adjusted for age
				Paint manufacture	(3)	0.7	NG	Not significant
Kjuus *et al.* (1986)	Norway, 1979–83	Hospital	Interview and worksite records	Painting and paperhanging	176 men (5)	1.7	0.4–7.3	Occupation is longest job held; considered exposed if ≥3 years; adjusted for smoking
				Paints , glues, lacquer	17	1.2	0.6–2.6	
Lerchen *et al.* (1987)	USA, 1980–82	Population and rosters of elderly	Interview	Ever construction painters	333 men (9)	2.7	0.8–8.9	Adjusted for age, ethnicity and smoking
				Asbestos	40	1.1	0.7–1.7	

Table 24 (contd)

Reference	Location, time	Type of controls	Source	Exposure	No. of cases (no. of painters)	RR	95% CI	Comments
Siemiatycki *et al.* (1987a)	Canada, 1979–85	Other cancers	Interview	Listed as white spirits, but in exposed group construction is 21% of total., mostly painters	857 males oat-cell 159 (36)	1.1	0.8–1.4	Adjusted for age, socio-economic status, ethnicity, cigarette smoking, blue/white collar; 90% CI
					squamous-cell 359 (92)	1.2	1.0–1.5	
					adenocarcinoma 162 (37)	1.0	0.7–1.3	
					other types 177 (32)	0.8	0.6–1.1	
				Long duration, high exposure	44	1.7	1.2–2.3	
				Construction workers		1.4	NG	
Levin *et al.* (1988)	China, 1984–85	Population	Interview	Ever painter	733 men (15)	1.4	0.5–3.5	Questionable trend; adjusted for age and smoking
Ronco *et al.* (1988)	Italy, 1976–80	Deaths without smoking-related diseases	Interview	Painter	164 men (5)	1.3	0.43–4.1	Adjusted for age, smoking and other employment in suspect high-risk occupations
Multisite case-control studies								
Viadana *et al.* (1976); Decouflé *et al.* (1977); Houten *et al.* (1977)	USA, 1956–65	Noncancer admissions	Interview at admission	Painter	(42)	1.7	NG	Significant; adjusted for age; non-significant when adjusted for smoking and age
Coggon *et al.* (1986a)	UK, 1975–80	Other cancers	Interview	Painter	738 men (20)	1.3	NG	Adjusted for age, smoking, residence, respondent

RR, relative risk; CI, confidence interval; NG, not given; SMR, standardized mortality ratio

Table 25. Case–control studies of lower urinary tract cancer among persons exposed in paint manufacture and painting

Reference	Location, time	Type of controls	Source	Exposure	No. of cases (no. of painters)[a]	RR	95% CI	Comments
Wynder *et al.* (1963)	USA, 1957–61	Hospital, without smoking-related disease	Interview	Ever painter	300 (18)	[2.2]	[1.0–4.5]	No adjustment for smoking
Cole *et al.* (1972)	USA, 1967–68	General population	Interview	Painter	461 (28 men)	1.2	0.71–1.9	Adjusted for age and smoking
Howe *et al.* (1980)	Canada, 1974–76	Neighbourhood	Interview	Commercial painting	480 men (≥24)	1.0	0.6–2.3	Unadjusted. After correction for exposure to other suspect 'high-risk' industry, RR for spray painter, 1.0
				Ever spray painting	(≥16)	1.8	0.7–46	
Silverman *et al.* (1983)	USA, 1977–78	Population	Interview	Ever painter	303 men (15)	1.0	0.5–2.2	Unadjusted
				Car painter	(3)	0.5	0.1–2.1	
				Paint manufacture	(1)	0.2	0–2.2	
Schoenberg *et al.* (1984)	USA, 1978–79	Population	Interview	Ever painter	658 men (34)	1.4	0.85–2.3	Adjusted for age, smoking and other employment
				Paint exposure	(111)	1.6	1.2–2.1	
Vineis & Magnani (1985)	Italy, 1978–83	Hospital; other urological and surgical	Interview	Painter in building industry	512 men (12)	1.0	0.40–2.2	Adjusted for age and smoking
				Car painter ≥5 years	(7)	2.0	0.60–7.0	
				Carpentry painter	(1)	0.6	0.04–8.4	
				Spray painter in different industries	(2)	1.2	0.20–5.8	
Morrison *et al.* (1985)	USA, UK, Japan, 1976–78	Population	Interview	Paint and paint manufacture	USA, 430 (35)	1.5	0.9–2.4	Adjusted for age and smoking; 90% CI
					UK, 399 (23)	0.7	0.5–1.2	
					Japan, 226 (5)	0.7	0.3–1.7	
Claude *et al.* (1988)	FRG, NG	Hospital urological and homes for elderly	Interview	Ever painter	531 men (15)	1.3	0.59–2.7	Trend, p = 0.04 for exposure to spray paints
				Lacquer and paint	(78)	1.5	1.1–2.2	
				Spray paints	(52)	2.9	1.7–4.9	
Jensen *et al.* (1987)	Denmark, 1979–81	Population	Interview	Different painting industries	371 (13)	2.5	1.1–5.7	Adjusted for age, sex and smoking
				Painter 10 years		1.4	1.0–1.9	

Table 25 (contd)

Reference	Location, time	Type of controls	Source	Exposure	No. of cases (no. of painters)[a]	RR	95% CI	Comments
Iscovich *et al.* (1987)	Argentina, 1983–85	Neighbourhood and hospital	Interview	Ever painter	117 (3)	0.55	[0.12–2.5]	Adjusted for age and tobacco smoke, pooling the two control groups
Schifflers *et al.* (1987)	Belgium, 1984–85	Population	Interview	Painter in high-risk occupation	74 (NG)	NG	NG	No increased risk reported
Risch *et al.* (1988)	Canada, 1979–82	Population	Interview	Exposed to paints in full-time job at least 6 months, 8–28 years before diagnosis	781 (204 men, 14 women)	1.1 3.9	0.77–1.6 0.9–26.7	Adjusted for smoking
				Commercial painting	(49 men)	0.90	0.39–2.1	
				Spray painting	(67 men)	0.91	0.48–1.7	
Siemiatycki *et al.* (1987a)	Canada, 1979–85	Other cancers	Interview	Listed as white spirits, but in exposed group construction is 21% of total, mostly painters	486 (91)	1.0	0.8–1.2	Adjusted for age, socioeconomic status, ethnicity, cigarette smoking, blue/white collar work; 90% CI
Multisite studies								
Coggon *et al.* (1986b)	UK, 1975–80	Other cancers	Interview	Painter	179 (10)	0.7	NG	Adjusted for age, smoking, residence, respondent; bladder and renal pelvis; men aged 18–54 only
Viadana *et al.* (1976); Decouflé *et al.* (1977); Houten *et al.* (1977)	USA, 1956–65	Noncancer admissions	Interview at admission	Painter	(16)	1.6	NG	Not significant

[a]If only discordant pairs noted, no. of painters $\geq$ number of discordant pairs given

RR, relative risk; CI, confidence interval; NG, not given

[The Working Group noted that the populations studied by Coggon *et al.* (1986a,b) and Magnani *et al.* (1987) may overlap and that only deaths in relatively young men were considered.]

(ii) *Cancer of the lung*

These studies are summarized in Table 24.

In an early descriptive study, Wynder and Graham (1951) studied a total sample of 857 incident cases of lung carcinoma diagnosed in one hospital in St Louis, MO, USA, over an unspecified period. Of 200 who were 'believed or known to have been exposed to irritative dusts and/or fumes', 11 were painters. [The Working Group found it difficult to clarify the information on the comparison groups.]

Breslow *et al.* (1954) identified 518 cases of lung cancer in 11 Californian hospitals during the period 1949–52. Controls were selected from patients admitted to the same hospital for a condition other than cancer or a chest disease, and matched for age, sex and race. Detailed occupational and smoking histories were obtained by interview. The authors reported that 22 cases had been employed as construction or maintenance painters for at least five years, as had 12 controls [RR, 1.9; 95% CI, 0.93–3.8]. Smoking was not controlled for, although smoking histories had been recorded.

Menck and Henderson (1976) identified deaths from lung cancer for the years 1968–70 (2161 cases) and incident cases of lung cancer for the years 1972–73 (1777 cases) from the Los Angeles County Cancer Surveillance Program. Both were classified by occupation and industry on the basis of either death certificates or hospital records. Of the 3938 subjects, 689 had no reported occupation and 1222 no reported industry of employment. Employment of the population aged 20–64 was estimated from a sample of the population in the 1970 census, and the risk of lung cancer for each occupation was compared to the risk in the total population. The SMR for lung cancer in painters was significantly elevated (45 deaths; SMR, 158; $p < 0.01$; see also Table 24).

Milne *et al.* (1983) compared the occupation and industry of 925 (747 male and 178 female) cases of lung cancer in Alameda County, California, USA, 1958–62, with those of people who had died of other cancers. Usual occupation and industry as stated on the death certificate were coded using the US census classification. When occupations were examined separately, male painters had a significantly increased risk for lung cancer (24 cases) when compared either with all cancer deaths (RR, 1.7; $p < 0.05$) or with those dying of cancers other than of the pancreas, nasal sinus, kidney, bladder, bone and haematopoietic organs (RR, 1.8; $p < 0.01$). There was no increased risk associated with employment in the paint manufacturing industry (RR, 0.7; three cases; see also Table 24).

A study of 176 male incident lung cancer cases, under 80 years of age, admitted in 1979–83 to two hospitals in two neighbouring counties in Norway was conducted by Kjuus *et al.* (1986). Controls were matched on age through admission lists or from the same department records; persons with physical or mental handicaps, general poor health or an admission diagnosis of chronic obstructive pulmonary disease were excluded from the control group. Occupational histories were determined by interview and work site records then coded by job title and separated into three groups according to potential exposure to lung

carcinogens, which included painting and paints. Three years was considered to be the minimal exposure classified as positive, and occupation was classified as the longest job held; exposures were included only up to 1970. Within the group, the RR for painting and paperhanging was 1.7 (95% CI, 0.4–7.3; five cases), adjusted for smoking. The RR for lung cancer associated with exposure to paints, glues and lacquer was 1.2 (95% CI, 0.6–2.6; 17 cases), adjusted for smoking, in comparison with all other subjects.

Occupational histories obtained by interview were compared in a case–control study of 506 lung cancer patients (333 men and 173 women) diagnosed in 1980–82, according to the population-based New Mexico Tumor Registry, and 771 controls selected through random telephone numbers or from rosters of elderly (Lerchen *et al.*, 1987). Next-of-kin provided the information for half of cases and 2% of controls. Jobs held by individuals from age 12 years were classified according to an a-priori list of potentially hazardous occupations. Construction workers and painters were included in high-risk occupations; employment for one year or more was classified as ever having been employed in an industry. The RR for lung cancer in men associated with employment as a construction painter was 2.7 (nine cases; 95% CI, 0.8–8.9) compared to never having been employed in that occupation and adjusted for age, ethnicity and smoking.

In the study of Siemiatycki *et al.* (1987a,b), described in detail in the monograph on some petroleum solvents (p. 70), construction workers exposed to white spirits, many of whom were painters, were described as having an excess risk for lung cancer (RR, 1.4 [numbers not given]).

In a cancer registry-based case–control study, Levin *et al.* (1988) identified 833 male lung cancer cases diagnosed between February 1984 and February 1985 in Shanghai, China, and 760 randomly selected male controls from the general urban Shanghai population, frequency matched within five-year age strata. Personal interviews to obtain occupational and smoking histories were obtained for 733 cases and 760 controls. More than 60 industries and occupations were examined; ever *versus* never having worked as a painter was associated with a RR, adjusted for age and smoking, of 1.4 (95% CI, 0.5–3.5). The RR varied according to duration of employment as a painter as follows: < 10 years, 1.9 (seven cases); 10–19 years, 2.8 (two cases); 20–29 years, 2.2 (five cases); ≥30 years, 0.3 (one case; questionable trend). The authors cited multiple comparisons and the use of broad occupational groups as limitations of the study.

Ronco *et al.* (1988) reported a population-based case–control study from two areas in northern Italy which included 164 male lung cancer cases identified from death records during 1976–80 and 492 controls who had died of conditions other than chronic lung disease or smoking-related cancers. Information on smoking and occupation was obtained through interviews of next-of-kin. Many exposures suspected of increasing the risk for lung cancer were evaluated, and individuals who had not held any job in any industry that was associated with exposure to a known or suspected lung carcinogen were classified as nonexposed. The RR for painters, adjusted for age, smoking and employment in other studied exposures, was 1.3 (five cases; 95% CI, 0.43–4.1).

Malker *et al.* (1985) examined the risk for pleural mesothelioma in relation to occupational exposures, including painting. The investigators used the Swedish population-based registries to link incident cancer cases during 1961–79 with 1960 census data on occupation and industry. Altogether, 318 cases of pleural mesothelioma occurred. Standardized incidence ratios (SIR) were calculated for occupations and industrial categories. For workers in the construction industry as a whole, a significant SIR of 1.6 was seen based on 63 cases; painting as a specific industry comported a higher significant SIR (2.9, based on 13 cases); painters and paperhangers as a specific craft showed an SIR of 2.0 (based on 12 cases), which was significant. [The Working Group noted that painters in the construction industry are probably exposed to asbestos.]

(iii) *Cancer of the larynx*

A case–control study of incident laryngeal cancer was carried out by Brown *et al.* (1988) in Texas. Cases consisted of all diagnoses of primary laryngeal cancer among white males aged 30–79 selected from 56 participating hospitals, comprising 220 living cases and 83 dead cases identified during the period 1975–80. Controls consisted of an equal number of white males without respiratory cancer selected from various sources and frequency matched on age, vital status, ethnicity and county of residence. Occupational exposures were examined, controlling for cigarette smoking and alcohol consumption. The RR for painters was elevated (11 cases; RR, 2.3; 95% CI, 0.84–6.3), and a significantly elevated risk was found for workers reportedly exposed to paint (32 cases; RR, 1.8; 1.0–3.2). No clear pattern was evident by duration of exposure.

(iv) *Cancer of the urinary tract*

These studies are summarized in Table 25.

Wynder *et al.* (1963) examined occupational and other risk factors associated with bladder cancer in 300 male patients from seven New York hospitals in 1957–61. Controls consisted of an equal number of male hospital patients who did not have myocardial infarction or cancers of the respiratory system or upper alimentary tract and were matched by age and time of admission. Interviews were conducted directly with the patients. The investigators reported 18 painters among cases and 12 among controls. [The Working Group calculated the RR to be 2.2 (95% CI, 1.0–4.5) for the group that had ever worked as a painter; no adjustment was made for cigarette smoking.]

Cole *et al.* (1972) conducted a case–control study of transitional- or squamous-cell carcinoma of the lower urinary tract in eastern Massachusetts using newly diagnosed cases aged 20–89 during an 18-month period ending 30 June 1968 (Cole *et al.*, 1971). Out of 668 cases ascertained, a random sample of 510 was selected for interview; a usable occupational history was obtained for 461. Controls were selected from the general population of the same area and matched on age and sex. Certain occupations (including painting) were classified as 'suspect'; and each of these groups was compared to nonsuspect industries. The RR for lower urinary tract cancer in male painters, adjusted for age and smoking, was 1.2 (28 cases; 95% CI, 0.71–1.9).

Howe *et al.* (1980) conducted a case–control study of bladder cancer in three areas of Canada; they identified 821 cases through provincial cancer registries in 1974–76 and

matched them by age, sex and neighbourhood to 821 controls. Personal interviews were obtained for 632 cases (480 men and 152 women; 77%) and an equal number of controls. Among men, working as a painter was not associated with a risk: the RR for commercial painting was 1.0 (24 cases in discordant pairs; 95% CI, 0.6–2.3); that for spray painting was 1.8 (16 cases in discordant pairs; 0.7–4.6), which was reduced to 1.0 after correction for exposure in other suspect 'high-risk' industries.

As part of the US National Bladder Cancer Study, Silverman *et al.* (1983) conducted a population-based case-control study of bladder cancer in the Detroit, MI, USA, area. They identified 420 male cases diagnosed with transitional- or squamous-cell carcinoma of the lower urinary tract aged 21–84 between 1977–78; interview was obtained for 339 (81%), but the analysis was restricted to 303 white males. Controls were 296 white males stratified for age who were selected from a random digit-dialling survey for those under age 65 and from a random sample of the Health Care Financing Administration lists for those over 65. Employment was measured as 'ever' or 'usual' occupation or industry; 'usually unexposed' were those not employed in the industry of interest. The findings suggest no increased risk for bladder cancer for painters in general (15 cases; RR, 1.0; 95% CI, 0.5–2.2), for painters in the automobile industry (three cases; 0.5; 0.1–2.1) or for paint manufacturers (one case; 0.2; 0–2.2).

A similar case-control study of bladder cancer in 658 white male incident cases aged 21–84 during 1978–79 and of 1258 population controls was conducted in New Jersey, USA, by Schoenberg *et al.* (1984). Controls were selected as by Silverman *et al.* (1983). The RR for bladder cancer in men ever employed as painters, adjusted for age, was 1.4 (34 cases; 95% CI, 0.85–2.3). When occupations were classified by materials used, paint exposure was associated with a risk for bladder cancer (111 cases; RR, 1.6; 95% CI, 1.2–2.1). The risk was higher for those first exposed under age 41 and did not increase with duration of exposure.

A case-control study of bladder cancer in Italy (Vineis & Magnani, 1985) involved 512 male cases aged under 75 between 1978–83 and 596 hospital controls. The controls were matched by age and were subjects with benign urological conditions or surgical conditions. Occupational and smoking histories were obtained by interview. No increased risk was seen for painters in the building industry (RR, 1.0; 95% CI, 0.40–2.2; 12 cases), painters in carpentry (RR, 0.6; 0.04–8.4; one case) or spray painters (RR, 1.2; 0.20–5.8; two cases), but the RR for car painters was 2.0 (95% CI, 0.60–7.0; seven cases).

Morrison *et al.* (1985) examined 15 occupations and the risk for lower urinary tract cancer in Nagoya, Japan (1976–78), Manchester, UK (1976–78), and Boston, USA (1976–77), using incident male cases aged 21–89 and population-based controls. They identified 741 cases in Boston, 577 in Manchester and 348 in Nagoya. Interviews were obtained for 81% of the cases in Boston, 96% in Manchester and 84% in Nagoya; the corresponding figures for the controls were 80%, 90% and 80%. The analysis was limited to 430 cases and 397 controls in Boston, 399 cases and 493 controls in Manchester and 226 cases and 443 controls in Nagoya, for whom smoking histories were known. Occupational exposure to paint or paint manufacture was associated with a risk of bladder cancer only in the Boston population (35 cases; RR, 1.5; 90% CI, 0.9–2.4). This ratio was controlled for age and smoking history. [The

Working Group noted that no specific information was available on how the controls were selected.]

Two publications from the Federal Republic of Germany (Claude *et al.*, 1986, 1988) reported two hospital-based case–control studies of tumours of the lower urinary tract. A total of 340 men and 91 women with such cancer between 1977–82 were matched by age and sex to either hospital patients primarily from urology wards or, for those over 65, to people in homes for the elderly. Subjects were interviewed about occupations, specific exposures and life-style factors. There was no reported excess risk for the occupational category of painting, but the RRs associated with specific exposures suggested a risk of painting in men. Spray painting was associated with an increased risk for cancer of the lower urinary tract (RR, 4.7; 95% CI, 2.1–10.4; 28 cases in discordant pairs), as was exposure to lacquer (RR, 1.6; 95% CI, 0.98–2.5; 45 cases in discordant pairs; Claude *et al.*, 1986). In order to examine occupational risks more extensively, an additional 191 male cases were included, to make a total of 531 (Claude *et al.*, 1988). Painting as an occupation was associated with an increased risk for bladder cancer (RR, 1.3; 95% CI, 0.59–2.7; 15 cases). An examination of the specific exposures indicated significant excess risks for cancer of the lower urinary tract for any exposure to spray paints (RR, 2.9; 95% CI, 1.7–4.9; 52 cases), to lacquer and paints (RR, 1.5; 95% CI, 1.1–2.2; 78 cases) or to chromium/chromate (RR, 2.2; 95% CI, 1.4–3.5). After correction for smoking, a significant trend of increased risk with increasing duration of exposure for individuals exposed to spray paints and chromium/chromate could be seen. [The Working Group questioned the choice of controls and considered that there may have been overlap between the exposure categories.]

Jensen *et al.* (1987) carried out a case–control study of bladder cancer in Denmark and interviewed 371 patients with invasive and noninvasive lesions diagnosed during 1979–81. The occupations of cases were compared with those of 771 controls selected from residents in the same area. Detailed occupational histories were taken, which included industry, type and place of work and duration; the information was coded according to industry. Significantly more cases than controls were employed in furniture lacquering and painting, industrial painting, sign-post painting, painting firms or car painting (13 cases; RR, 2.5; 95% CI, 1.1–5.7). Employment as a painter for ten years gave a RR of 1.4 (95% CI, 1.0–1.9).

Iscovich *et al.* (1987) performed a case–control study of 117 bladder cancer cases diagnosed in Argentina in 1983–85 and individually matched on age and sex to one neighbourhood and one hospital control. Hospital controls were selected from the same hospital as the case; about 12% of patients had diseases known to be associated with tobacco smoking. Neighbourhood controls were selected from among persons living in the same street block as the cases. A detailed questionnaire, containing information on smoking, demographic, socioeconomic and medical variables and occupational history for the three occupations of longest duration as well as the most recent one was administered. No increased risk for bladder cancer was observed among painters (three cases; RR, 0.55; [95% CI, 0.12–2.5]).

A pilot case–control study of bladder cancer in Belgium in 1984–85 (Schifflers *et al.*, 1987) included 74 cases and 203 population controls selected from electoral rolls and matched for age and sex. While cases were interviewed by the investigators, most of the

controls were interviewed by others. A group of 16 jobs, including painting, were defined as hazardous and associated with a high risk for bladder cancer, but exposure to painting as a specific job did not show a significant excess.

A case–control study from Denmark (Jensen *et al.*, 1988) concentrated on cancers of the renal pelvis and ureter. The 96 cases, aged below 80, were identified from 27 hospitals in 1979–82, and three hospital controls were matched to each case on hospital, age and sex. Patients with urinary tract and smoking-related diseases were not eligible as controls. Questionnaire data on smoking and on occupation and occupational exposures were obtained. An elevated risk for upper urinary tract cancer was associated with occupational exposure as painter or paint manufacturer (RR, 1.8, adjusted for sex and lifetime tobacco consumption; 95% CI, 0.7–4.6; ten cases).

A case–control study of bladder cancer was carried out during the period 1979–82 in Alberta and in Toronto, Ontario (Risch *et al.*, 1988). Cases aged 35–79 were identified through a cancer institute, from a province-wide tumour registry in Alberta, and through review of hospital records in Ontario. Interviews were carried out with 835 (67%) of the cases (826 histologically verified) and 792 (53%) of the controls about jobs in 26 industries that had previously been examined in studies of bladder cancer, and on occupational exposures to fumes, dust, smoke and chemicals. The analysis was carried out on the 781 matched sets for which adequate information was available. Occupational exposure to paints in a full-time job for at least six months, eight to 28 years before diagnosis was not associated with an increased risk for bladder cancer in men (age-adjusted RR, 1.1; 95% CI, 0.77–1.6; 204 cases) but it was for women (RR, 3.9; 0.9–26.7; 14 cases). Little difference in risk was seen between commercial (RR, 0.90; 95% CI, 0.39–2.1; 49 cases) and spray (RR, 0.91; 95% CI, 0.48–1.7; 67 cases) painting in men. The authors noted the problems associated with the very low response rate, the inclusion of cases with borderline malignancies and the potential for recall bias.

In the study of Siemiatycki *et al.* (1987a,b) (see p. 70), an increased risk for bladder cancer was seen among people exposed to white spirits, 21% of whom worked in construction trades, mostly comprising painting.

(v) *Cancer of the biliary tract*

Cases of biliary tract cancers were identified from the National Swedish Cancer Registry for the period 1961–79, and the occupations of the patients identified from the 1960 census of occupations (Malker *et al.*,1986). SIRs were calculated using the incidence rates for the total population and data from the 1960 census with regard to occupation and industrial employment, adjusted by region as well as by age and sex. There were 1304 cases of gall-bladder cancer and 764 cases of other biliary tract cancers in men, and 947 and 346 cases, respectively, in women. Significant SIRs of 1.3 (32 cases) and 1.4 (19 cases) for male painters and paperhangers were reported for gall bladder and other biliary cancers, respectively.

(vi) *Cancer of the pancreas*

Norell *et al.* (1986) reported on both a case–control study of pancreatic cancer and a retrospective cohort study of workers based on registry data in Sweden during 1961–79. Information on occupation was obtained through questionnaires. The case–control study in-

cluded 99 cases of pancreatic cancer (aged 40–79) and 163 hospital controls of the same age and sex with inguinal hernia and 138 population controls of the same age, sex and residence. A significant excess risk was seen for exposure to paint thinners (ten cases; RR *versus* population controls, 2.5; 90% CI, 1.1–5.9; RR *versus* hospital controls, 1.4; 90% CI, 0.7–2.9). In the cohort study, a 20% excess of pancreatic cancer was seen in workers (aged 20–64) in paint and varnish factories (90% CI, 0.7–1.9) and a 30% excess for floor polishing (90% CI, 0.6–2.3).

(vii) *Haematopoietic neoplasms*

Studies on leukaemia are summarized in Table 26.

In a case–control study of leukaemia in three geographical areas of the USA in 1959–62, information was collected on occupations and other subjects by personal interview (Viadana & Bross, 1972). The controls were a random sample from households in the area matched for age and sex. The analysis was limited to 1345 adult leukaemia cases and 1237 adult controls in whites. No association was seen between any occupation and leukaemia in women. The risk for leukaemia in men appeared to be associated with work in the construction industry, and specifically with painting. The risk for painters *versus* nonpainters was 2.8 [1.4–6.0]; that in comparison with clerks was 3.1.

Timonen and Ilvonen (1978) interviewed 45 adults in northern Finland with acute leukaemia or chronic myeloid leukaemia between 1973–77 and a control group of 45 patients from the same hospital about use of drugs and chemicals, including paint. Four cases and four controls had been exposed to paint containing benzene derivatives and lead.

Flodin *et al.* (1986) performed a case–control study on acute myeloid leukaemia on 59 cases aged 20–70 years in 1977–82 from five hospitals in Sweden. Living patients and controls replied to a questionnaire about solvent exposure. Two series of controls were used: 236 matched for sex, age and residence, and 118 selected randomly from the same general population. For 'solvents, all kinds', there were 11 cases exposed and 58 controls (crude rate ratio, 1.2); no case but five controls were classed as 'painters'.

A population–based case–control study of 125 adult leukaemia cases and an equal number of controls matched for age, sex and residence was performed in Sweden in 1980–83 (Lindquist *et al.*, 1987). Information on occupation was obtained by a standardized questionnaire. 'Painters' included spray painters, car painters, machine painters, boat painters, asphalt painters and building painters. Thirteen cases and one control had been painters (RR, 13.0; 95% CI, 2.0–554). The median duration of exposure for painters was 16 years. After exclusion of case–control pairs with a 'painter', 26 patients and seven controls had worked in occupations which also involved exposure to paint and/or solvents and/or glues (RR, 3.7; 95% CI, 1.6–10.1).

Linet *et al.* (1988) linked records for Swedish men by major industry and occupational categories from the 1960 census to cancer registry data for 1961–79 to calculate SIRs for leukaemia subtypes. Expected numbers were based on a 19-year follow-up, taking account of age, region and birth cohort. Among men classified as painters or paperhangers, SIRs were 1.1, 1.0, 1.1 and 0.8 for acute lymphocytic, chronic lymphocytic, acute nonlymphocytic and chronic myelocytic leukaemia, respectively (based on three, 41, 33 and 14 cases, respectively).

Table 26. Case–control and other studies of leukaemia among persons exposed in paint manufacture and painting

Reference	Location, time	Type of controls	Source	Exposure	No. of cases (no. of painters)[a]	RR	95% CI	Comments
Viadana & Bross (1972)	USA, 1959–62	Population	Interview	Painter	845 men (31)	2.8	[1.4–6.0]	In comparison with non-painters
						3.1	NA	In comparison with clerks
Timonen & Ilvonen (1978)	Finland, 1973–77	Hospital	Interview	Paint containing benzene derivatives and lead	45 adults (4)	1.0	–	
Flodin *et al.* (1986)	Sweden, 1977–82	Population	Interview	Painter	59 adults			
Lindquist *et al.* (1987)	Sweden, 1980–83	Population	Interview	Painter	125 adults (13)	13	2.0–554	Adjusted for other exposures
				Other professions exposed to paint and/or solvents and/or glues		3.7	1.6–10.1	
				Daily exposure to organic solvents (white spirits) and gasoline		3.0	1.1–9.2	
				Organic solvent		2.0	(significant)	
				Petroleum products		1.4	(significant)	
Linet *et al.* (1988)	Sweden, 1961–79	Record linkage registry to census (cohort)	1960 Census record		(91)			
					Acute lymphocytic (3)	SIR, 1.1		
					Chronic lymphocytic (41)	SIR, 1.0		
					Acute nonlymphocytic (33)	SIR, 1.1		
					Chronic myelocytic (14)	SIR, 0.8		

A total of 25 cases of Hodgkin's disease in men aged 20–65 was studied in 1978–79 using two controls selected from the Swedish population registry (Olsson & Brandt, 1980). Subjects were asked about occupations, and occupational exposure was defined as handling organic solvents every working day for at least one year within the closest ten-year period. There was a significant association between Hodgkin's disease and exposure to solvents (12 cases; RR, 6.6; 95% CI, 1.8–23.8). Three of the 12 cases and only one of six controls exposed to solvents were painters (RR, 1.7; [0.09–54.6]); the RR for painters among all subjects was 6.7 [0.56–177.0]. [See also the monograph on some petroleum solvents.]

Vianna and Polan (1979) studied mortality in 1950–69 from reticulum-cell sarcoma, lymphosarcoma and Hodgkin's disease among 14 occupational groups considered to be exposed to benzene and/or coal-tar fractions in New York State. The exposed populations were estimated from census data, and deaths were obtained from health department records; mortality, adjusted for age, was presented separately for each cancer site and compared with rates for the state. Among 21 951 painters, the SMR for reticulum-cell sarcoma was 110 (based on nine cases), that for lymphosarcoma, 97 (15 cases) and that for Hodgkin's disease, 135 (21 cases).

Friedman (1986) carried out a case–control study of multiple myeloma among members of the Kaiser Foundation Health Plan in California, USA, and identified 327 cases during the period 1969–82. These were matched by sex, age, race, date of enrollment and residence with 327 controls on the rolls at the time of case diagnosis. Information on occupation was obtained from medical records. Painters as an occupational group occurred more frequently among cases (6) than controls (2). [The Working Group noted that it was not stated how frequently information on occupation was available.]

Morris *et al.* (1986) conducted a multicentre population-based case–control study in the USA of 698 newly diagnosed cases of multiple myeloma aged under 80 during 1977–81 and 1683 neighbourhood controls matched by age, sex and race. In personal interviews with subjects themselves or with next-of-kin, exposures were ascertained through a question about any exposure to toxic substances. A toxicologist grouped exposures into 20 categories, including 'paints, paint-related products and/or other organic solvents', which resulted in a RR adjusted for age, sex, race and study centre of 1.6 (51 cases exposed to paints and/or solvents; 95% CI, 1.1–2.4); of these cases, 40 had been exposed to paints and paint-related products. This risk showed little variation according to time since first exposure. When only cases who had been interviewed themselves were included, the adjusted RR for paints and/or solvents was 1.8 (39 cases; 95% CI, 1.2–2.7). [The Working Group noted that there may have been bias in the reporting of exposure.]

A case–control study of multiple myeloma in six areas of England and Wales was carried out by Cuzick and De Stavola (1988). A total of 399 cases identified at major regional centres between 1978 and 1984 and 399 age- and sex-matched hospital controls were interviewed about their past occupation and exposure to chemicals and radiation, as well as prior and family history of disease and immunizations. The risk of multiple myeloma in painters, including spray painters, was 1.9 (15 exposed cases; [95% CI, 0.76–4.7]).

Olsson and Brandt (1988) reported a case–control study of 167 male cases of non-Hodgkin's lymphoma aged 20–81 seen in the oncology department of the University Hospital of Lund, Sweden, in 1978–81. Exposure was assessed by interview by one of the authors using a standardized questionnaire, as in the study of Olsson and Brandt (1980; see p. 417). Two control groups comprising a total of 130 men who had been interviewed for two other case–control studies were used to estimate the exposure frequency. The RR for 'organic solvents' was 3.3 (63 exposed cases; 95% CI, 1.9–5.8). The risk for supradiaphragmatic lymphoma was higher (RR, 3.4; 95% CI, 2.3–5.2) than that for lymphomas localized below the diaphragm (RR, 1.4; 95% CI, 1.0–2.0). The risk increased with duration of solvent exposure. Occupational exposure to solvents was associated with employment in machine shops, chemical industry, painting, printing, wood industry and many other types of work; 14% of this population were painters.

(viii) *Cancer of the prostate*

In a cancer registry–based case–control study in Missouri, USA, conducted by Brownson *et al.* (1988), 1239 cases of histologically confirmed prostatic cancer in white males diagnosed between July 1984 and June 1986 were compared to 3717 white male cancer controls diagnosed in the same time period and frequency–matched by age. Information on occupation, collected routinely using a standardized protocol in all hospitals, was coded at the Registry as usual occupation and industry using the 1980 US census codes. When compared to workers in 'low–risk' industries (wholesale and retail trade, finance, insurance, real estate, business services and professional services), an elevated age–adjusted RR for prostatic cancer was apparent for men whose usual industry was coded as manufacturing of paints and varnishes (five cases; RR, 5.7; 95% CI, 1.4–24.3). However, in the analysis of usual occupation, no risk for exposure to paint was seen. The authors recognized several study limitations, including the use of crude occupational information, multiple comparisons, and use of cancer patients as controls.

(ix) *Cancer of the testis*

Swerdlow and Skeet (1988) identified 2250 cases of testicular cancer from the South Thames Cancer Registry, UK, for the period 1958–77. The proportion of painters and decorators among cases was compared with that among controls with cancers other than those of the genital system or at an unspecified site and among controls with cancers sampled such that no site represented more than 15% of the cancers in an age group. Occupation was identified from the records for 75% of cases and 73% of controls. The risk for testicular cancer among painters and decorators was about half that in the comparison group of professional, technical workers and artists (RR, 0.45; 15 cases), for both seminoma (RR, 0.44) and teratoma (RR, 0.55).

(x) *Cancer of the nasal cavity*

Hernberg *et al.* (1983) conducted a case–control study of nasal and sinonasal cancers among cases collected from the cancer registers in Finland and Sweden and from hospitals in Denmark in 1977–80. The 167 cases in live patients who agreed to interview were matched by age, sex and country to controls with colon and rectal cancer. Many of the patients in the subgroup with lesions in the maxillary sinus were not interviewed. Exposures were coded by

an industrial hygienist on the basis of intensity, duration and time. Smoking histories were evaluated for the period ten years prior to diagnosis, and smokers were found to be more frequent among cases (54.5%) than among controls (45.5%); the investigators indicated only that snuff use was not an important risk factor. Exposure to paints and lacquers reportedly showed a strong association with nasal cancer, but the investigators indicated that exposure to wood dust was generally a confounding factor. Two cases and no control had been exposed only to lacquers and paints, and both cases had had other potentially carcinogenic exposures.

(d) Cancer in children in relation to parental exposure

Fabia and Thuy (1974) analysed data on paternal occupation for 386 children aged less than five years who had died from malignant disease in the province of Québec, Canada, in 1965–70, and for the 772 control children whose birth registrations immediately preceded and followed those births. Father's occupation at the time of birth, as reported on the birth certificate, was recorded; no specific occupation was given for 30 cases or 56 controls. For ten cases and 11 controls, the paternal occupation at birth was described as painter, dyer and cleaner, excluding other hydrocarbon-related occupations (RR, 2.0; 95% CI, 0.86–4.7) [the Working Group calculated that the RR among those whose father was a painter was 1.2 (eight cases; 95% CI, 0.42–3.6)]. Among the 218 children with leukaemia, five of the fathers were in this occupational group; among the 101 children with central nervous system tumours and the 25 with Wilms' tumour, one (1%) and none (0), respectively, of the fathers were in this occupational group.

Hakulinen *et al.* (1976) carried out a case–control study of all 1409 children under 15 years of age with cancer reported to the Finnish Cancer Registry during the period 1959–68. After excluding twins and cases for which the father's occupation was unobtainable, 852 cases were available for analysis. The child born immediately before the case in the same maternity welfare district was chosen as a control. Father's occupation recorded at the time of conception was compared for cases and controls. Father's occupation described as 'painter, dyer, printer' was recorded for 12 cases and 15 controls; leukaemia and lymphomas occurred in one case and six controls, brain tumours in five cases and three controls and other tumours in six cases and six controls.

Kwa and Fine (1980) carried out a case–control study of 692 children born in Massachusetts, USA, who died before the age of 15 during 1947–57 and 1963–67. Controls were chosen from among the children whose birth registration immediately preceded and followed that of the case subject, giving a total of 1384 controls. Father's occupation at the time of birth registration was described as 'painter, cleaner, dyer' for 10 cases and 24 controls, comprising seven leukaemias or lymphomas, one neurological cancer and one urinary-tract cancer.

Zack *et al.* (1980) interviewed the parents of 296 children with cancer attending the Texas Children's Hospital Research Hematology Clinic in Houston, TX, USA, from March 1976 to December 1977. Controls were chosen from among relatives of cases, from among children in the neighbourhoods where the cases lived and from among children who did not have cancer attending the same clinic (33% had haemostatic defects, 24% various anaemias and 23% nonhaematological disorders). Job history from the year before the birth of the child

until one year before cancer diagnosis was assessed by personal or telephone interview of a parent. The fathers of none of the cases were described as 'painter, dyer or cleaner'; the corresponding figures for fathers of controls were one for relatives, two for neighbours and one for children attending the same clinic. [The Working Group noted that the selection criteria were given for neither cases nor controls and that it was unclear whether information on exposures was obtained from mothers, from fathers or from both.]

Hemminki *et al.* (1981) described the paternal occupations of 2320 children aged 0–14 with cancer reported to the Finnish Cancer Registry in 1959–75, many of whom had been included in the study of Hakulinen *et al.* (1976). Controls were chosen from among children whose birth had been registered immediately before and immediately after that of the index child. Parental occupation was taken as that in the maternity welfare clinic records at the time of pregnancy. The overall RR for a father's occupation as painter was 1.4 ([95% CI, 0.67–2.9]; based on 40 discordant pairs); the odds ratio for leukaemia was 1.5 ([0.22–10.3]; based on 12 discordant pairs) and that for brain tumours was 2.6 ([0.70–9.6]; based on 14 discordant pairs). The excess of brain tumours was most marked for the more recent study period, 1969–75, in which a significantly elevated RR of 5.0, based on seven discordant pairs, was reported. Maternal occupation was recorded for 2659 children, but no data on mother's exposure to paint was presented. The authors noted that for the earlier period (1959–68) only 63% of the cases had been included in the analysis; but for 1969–75, 86% of cases were included.

In a case–control study (Peters *et al.*, 1981), cases of brain tumours in children under ten years of age at diagnosis in 1972–77 were identified from the Los Angeles County Cancer Surveillance Program. Controls were matched to each case by sex, race and year of birth; matching for social class was attempted by trying to locate the control from among friends of the case or from the same neighbourhood. Mothers of 98 cases (84% of those available) and of 92 controls were interviewed by telephone, and the 92 matched pairs were analysed. Information included working and exposure histories of the mother and father before the pregnancy, during the three trimesters of pregnancy, during nursing and at the time of diagnosis. The authors noted the possibility of biased reporting and recording of exposures. Seven fathers of cases were reported to have had exposure to paints at any time from one year before conception up to the time of diagnosis; the father of one control had been similarly exposed. [The Working Group noted that this study addressed any exposure to paints and not only occupational exposures.]

Sanders *et al.* (1981) studied 6920 children under the age of 15 years who had died of malignant disease in England and Wales in 1959–63 and 1970–72. Father's occupation reported on the child's death certificate was compared with that recorded on the death certificate for a total of 167 646 childhood deaths that had occurred during the same periods. The PMRs for father's occupation described as 'painter or decorator' were 97 (based on 93 cases of cancer) in 1959–63 and 74 (based on 34 cases) in 1970–72. [The Working Group noted that data on specific cancer sites were not given.]

Associations between paternal occupation and childhood leukaemia and brain tumours were investigated in a case–control study in Maryland, USA (Gold *et al.*, 1982). Patients un-

der the age of 20 with leukaemia (1969–74) or brain tumours (1965–74) were ascertained in the Baltimore Standard Metropolitan Statistical Area from death certificates and records from 21 of 23 Baltimore hospitals. Two control groups consisted of children with no malignant disease, selected from birth certificates at the Maryland State Health Department, and of children with malignancies other than leukaemia or brain cancer. Information on occupational exposures of both parents before the birth of the child and between birth and diagnosis was collected by interviewing the mother. A total of 43 children had leukaemia and 70 had brain tumours. Paternal occupational category 'painter' was reported for one case of leukaemia, compared with three normal controls and none of the cancer controls, and no case of brain tumour, compared with one case in normal controls and none in cancer controls.

Wilkins and Sinks (1984) carried out a case–control study of 62 children with Wilms' tumour identified between 1950 and 1981 at the Columbus (Ohio) Children's Hospital Tumor Registry for whom paternal occupation was available from the child's birth certificate. Two groups of controls were chosen from birth certificates, the first matched individually for sex, race and year of birth, and the second for sex, race, year of birth and mother's county of residence at the time of the child's birth. Three of the fathers of cases were reported to be painters compared to one and none in the two sets of controls.

Van Steensel-Moll *et al.* (1985) carried out a case–control study of 713 children under 15 years of age with leukaemia diagnosed between January 1973 and January 1980 in the Netherlands. Controls were chosen from census records, matched by region, sex and age (to within two months). Information on occupational and other exposures of both parents during pregnancy was obtained by postal survey; the response rate was 88% for parents of cases and 66% for those of controls. The analysis was restricted to 519 patients with acute lymphocytic leukaemia and 507 controls. Twenty-five mothers of children with leukaemia and 11 mothers of controls reported having had occupational exposure to 'paint, petroleum products or other chemicals' during pregnancy (RR, 2.4; 95% CI, 1.2–4.6). These exposures were reported by 140 fathers of children with leukaemia and 113 fathers of controls (1.2; 0.8–1.7). The RR for paternal occupation described as 'painter, cleaner or dyer' was 1.6 (0.5–5.0) for exposures during pregnancy (eight cases) and 1.3 (0.4–4.0) for such exposures one year before the diagnosis of leukaemia (eight cases).

Lowengart *et al.* (1987) reported a case–control study of 123 children aged ten years or under with leukaemia identified in the Los Angeles County Surveillance Program in 1980–84, representing 57% of eligible cases. Controls were selected from among friends of cases or by random-digit dialling. Interviews were carried out by telephone and included questions on exposure to paints or pigments before, during and after pregnancy and on experiences the children had had from birth to the reference date. The specific types of exposure included in the general category 'paints or pigments' were spray paints, other paints, dyes or pigments, printing inks and lacquers or stains. Excess risks were observed for exposure of fathers to spray paints during the pregnancy (RR, 2.2; [95% CI, 0.91–5.3]) and after the pregnancy (2.0; 0.96–4.4) and for exposure to dyes and pigments during the pregnancy (3.0; [0.41–2.2]) and after the pregnancy (4.5; 0.93–42.8). The RR associated with 'spray paints' or 'dyes or pigments' was higher (RR, 2.5) if the father's exposure had been frequent ($\geq$50

times per year) than if it had been less frequent (< 50 times per year; RR, 1.8) after the birth of the child. Data on maternal occupational exposure were not presented. Use of paints or lacquers in the home by the mother and/or father during the pregnancy and lactation gave a RR of 1.4 (0.79–2.6). [The Working Group noted that the exposure categories overlapped.]

Johnson *et al.* (1987) analysed paternal occupational exposures recorded on the birth certificate of 499 children aged 0–14 who had died of an intracranial or spinal cord tumour in Texas in 1950–79. Children who had been born outside Texas were excluded. Controls were chosen from a 1% sample of live births in Texas during the same period. Maternal occupation could not be assessed. A RR of 1.0 (95% CI, 0.3–3.3) was reported for paternal occupation described as a painter.