OCCUPATIONAL DISEASES

A Guide to Their Recognition

Revised Edition

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Editors —

Marcus M. Key, M.D.
Austin F. Henschel, Ph.D.
Jack Butler, M.D.
Robert N. Ligo, M.D.
Irving R. Tabershaw, M.D.
Manuscript Editor —
Lorice Ede, J.D.

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
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Center for Disease Control
National Institute for Occupational Safety and Health

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DEPOSITION

There are four major factors that influence the site of the ultimate toxicologic response to inhaled particulates: 1) the anatomic arrangement and physical dimensions of the respiratory system, 2) the physiologic character of breathing rate and depth, 3) the physical nature of the particle-size, surface area, "solubility," and hygroscopicity, and 4) the biochemical reactivity of the soluble components of the particle.

The knotty problem of handling particles of all sizes, shapes, and densities has been resolved by relating all particles to a median aerodynamic diameter. This is the diameter of a unit-density sphere with the same settling velocity as the particle of concern. The cut-off point for respirable size is conventionally taken as 5 μm expressed as an aerodynamic diameter.

The aerodynamic diameter of particulates determines which particles will or will not present exposure to the respiratory system and gives some indication of the degree of impaction in the various compartments of the respiratory system, and hence the site of particle deposition. Thus, a particle such as uranium dioxide with a high density of 10.9 and diameter < 0.5 μm will behave as a unit-density spherical particle of 1.65 μm, and can be expected to settle out of undisturbed air at the rate of a larger diameter particle, and impact more in the upper respiratory passages than its measured size would indicate. The sedimentation rate of fibers depends on their diameter and is independent of length. Fiber geometry is also important in relation to certain toxicologic properties, for example, in the induction of mesotheliomas.

To simplify calculations of the deposition pattern of the aerosol of concern, the manifold compartments of the respiratory system are reduced to three: the nasopharyngeal, tracheobronchial, and pulmonary (2). If the particulates are assumed to be present as log-normal distributions and three tidal volumes are used, a table can be developed showing the amount of particles deposited in each of the three compartments according to unit-density sizes, ranging from 0.01 to 10 μm. As might be expected, no particles less than 0.6 μm were deposited in the nasopharynx at any of the three breathing rates, whereas practically all of the particles greater than 6 μm were deposited at this site at all breathing rates. Thus, the major site of deposition of the smaller particles is the lung; deposition in the tracheobronchial compartment never exceeded 25 to 30% of the total particles inspired even at the smallest sizes (around 0.01 μm) and the slowest breathing rate (3). Although various degrees of mouth-breathing would upset the calculations of deposition in the upper respiratory tract, it is not believed to affect seriously pulmonary deposition.

Hygroscopicity, however, seriously affects deposition of smaller, highly water-soluble particles by increasing their size as they travel down the respiratory tract in its 95% humidity. Thus, in some instances, it is possible for a small size particle in an atmosphere of low humidity to

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so increase its size in the respiratory tract as to alter considerably the deposition pattern characteristic of the entering particle size.

As is true with all generalizations, differences in deposition patterns can be expected, and indeed have been demonstrated, particularly for the lung where ventilation among the five lobes is normally variable.

Another factor which alters deposition patterns is electric charge, commonly associated with particle sizes less than 0.1 μm in diameter, i.e., newly generated fume. Such particles have enhanced nasopharyngeal deposition.

CLEARANCE AND RETENTION

In evaluating the health hazards from inhaled aerosols, four combined physio-chemical actions must be considered: 1) ciliary movement, 2) phagocytosis and lymphatic drainage, 3) direct intercellular penetration, and 4) solubilization or leaching.

Respiratory tract cilia do not extend beyond the terminal bronchioles. Particle clearance by upward ciliary movement takes place from the terminal bronchioles upward to the throat where the particles are transferred to the gastrointestinal tract by swallowing. Thus, exposure of the respiratory tract to particles may also involve exposure of the intestinal tract. With the exception of soluble particles impacting in the nasal passages and being absorbed there, clearance by solubilization from the tracheobronchial passages is not important.

Phagocytosis represents the major mechanism for clearing most particulates from the lung. Moreover, the presence of dusts stimulates the appearance of phagocytes at the site, so that repetitive exposures increase the rate of phagocytosis and hence the rate of clearance of dust from the lungs. Lymph drainage of the dust-filled phagocytes to the lymph nodes represents 2 to 10% of the clearance of the total pulmonary dust burden for certain insoluble oxide dusts.

Direct intercellular penetration offers another clearance mechanism of variable magnitude depending on the solubility, shape, and biologic activity of the dust. Thus, a particle that is not readily coated with serous protein, or other lung constituent, would penetrate the cell and then be cleared by this mechanism more readily than one that is coated.

Obviously solubilization represents the dominant clearance factor for particles readily soluble in respiratory tract fluids. Highly soluble dusts, such as the chromates of the alkali metals, pass through the lungs in a matter of minutes, and even grossly insoluble mineral dusts, such as certain types of asbestos, are subject to leaching of their metals and consequent clearance of these elements from the lung. A partial listing of inorganic compounds according to three pulmonary clearance classifications has been attempted for those compounds cleared in less than one day to 10 days, those requiring more than 10 days to 100 days for clearance, and those greater than 100 days, clearance time being expressed as biologic half-life (2).

following groupings comprise the majority of the occupational dermatoses:

(1) Acute contact eczematous dermatitis characterized by erythema, edema, papules, vesicles or bullae, crust, scale, and finally, desquamation. These are the signs of an inflammatory eczematous dermatitis caused by contact with a primary irritant or a sensitizer or a photosensitizer.

(2) Chronic eczematous dermatitis characterized by erythema, lichenification, scaling, dryness, and fissuring resulting from contact with substances which dehydrate the skin as alkali, liquids and dusts, solvents, soaps and detergents.

(3) Folliculitis and acneform dermatoses including chloracne characterized by plugged sebaceous follicles and nodular and suppurative lesions. Chloracne also shows multiple cystic lesions which contain straw-colored material. These dermatoses are caused by contact with insoluble oils, greases, tars, waxes, and certain chlorinated hydrocarbons as the chloronapthalenes.

(4) Neoplastic (benign and malignant) types as keratoses, papillomata, epitheliomas, and carcinomas of the exposed areas. These usually are caused by certain petroleum products, coal tar and certain derivatives, sunlight, and ionizing radiation.

(5) Pigmentary disturbances characterized by an increase or decrease of pigment in the epidermis. Increased pigmentation can result from contact with coal tar compounds, certain petroleum oils, vegetables, fruits, sunlight, and trauma. Decreased or absent pigmentation may result from burns; forceful trauma; chronic dermatitis; monobenzyl ether of hydroquinone; and certain phenolics as tertiary butyl catechol, tertiary amyl phenol, and tertiary butyl phenol.

(6) Granulomatous dermatoses characterized by chronic indolent focal inflammations which tend to heal with scar. These lesions can result from bacterial, viral, fungal or inanimate agents as asbestos, beryllium, and silica.

(7) Ulcerative lesions characterized by a loss of tissue on a cutaneous or mucous membrane surface leading to necrosis. Ulcerations can be caused by arsenic trioxide, calcium compounds, cement and concrete, chromic acid, burns and trauma. They may also result from purposeful or unconscious manipulation.

(8) Miscellaneous lesions. Some occupational dermatoses, because of their unusual nature, do not fit into the above classifications. Among such miscellaneous lesions are:

(a) alopecia induced by chloroprene;

(b) acro–osteolysis, with or without Raynaud's;

(c) sclerodermoid changes believed due to vinyl chloride polymerization;

(d) discolorations of the hair, skin, and nails due to various chemicals;

(e) porphyria cutanea tarda caused by a certain chlorinated hydrocarbon intermediate.

OCCUPATIONS AND AGENTS

The following is a list of occupations each accompanied by certain agents frequently associated with that occupation and capable of producing a dermatosis. Additional agents for the occupations listed as well as additional occupations will be found in other sections, principally the one on chemical hazards.

Abrasive Wheel Makers
- carborundum
- emery
- resin glues

Agricultural Workers
- See Farmers

Aircraft Workers
- adhesives (resins)
- alkalis
- bichromates
- chromates
- chromic acid
- cutting fluids
- cyanides
- epoxy resins
- flame retardants
- glass fibers
- hydraulic fluids
- hydrofluoric acid
- lubricants
- nitric acid
- oils
- paints
- plastics
- rubber
- solvents
- thinners
- ultraviolet light
- vibrating tools
- X-rays

Animal Handlers
- antibiotics
- bacteria
- cleaners & detergents
- deodorants
- feeds
- fungi
- germicides
- insecticides
- medicaments
- parasites
- pesticides
- viruses

Artists (Painters)
- acrylics
- epoxies
- paint removers
- pigments
- plasticizers
- solvents

Artists (Sculptors)
- dusts
- plaster of Paris
- pneumatic tools
- polishes

Athletes
- adhesives
- antibiotics
- bacteria
- lime
- medications
- protective gear
- soaps

Automobile Workers (Assembly)
- adhesives
- asbestos
- antifreeze
- brake fluids
- brake linings
- flame retardants
- gasoline
- hydraulic fluids
- oils
- rubber
- solvents

Automobile Work
- abrasives
- adhesives
- alkalis
- lead
- paints
- rubber compo
- solder
- solvents

Automobile Wor
- acids
- adhesives
- alkalis
- antifreeze
- brake fluids
- brake linings
- cleansers
- epoxy resins
- gasoline
- hydraulic flui
- lubricants
- rubber
- solvents
- thinners

Bakers
- benzoyl pero
- cinnamon
- dough
- dusts
- flavors (oils
- flour
- fungi
- heat
- moisture
- spices
- sugar

Barbers
- ammonium
- antiseptics
- bacteria
- cosmetics
- depilatories
- detergents
- dyes
- fungi

around respiratory bronchioles surrounded by a halo of dilated airspaces (10). In addition to the accumulations of coal in the macule, there is a slight increase in reticulin fibers and, to a lesser extent, collagen fibers. The presence of coal macules around the walls of the respiratory bronchioles may lead to atrophy or even to disappearance of the smooth muscle, this leading to a permanent dilatation of these small airways commonly called focal emphysema.

In about 1 to 2 percent of miners with simple dust accumulation, large, solid, black masses develop which represent accumulations of coal dust within macrophages and between reticulin and collagen fibers. These lesions are commonly formed in the upper lobes and differ from silicotic conglomerate masses in that the masses are not composed of discreet compressed nodules. The cause of the large lesions ("progressive massive fibrosis") in coal workers is not known. They are probably not due to coexisting tuberculous infection, but may represent an immunological reaction to the accumulated dust load. See Figure 5.

Caplan described the appearance of multiple rounded nodules in the lungs of coal miners with rheumatoid arthritis that subsequently proved to be necrobiotic nodules resembling those seen in rheumatoid arthritis (11). Microscopically, these lesions demonstrate a pale, necrotic center surrounded by granulomatous tissue having a typically "palisaded" appearance at the periphery of the nodule. Typical Caplan nodules have subsequently been reported in other occupations than coal mining, suggesting that they are not specifically related to coal dust exposure.

MIXED DUST PNEUMOCONIOSIS

In the mixed dust pneumoconioses the pathology depends to a large extent upon the relative proportion of free silica or quartz present in the airborne dust. Those with a quartz content of less than about 0.1 percent tend to develop small nodular areas in the lungs in almost direct proportion to the total amount of dust deposited, but little in the way of reticulin or collagen fibrosis, and very little emphysema. The pathological lesions more nearly resemble those found in coal miners.

On the other hand, dusts in which the quartz content ranges from about 2 percent to about 18 or 20 percent of the total dust tend to produce lesions that more nearly resemble those seen in classical silicosis.

Some examples of dusts that contain almost no free quartz are kaolin, talc, iron oxide associated with welding, coal, and coke used in making carbon electrodes.

DIFFUSE INTERSTITIAL FIBROSIS

There are a number of pneumoconioses that tend to produce diffuse interstitial fibrosis as their characteristic pathological lesion (6). Among these are berylliosis, aluminosis, Shaver's disease, and asbestosis. It appears likely that certain slowly dissolving constituents in the dusts give

CATEGORY

Profusion refers to the number of small opacities per unit area. Thus, the lung fields are divided into three zones on each side, and the number of opacities within each zone is graded. Standard radiographs are available for comparison which divide the profusion into categories 0, 1, 2, and 3. Category 0 refers to the absence of opacities or the presence of less profuse opacities than in category 1; category 1 shows small rounded opacities present, but few in number, and the normal lung markings are usually visible; category 2 shows numerous small rounded opacities, and the normal lung markings are still visible; category 3 shows very numerous small rounded opacities, and the normal lung markings are partly or totally obscured.

Actually, there is a continuum of changes from normality to the most advanced category and, to recognize this, the British National Coal Board developed a 12-point scale (13). This scale permits subdivisions of profusion into finer grades and is useful in epidemiological studies where progression of pneumoconiosis is important. The radiograph is classified into one of the four categories in the usual way by comparison with the standard midcategory films. If, during the process, the category above or below was considered as a serious alternative, this is also recorded. Thus, if a category ½ is recorded, it means that on comparison with standard radiographs the radiograph most nearly matched the category 1, but category 2 was seriously considered as an alternative.

The extent of pneumoconiosis is recorded by noting which of the lung zones are involved. Each lung is divided into three roughly equal zones by imaginary lines drawn at approximately one-third and two-thirds of the vertical distance between the apex of the lung and the dome of the diaphragm. Thus, each lung is divided into upper, middle, and lower zones for the purposes of recording the extent of pneumoconiosis.

IRREGULAR OPACITIES

Small irregular opacities are classified in much the same way as the small regular opacities, by type, profusion, and extent. Irregular opacities characteristically occur in asbestosis, but also occasionally in the other pneumoconioses. The variability, however, of these opacities in shape and width makes it virtually impossible to provide quantitative dimensions as is done in the rounded opacities; therefore, the types are divided on the basis of thickness. The s type refers to fine irregular, or linear, opacities; the t type refers to medium irregular opacities, and the u type refers to coarse (blotchy) irregular opacities. Standard radiographs of the three types of irregular opacities are available for comparison. Profusion of irregular opacities is graded in exactly the same way as is done in the rounded small opacities.

PLEURAL CHANGES

Certain pleural changes have recently become recognized as accompaniments to the parenchymal changes referred to above as part of

Table 3. Agent, pathology, and impairment associated with pneumoconioses.

Agent	Type of Pathology	Type of Respiratory Impairment
1. Silica		
Simple	Nodular fibrosis	Restrictive, diffusion
Complicated	Conglomerate nodular fibrosis	Restrictive, obstructive, diffusion
2. Hematite	Nodular fibrosis	Restrictive, diffusion
3. Mixed dusts		
Iron and silica	Nodular fibrosis (Rarely conglomerate nodular fibrosis)	Restrictive, diffusion
4. Silicates	Nodular fibrosis	Restrictive, obstructive
Talc	(Rarely conglomerate nodular fibrosis)	
Kaolin		
Bentonite		
Diatomite		
Tripoli		
Fuller's earth		
Mica		
Sillimanite		
Cement	Nonspecific bronchitis	Obstructive
5. Coal		
Simple	Peribronchiolar macules, focal emphysema	Obstructive (small airways)
Complicated	Conglomerate nodular fibrosis	Obstructive, restrictive, diffusion
6. Graphite	Peribronchiolar macules, focal emphysema	Obstructive (small airways)
7. Aluminum	Interstitial fibrosis	Restrictive, diffusion
8. Asbestos	Interstitial fibrosis	Restrictive, diffusion
9. Beryllium	Interstitial fibrosis (granulomata)	Restrictive, diffusion
10. Tungsten carbide	Interstitial fibrosis	Restrictive, diffusion
11. Barium	Simple dust accumulation	None known
12. Cerium	Simple dust accumulation	None known
13. Iron	Simple dust accumulation	None known
14. Tin	Simple dust accumulation	None known
15. Titanium	Simple dust accumulation	None known

The simple forms of classical silicosis, coal workers' pneumoconiosis, and the mixed dust pneumoconiosis, generally demonstrate mild obstructive impairment; whereas, the complicated forms (PMF) usually present mixtures of obstruction, restriction, and abnormalities of gas exchange.

RESTRICTIVE IMPAIRMENT

The restrictive pattern of physiological response is characterized by a reduction in lung volumes and ventilatory capacity, usually unaccompanied by an increased air flow resistance or hyperinflation. The restrictive pattern is also associated with an increased lung recoil, reduction in surface area for gas exchange and/or thickening of the air blood interface of the lungs.

The pneumoconioses that lead to diffuse interstitial fibrosis usually present the restrictive pattern of physiological impairment. In these, the earliest impairments are those involving gas exchange and diffusing capacity, and may be detectable only during exercise. In the later stages, gas exchange and diffusion abnormalities are detectable also at rest and are associated with a reduction in lung volumes, such as the vital capacity, total lung capacity, and the inspiratory reserve volume. Again, in cases where pneumoconiosis coexists with asthma or chronic bronchitis, this restrictive pattern may be associated with some element of obstructive impairment.

Asbestosis, berylliosis, aluminosis, and Shaver's disease are examples of pneumoconioses that are characteristically associated with the restrictive pattern of physiological impairment.

A summary of agent and type of pathology and respiratory impairment is given in Table 3.

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contains 50-100% benzene, the remainder consisting of toluene, xylene, and other constituents which distill below 120 C.

SYNONYMS

Benzol, phenyl hydride, coal naphtha, phene, benxole, cyclohexatriene.

POTENTIAL OCCUPATIONAL EXPOSURES

Benzene is used as a constituent in motor fuels, as a solvent for fats, inks, oils, paints, plastics, and rubber, in the extraction of oils from seeds and nuts, and in photogravure printing. It is also used as a chemical intermediate. By alkylation, chlorination, nitration, and sulfonation, chemicals such as styrene, phenols, and maleic anhydride are produced. Benzene is also used in the manufacture of detergents, explosives, pharmaceuticals, and dyestuffs.

A partial list of occupations in which exposure may occur includes:

- Adhesive makers
- Asbestos product impregnators
- Dry-battery makers
- Chemists
- Benzene hexachloride makers
- Burnishers
- Carbolic acid makers
- Chlorinated benzene workers
- Detergent makers
- Dye makers
- Furniture finishers
- Glue makers
- Linoleum makers
- Maleic acid makers
- Nitrobenzene makers
- Petrochemical workers
- Putty makers
- Rubber makers
- Styrene makers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal emergency standard for benzene effective May 21, 1977, is 1 ppm for an 8-hour TWA, with 5 ppm as a maximum peak above the acceptable ceiling for a maximum duration of 15 minutes.

ROUTES OF ENTRY

Inhalation of vapor which may be supplemented by percutaneous absorption although benzene is poorly absorbed through intact skin.

HARMFUL EFFECTS

Local—

Exposure to liquid and vapor may produce primary irritation to skin, eyes, and upper respiratory tract. If the liquid is aspirated into the lung, it may cause pulmonary edema and hemorrhage. Erythema, vesiculation, and dry, scaly dermatitis may also develop from defatting of the skin.

Systemic—

Acute exposure to benzene results in central nervous system depression. Headache, dizziness, nausea, convulsions, coma, and death

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SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Cement is used as a binding agent in mortar and concrete (a mixture of cement, gravel, and sand). Potentially hazardous exposure may occur during both the manufacture and use of cement.

A partial list of occupations in which exposure may occur includes:

Asbestos cement workers	Drain tile makers
Brick masons	Heat insulation makers
Bridge builders	Oil well builders
Building construction workers	Silo builders
Burial vault builders	Storage tank builders
Cement workers	Tunnel builders
Concrete workers	Water pipe makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for Portland cement is 50 mppcf.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

Exposure may produce cement dermatitis which is usually due to primary irritation from the alkaline, hygroscopic, and abrasive properties of cement. Chronic irritation of the eyes and nose may occur. In some cases, cement workers have developed an allergic sensitivity to constituents of cement such as hexavalent chromate. It is not unusual for cement dermatitis to be prolonged and to involve covered areas of the body.

Systemic—

No documented cases of pneumoconiosis or other systemic manifestations attributed to finished Portland cement exposure have been reported. Conflicting reports of pneumoconiosis from cement dust appear related to exposures that occurred in mining, quarrying, or crushing silica-containing raw materials.

MEDICAL SURVEILLANCE

Preemployment and periodic medical examinations should stress significant respiratory problems, chest X-ray, pulmonary function tests, smoking history, and allergic skin sensitivities, especially to chromates. The eyes should be examined.

SPECIAL TESTS

Patch test studies may be useful in dermatitis cases.

PERSONAL PROTECTIVE METHODS

In areas exceeding safe dust levels, masks with proper cartridges

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SODIUM H

DESCRIPTION

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SYNONYMS

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POTENTIAL

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long latent or lapse times following onset of exposure may yield erroneous conclusions as to the lack of health effects of those chemicals in the work environment that are being studied.

ANIMAL STUDIES

In epidemiologic studies, once a chemical or exposure condition has been shown to cause cancer, preventive measures may not be adequate to protect those who have had previous exposures, but who have not lived long enough for effects to be expressed in terms of clinical illness. A major advantage of experimental animal studies is the possibility of detecting a chemical cancer hazard earlier than if one waited for epidemiologic evidence of cancer in man to become available. Under such circumstances, preventive action can be taken much sooner.

To date, there are a number of instances in which data on cancer in experimental animals have been used to establish occupational health regulations in the United States. It is increasingly evident that experiments in animals can be important indicators of cancer risk for man. Almost all chemicals shown to be carcinogenic in man by epidemiologic studies have also been shown to be carcinogenic in appropriate animal models. Although this does not necessarily mean that a positive test for cancer in animals provides incontrovertible evidence of cancer risk for man, it does indicate that the chemical should be considered at least as a potential carcinogen for man.

Experts frequently recommend testing chemicals in more than one animal species, primarily to avoid false negative results. Nevertheless, this should not be interpreted to mean that, before a chemical can be called a carcinogen, it must be positive in two or more species tested. Naturally, however, the greater the number of studies that show that certain chemicals produce cancer in different species of laboratory animals, the greater the confidence in the conclusion that those substances pose a carcinogenic threat to man.

POTENTIAL OCCUPATIONAL EXPOSURES

The boundaries of potential occupational exposures to chemical carcinogens are ever expanding. The following occupations are some of those subject to recent investigations.

- Asbestos workers
- Auto repairmen
- Bakery workers
- Clothing pressers
- Coke oven workers
- Dairy industry workers
- Dental laboratory technicians
- Electricians
- Leather workers
- Photoengravers
- Roofers
- Rubber workers
- Vinyl chloride workers

Table 4 presents a list of occupational chemicals and substances which cause, or are suspected of causing, cancer and the target organ or tissue. It should be emphasized that this list is substantially incomplete in that many, if not most, chemicals in the workplace have not been adequately tested for their carcinogenic potential. As such, however, the format of Table 4 attempts to organize a growing body of data in a manner that may be useful for physicians in making a differential diagnosis of the possible occupational etiology of cancer cases in individuals. Additionally, this list may lead physicians and other health professionals to become more aware of the magnitude of the growing problem of chemical carcinogens in the workplace. Since occupational carcinogens may effect virtually all organ systems, physicians should be alert to investigate situations where clinically evident cancer could be associated with on-the-job chemical exposures.

One helpful data source for physicians is the registry of suspected carcinogens maintained by NIOSH as a subfile of the Registry of Toxic Effects of Chemical Substances (3). This Registry contains approximately 1,500 suspect carcinogens, most of which have not been adequately tested. Their inclusion on this list does not represent a process of substantive evaluation with respect to the adequacy of scientific data related to carcinogenicity. Rather, this list is a useful starting point to ascertain the extent of data regarding carcinogenic responses for a given compound. Even with these caveats, it should be evident that a number of compounds in this list may be shown to be carcinogenic in man following more detailed evaluation.

Observations by alert physicians and alert workers have frequently helped to identify problems of carcinogenic risk in the workplace long before they might otherwise be realized. Examples of this are hepatic angiosarcoma, a rare liver cancer caused by occupational exposure to vinyl chloride, and leukemia among workers in the manufacture of styrene-butadiene rubber. A number of other occupational chemicals have been shown to produce "marker" or unusual forms of cancer, such as the pleural and peritoneal mesotheliomas due to asbestos, and hepatic angiosarcoma due to inorganic arsenic. It is likely that careful follow-up of rare cancers or unusually high incidences of common cancers may help to uncover unsuspected chemical cancer hazards among other worker populations.

Unsuspected occupational cancer problems might also be predicted on the basis of structural similarity with certain chemicals and substances already shown to cause cancer in humans or animals. For example, Table 5 lists compounds that by virtue of their structural similarity to vinyl chloride would be suspected of posing possible carcinogenic risks to man. Surveillance by alert physicians of workers exposed to these substances might help to early identify potential future problems. Similarly, it is most important for clinicians to be aware of newer data on carcinogenesis emerging from experimental studies on animals. For example, preliminary data have indicated that trichloroethylene produces liver cancer in experimental animals (4), and data from Russia have suggested a human carcinogenic lung and skin response to chloroprene (5).

Table 4. Confirmed and suspected occupational carcinogens by target organ.

Target Organ/Tissue	Occupational Carcinogen	
	Confirmed	Suspected
Bone		Beryllium
Brain	Vinyl Chloride	
Gastroenteric Tract	Asbestos	
Hematopoietic Tissue (leukemia)	Benzene Styrene Butadiene and other Rubber Manufacture Substances	
Kidney	Coke Oven Emissions	Lead
Larynx	Asbestos, Chromium	
Liver	Vinyl Chloride	Aldrin Carbon Tetrachloride Chloroform DDT Dieldrin Heptachlor PCB's Trichloroethylene
Lung	Arsenic Asbestos Bis (chloromethyl) ether Chloromethyl methyl ether Chromates Coke Oven Emissions Mustard Gas Nickel Soots and Tars Uranium Vinyl Chloride	Beryllium Cadmium Chloroprene Lead
Lymphatic Tissue		Arsenic Benzene
Nasal Cavity	Chromium, Isopropyl Oil, Nickel, Wood Dusts	
Pancreas		Benzidine PCB's
Pleural Cavity	Asbestos	
Prostate		Cadmium
Scrotum	Soots and Tars	
Skin	Arsenic Coke Oven Emissions Cutting Oils Soots and Tars	Chloroprene
Urinary Bladder	4-Aminobiphenyl Benzidine B-Naphthylamine	Auramine 4-Nitrodiphenyl Magenta