OCCUPATIONAL SAFETY AND HEALTH SERIES
No. 67

OCCUPATIONAL LUNG DISEASES: PREVENTION AND CONTROL

INTERNATIONAL LABOUR OFFICE  GENEVA
PREFACE

This information package on the prevention of occupational lung diseases and lung cancer has been prepared in response to the concern about the health of millions of workers all over the world exposed in the course of their work to hazardous substances and materials. It aims to encourage and support governments and employers' and workers' organisations in implementing the relevant ILO standards.

It deals with occupational lung diseases (including bronchial cancer) corresponding to the list of occupational diseases (amended 1980), appended as the Annex to the Employment Injury Benefits Convention, 1964 (No. 121), as well as with acute respiratory diseases caused by irritant gases and vapours not included in the list. These diseases usually result from short-term exposure to high concentrations of these substances and are generally classified as occupational injuries.

The first three chapters summarise the basic principles of the prevention and control of occupational lung diseases, including lung cancer, in a comprehensive way and provide orientation to more specific information and action.

The fourth chapter describes in more detail individual occupational diseases, their causes, occupations bearing a particular risk, and specific preventive and control measures.

It is our aim that this publication may serve as educational material and a guide to those responsible for the working environment and the health of the workers at the enterprise level and to the representatives of employers' and workers' organisations dealing with occupational safety and health, as well as to administrators and professionals in the field of occupational safety and health.

The ILO wishes to express its thanks to Dr. A. David for the preparation of this guide.
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1. OCCUPATIONAL LUNG DISEASES: MAGNITUDE OF THE PROBLEM

1.1 Lungs as the target of air pollutants

Without breathing, there is no life: if breathing stops, we die. In order to live, we have to inspire the air surrounding us, regardless of its quality.

At rest, an adult person breathes about 14 times in a minute and inhales about half a litre of air by each breath. This makes seven litres of lung ventilation per minute, 420 litres per hour and about ten cubic metres in 24 hours. During physical work, the frequency and depth of the breaths increase many times above their rest values in order to supply the necessary amount of oxygen to the working muscles and remove the produced carbon dioxide. Some ten cubic metres of air inhaled over an eight-hour shift may be needed for an average physical workload.

If the air contains ten milligrammes of respirable dust particles per cubic metre - (a concentration of dust between approximately 1 and 10 \( \mu \)m is established in most countries as the exposure limit for dust without evident harmful properties) - the worker would inhale 100 milligrammes of dust per shift or some 20 grammes per year. Twenty grammes are a good spoonful of dust.

Luckily enough, the respiratory system has some efficient clearance mechanisms to rid itself of most of the inhaled dust. Nevertheless, in the lungs of deceased coal miners, around 10 grammes of dust have been found as the average.\(^1\)

Many types of air pollutants can cause damage to the lungs. As very few workplaces in either industry or agriculture are completely free free from air pollution by harmful gases, vapours, mist, fumes or dust, a great number of workers are exposed to them. It is no wonder that occupational lung diseases present a frequent work-related health problem.

It should be mentioned that the lungs are not the only target for many harmful agents; most inhaled toxic substances can pass through the lungs into the bloodstream without any damage to the lungs. However, when distributed by the blood circulation to various organs, they can damage them or cause general poisoning. In the work setting, absorption through the lungs is the most important way of entry for toxic chemicals, followed by absorption through the skin and ingestion. However,
1.2 Frequency of occupational lung diseases

The exact number of patients suffering from occupational lung diseases is unknown due to the imperfection of the notification systems for occupational diseases in many countries and the underreporting of these diseases. Some approximation can be made from a limited number of statistics published in some industrialised countries which base their reporting on the list of occupational diseases (amended 1980), appended to the ILO Employment Injury Benefits Convention, 1964 (No. 121). It can be estimated that the incidence of occupational diseases may be about one case per 1,000 workers annually as the average. Among all occupational diseases, some 10 to 30 per cent are occupational lung diseases. In countries with large mining activities, pneumoconioses (lung diseases caused by mineral dust such as silicosis or coalworker's pneumoconiosis) prevail, but other occupational lung diseases, especially allergic diseases caused by chemicals or dust of vegetable and animal origin are being reported in increasing numbers. The ILO has assessed that about 40,000 new cases of pneumoconioses may occur world-wide annually.

Many of the occupational lung diseases have a serious outcome. Up to 3 per cent of deaths of chronic respiratory diseases in New York State are reportedly associated with occupation.

Several reports at the VIIth International Pneumoconioses Conference in Pittsburgh, 1988, indicated the technical difficulties of full engineering control of airborne dust in mines. Reports from developing countries showed that the concentrations of airborne dust in mines are in general substantially higher than in industrialised countries. All these facts signal the importance of the subject and the need for continuing co-ordinated preventive action.

1.3 Action of the ILO

As occupational lung diseases belong among the most serious work-related diseases, they have been the subject of systematic attention by the ILO since the early thirties. In 1930, the first International Conference of Experts on Pneumoconioses was organised in Johannesburg and initiated two important, and still ongoing, activities. The experts agreed on the first international classification of pneumoconioses which later

The second activity arising from the first Conference has been the organisation, in co-operation with national authorities, of a series of International Pneumoconioses Conferences: in Geneva (Switzerland) 1938, Sydney (Australia) 1950, Bucarest (Romania) 1971, Caracas (Venezuela) 1978, Bochum (Federal Republic of Germany) 1983 and Pittsburgh (United States) 1988. The next Conference, to be held in Prague, Czechoslovakia, in 1992, is in preparation. In view of the increasing importance of other work-related respiratory diseases besides pneumoconioses, it was agreed to change the title of the next meeting to the VIIIth International Conference on Occupational Lung Diseases. It will be held in Prague, Czechoslovakia in 1992.

The continuation of these activities for 60 years indicates both the importance of occupational lung diseases and the difficulties in implementing efficient preventive and control measures at national and enterprise levels.

In 1934, the Workmen's Compensation (Occupational Diseases) Convention (Revised) (No. 42) listed, for the first time, silicosis and silico-tuberculosis among those occupational diseases for which compensation could be awarded. The list of occupational diseases covered by the Employment Injury Benefits Convention, 1964 (No. 121), included all pneumoconioses caused by sclerogenic mineral dust (silicosis, anthraco-silicosis, asbestosis) and berylliosis; bronchial cancer of miners in uranium mines was covered by the item "diseases caused by ionising radiations". Further important diseases were added by the 1980 amendment of the list: diseases caused by hard-metal dust, by cotton and other vegetable fibres (byssinosis), extrinsic allergic alveolitis (farmer's lung and similar) and lung cancer or mesotheliomas caused by asbestos. Infectious and parasitic diseases of the lungs contracted in an occupation where there is a particular risk of contamination are also covered by the relevant item of the list.

In the earlier period, attention was mainly centred on the medical aspects of the diseases and to the social security of sick workers. Only later was more consideration given to preventive and control measures. Earlier publications in the ILO Occupational Safety and Health Series advised on dust measurement and control and brought international reports on the prevention and suppression of dust in mining, tunnelling and quarrying. At the level of international standards, a breakthrough was later achieved by the Working Environment (Air Pollution, Noise and Vibration) Convention, 1977 (No. 148) and Recommendation (No. 156) which provide for measures to be taken for the control of air pollutants at the workplace. Priority is
also given to preventive action for the protection of the health of workers is given also in further key instruments, the Occupational Safety and Health Convention, 1981 (No. 155) and Recommendation (No. 164) and the Occupational Health Services Convention, 1985 (No. 161) and Recommendation (No. 171). Specific aspects of preventive and control measures in exposure to asbestos are dealt with by the Asbestos Convention, 1986 (No. 162) and Recommendation (No. 172).

The ILO has provided a great deal of guidance, in particular through codes of practice on occupational exposure to airborne substances harmful to health, safety and health in the iron and steel industry, safety in the use of asbestos and safety and health for coal mines. Chronic non-specific respiratory diseases related to work were discussed at the Tenth Joint/WHO Committee on occupational health. Recently, the publication on the prevention and control of occupational cancer was revised and guidance on safety in the use of mineral and synthetic fibres prepared. General advice can be found in the ILO Encyclopaedia of occupational health and safety and in the services provided by the International Occupational Safety and Health Information Centre (CIS).

Notes


2 Der Bundesminister für Arbeit und Sozialordnung: Bericht der Bundesregierung über den Stand der Unfallverhütung und das Unfallgeschehen in der Bundesrepublik Deutschland im Jahre 1986 (Bonn, 1987).


7 E. Zsögen: "Dinamika i sostojanie professional' noj zabolevaemosti v vengerskoj narodnoj respublike" [Development and status of occupational diseases in Hungary], in Gigiena truda i professional'nye zabolevanija, 1989, No. 8, pp. 4-6.


10 ILO: Course on dust prevention in industry, Occupational Safety and Health Series No. 8 (Geneva, 1967).

11 ILO: Dust sampling in mines, Occupational Safety and Health Series No. 9 (Geneva, 1967).

12 ILO: Sixth international report on the prevention and suppression of dust in mining, tunnelling and quarrying (1973-77), Occupational Safety and Health Series No. 48 (Geneva, 1982).


19 ILO: Safety in the use of mineral and synthetic fibres, Occupational Safety and Health Series No. 64 (Geneva, 1990).

2. AIR POLLUTANTS AT THE WORKPLACE AND THEIR EFFECTS ON THE RESPIRATORY SYSTEM

2.1 Air pollutants

Ideally, workplaces should be kept free from health hazards by appropriate technologies, work practices and engineering control measures. Regrettably, most production processes are associated with the risk of releasing some type of material which may become airborne. If this is the case, preventive and control measures should be applied in order to keep the exposure of the workers to these substances at or below a limit which is harmless to the workers' health. A failure to do so may result in serious health impairment.

Possible sources of air pollution are numerous and can be found in most workplaces. Some of the commonest production processes associated with certain respiratory hazards\(^1\) are summarised in table 1.

Table 1. Some production processes and associated respiratory hazards (from note 1, modified)

<table>
<thead>
<tr>
<th>Process</th>
<th>Respiratory hazard</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blending and mixing (solids or liquids are mixed to form products, undergo reaction, etc.) e.g. in:</td>
<td>Dust, mist:</td>
</tr>
<tr>
<td>- agriculture (production of dry animal fodder)</td>
<td>- organic (vegetable, proteins) dust</td>
</tr>
<tr>
<td>- chemical industry</td>
<td>- irritants, allergens</td>
</tr>
<tr>
<td>- construction industry (e.g. asbestos cement production)</td>
<td>- mineral dust (silica, cement, asbestos)</td>
</tr>
<tr>
<td>- food industry (flour mills, bakeries)</td>
<td>- flour dust</td>
</tr>
<tr>
<td>Crushing and sizing (mechanically reducing the particle size of solids and sorting larger from smaller with screens or cyclons)</td>
<td>Dust:</td>
</tr>
<tr>
<td></td>
<td>- mineral (silica, coal, asbestos, other minerals)</td>
</tr>
<tr>
<td></td>
<td>- organic (flour mills)</td>
</tr>
<tr>
<td>Process</td>
<td>Respiratory hazard</td>
</tr>
<tr>
<td>------------------------------------------------------------------------</td>
<td>--------------------------------------------------------</td>
</tr>
<tr>
<td>Handling and transport of solids in powder form</td>
<td>Dust of the material handled or transported</td>
</tr>
<tr>
<td>Machinery (metals, plastics or wood are worked or shaped with lathes,</td>
<td>Airborne particles, cutting oil mist</td>
</tr>
<tr>
<td>drills, planing or milling machines)</td>
<td></td>
</tr>
<tr>
<td>Mining (drilling, blasting, conveyor transport of the material)</td>
<td>Dust (silica, coal, asbestos, other minerals)</td>
</tr>
<tr>
<td>Surface treatment:</td>
<td></td>
</tr>
<tr>
<td>- abrasive blasting (surface treatment with high-velocity sand,</td>
<td>Dust (silica, metal)</td>
</tr>
<tr>
<td>steel shot, etc.)</td>
<td></td>
</tr>
<tr>
<td>- acid or alkali treatment (dipping metal parts in open baths to</td>
<td>Acid mist, oxides of nitrogen</td>
</tr>
<tr>
<td>remove oxides, grease, oil and dirt with hydrochloric, nitric or</td>
<td></td>
</tr>
<tr>
<td>sulphuric acid, etc.)</td>
<td></td>
</tr>
<tr>
<td>- degreasing (removing grease, oil, etc.) with chlorinated hydrocarbons (e.g. perchloro-ethylene)</td>
<td>Thermal decomposition of chlorinated solvents (even through cigarette smoking) may form phosgene, hydrogen chloride and chlorine</td>
</tr>
<tr>
<td>- electroplating (coating metals, plastics or rubber with thin layers of metals such as chromium, nickel, etc.)</td>
<td>Acid or alkali mists, chromium or nickel mists</td>
</tr>
<tr>
<td>- grinding, polishing and buffing (an abrasive is used to remove or shape metal or other material)</td>
<td>Dust (abrasives, metal)</td>
</tr>
<tr>
<td>Textile fibre processing (carding, spinning, weaving)</td>
<td>Dust (cotton, flax, hemp)</td>
</tr>
<tr>
<td>Welding and metal cutting (joining or cutting metals by heating them to molten or semi-molten state) - arc welding, flame cutting and welding</td>
<td>Metal fumes, oxides of nitrogen, flux particulate</td>
</tr>
</tbody>
</table>
When gases and vapours are used for technological purposes, they are kept in enclosed apparatuses or containers for obvious reasons. Nevertheless, they can occasionally escape through various leaks or following some accident. Many liquids evaporate when kept uncovered, in particular at elevated temperature. Further, gases, vapours or fumes can be released as by-products of accidental chemical reactions, or from burning of different materials. All these situations may produce dangerous concentrations of air pollutants.

Another important source of air pollution may be the dust already settled on the floor and other surfaces, and which secondarily becomes airborne.

2.2 Properties of air pollutants

Air pollutants can be found in the following forms:

- gas;
- vapour, i.e. gaseous forms of a substance which is a liquid at ambient temperature;
- mist, i.e. airborne liquid droplets;
- fume, i.e. product of condensation of evaporated material (e.g. oxides of iron in welding) and smoke, i.e. particles from fuel combustion;
- dust, i.e. particles formed by mechanical disintegration of solids.

Airborne particles or droplets are also called aerosols.

The shape of most airborne particles is irregular. Particles which have a length to width ratio greater than 3:1 are conventionally called fibres.

The hazardous potential of air pollutants depends first on their capacity of penetration and deposition in the respiratory system. Particle size is considered to be the most important characteristic. Mist droplets can have diameters from 0.5 to 20 um, fume particles usually have average diameters of less than 1 um down to 0.01 um. Dusts usually range from less than 1 um to 100 um. Typical sizes of a number of pollutants are given in figure 1.
Figure 1. Examples of sizes of some airborne particles

CONVENTIONS
- Range of size
- Small range, average
- Doubtful values

REFERENCE SIZES
- 400
- 200
- 65
- 35
- 10
- 325
- 100
- 48
- 28

Particle size (μm)

0.0001 0.0005 0.001 0.005 0.01 0.05 0.1 0.5 1 5 10 50 100 500 1000 5000 10000

Aerosols
Normal impurities in quiet outdoor air
Metallurgical dust and fumes
Smelter dust and fumes
Foundry dust
Flour mill dust
Sprayed zinc dust
Sulfide ore, pulps for flotation
Sulfuric acid mist
Cement dust
Pulverized coal
Insecticide dust
Plant spores
Pollen
Sneezes
Fly ash
Sand tailings
Washed foundry sand

Tobacco mosaic virus
Tobacco necrosis virus
Carbon black
Magnesium oxide fumes
Rosin smoke
Silver iodide
Combustion nuclei
Sea salt nuclei

Tobacco smoke
Oil smoke
Combustion nuclei

Diameter of gas molecules

Zinc oxide fumes

Diameter of gas molecules

Insecticide dust

Fog

Tobacco
Virus B protein

Fog

Smelter dust and fumes

Foundry dust

Sulfide ore, pulps for flotation

Sulfuric acid mist

Cement dust

Pulverized coal

Insecticide dust

Plant spores

Pollen

Sneezes

Fly ash

Sand tailings

Washed foundry sand

REFERENCES

Screen mesh

Visible to eye

Human hair diameter

Fog

Tobacco
Virus B protein

Fog

Smelter dust and fumes

Foundry dust

Sulfide ore, pulps for flotation

Sulfuric acid mist

Cement dust

Pulverized coal

Insecticide dust

Plant spores

Pollen

Sneezes

Fly ash

Sand tailings

Washed foundry sand

REFERENCES

Screen mesh

Visible to eye

Human hair diameter
2.3 Deposition of inhaled air pollutants in the respiratory tract

The inhaled air passes through the upper airways - the nose, pharynx and larynx - and the trachea or windpipe and the bronchi to the inmost parts of the lungs, the alveoli (figure 2). The trachea divides into two main bronchi which lead to the right and left lung. The main bronchi further divide like branches of a tree into smaller bronchi and bronchioles. Instead of leaves, the bronchial tree ends in sack-like structures, the alveoli. In figure 2, the peripheral lung structures, i.e. the bronchioli and alveoli are disproportionately enlarged in comparison with the larger bronchi, in order to enable the visualisation of their shape. In reality, the diameter of the trachea is about 16 to 18 mm, large bronchi are 7 to 12 mm in diameter, bronchioles 0.5 to 0.8 mm and the alveoli about 0.25 mm. However, as the diameter of the airways decreases progressively from the trachea outwards, the cross-sectional area of the airway lumen steadily increases. Consequently, the velocity of the inhaled air gradually decreases from the nose to the alveoli which conditions the deposition of aerosols in the lungs.

The total surface of the alveoli is enormous - some 60 or 70 m² - which is necessary to allow for the efficient transport of oxygen to the blood circulating through the lungs and of carbon dioxide from this blood into the exhaled air. Similarly, all airborne substances coming into contact with the lungs are likely to penetrate further.

When a molecule of a gas or vapour, or a droplet or particle of an aerosol comes into contact with the bronchial or alveolar wall, it can adhere to it and be deposited in the lungs.

The penetration and deposition of gases and vapours in the lungs depends mainly on their solubility in the layer of moisture which steadily covers all the surfaces of the respiratory system. Very water soluble gases, such as sulphur dioxide and ammonia, at concentrations considered as exposure limits, seldom proceed much farther down the respiratory tract than the main bronchi. These gases are so soluble that they are almost completely absorbed in the upper airways and trachea. On the contrary, less water soluble gases some of which may be highly irritant, such as nitrogen dioxide, phosgene and ozone, reach into the deeper recesses of the respiratory tract, affecting mainly the bronchioles and the adjacent alveolar spaces.

The factors governing the deposition of aerosols differ from those for gases and vapours. Particles are deposited in the respiratory system mainly according to their aerodynamic diameter, which is a function of their size, shape and mass. The concept of aerodynamic equivalent diameter permits the transformation of the physical properties of particles of any
shape and mass to those of a hypothetical sphere of unit density 1 g/ml. All further statements of particle size refer to the aerodynamic equivalent diameter.

Figure 2. Schematic diagram illustrating flow through the upper and lower airways

(Reproduced with permission from S.W. Clark and D. Pavia: Aerosols and the lung: Clinical and experimental aspects (London, Butterworth, 1984).)

Particles of a diameter above 50 μm (i.e. 0.05 mm) are rarely inhaled at all as the air velocity of inspiration is usually too slow for them to be sucked into the nose.

Particles with a diameter larger than 10 μm (0.01 mm) are usually deposited in the upper airways. Generally, for particles smaller than 10 μm, penetration will be increased with decreasing particle size. The upper size limit for penetration into the alveoli is considered to be approximately 10 μm, but most of the particles of this size will be eventually deposited in the bronchial tree. Maximum alveolar penetration and deposition occurs with particles of approximately 1 or 2 μm diameter. However, the deposition rate depends also on the lung
ventilation. As an example, the deposition corresponding to a currently encountered ventilation rate is given in figure 3.

**Figure 3. Deposition as a function of particle size for 15 respirations/min, 1,450 cm³ tidal volume**

The facts concerning penetration and deposition of air pollutants in the lungs have to be remembered when assessing potential hazards in workplaces, evaluating the working environment and the efficiency of control measures. It should be remembered that the dust particles which can be seen by the naked eye are not usually harmful to the lungs: the danger lies with the smaller ones which are invisible without a microscope.

### 2.4 Reaction of the respiratory system to air pollutants

That part of inhaled matter which is not retained or deposited in the lungs is exhaled. Dissolved gases and vapours react with the bronchial or lung tissue, or pass through the alveolar wall into the bloodstream and are distributed in the body.

The deposited particles are subject to the clearance mechanisms of the lungs which are able to eliminate most of the particles. The trachea and bronchi are covered by a mucous layer which has a continuous upward movement to the nasopharynx (mucociliary movement). Particles deposited in the bronchi are moved there, and then expectorated or swallowed with the mucus.

The alveoli have no similar mucociliary clearance. Insoluble particles deposited in this area are engulfed by large phagocytic cells (macrophages). After engulfing the foreign particle, the macrophage can move to the bronchioli and then be transported by the movement of the mucus upwards and out of the respiratory system. Other macrophages may re-enter the pulmonary
tissue and remain there, or they may move to the lymphatic system. Certain dust, such as silica or asbestos, damages and kills the macrophages and may be found free in the lung tissue.

The respiratory system reacts by a limited number of responses to the presence of air pollutants.

2.4.1 Irritative lung injuries

A number of substances are irritants to the airways causing a burning sensation in the nose and throat (and usually also in the eyes), pain on the chest and coughing, and producing inflammation of the mucosa (tracheitis, bronchitis). Examples of irritants are gases such as chlorine, fluorine, sulphur dioxide, phosgene, oxides of nitrogen, mists of acids or alkali, fumes of cadmium, dust of zinc chloride, vanadium pentoxide.

High concentrations of chemical irritants may penetrate deep into the lungs and cause lung oedema (the alveoli are filled with liquid) or inflammation (chemical pneumonitis).

For further details, see section 4.8.

Dust which has no chemical irritative properties can also mechanically irritate bronchi, however only in substantially elevated concentrations (see also section 2.5).

2.4.2 Hypersensitivity reactions

Some substances can produce damage to the respiratory system due to hypersensitivity reactions which are the result of repeated contact with the agent. No health impairment is obvious after initial exposure. There are two main types of these reactions: bronchial asthma and extrinsic allergic alveolitis (see sections 4.4 and 4.5). Byssinosis (see section 4.3) and berylliosis (see section 4.6) also share several common features.

2.4.3 Pneumoconioses

Pneumoconiosis is defined as the accumulation of dust in the lungs and the reaction of the tissue to its presence. Cancer caused by the accumulated dust is not denoted as pneumoconiosis.

Some types of dust may simply accumulate in the lung tissue, without further significant pathological alterations of lung morphology or function. Examples are the accumulation of tin oxide (stannosis) or barium sulphate (baritosis) (see section 4.10.1).
Fibrogenic (sclerogenic) minerals cause proliferation of connective tissue in the lungs (scarring of the lungs). Examples are silicosis, asbestosis and coalworker's pneumoconiosis (see sections 4.1 and 4.2).

2.4.4 Lung infections

A number of infectious agents can cause lung inflammation: tuberculosis, anthrax, ornithosis, brucellosis, histoplasmosis, Legionnaires' disease, etc. (see section 4.9).

2.4.5 Cancer

Examples of airborne particles that can produce cancer of the lungs are arsenic and its compounds, chromates, particles containing polycyclic aromatic hydrocarbons and certain nickel-bearing dusts (see section 4.7). Asbestos fibres can cause bronchial cancer and mesothelioma of the pleura and peritoneum (see section 4.1.4). Deposited radioactive particles may expose lung tissue to high local doses of ionising radiation and be the cause of cancer.

2.5 Effects of environmental pollutants (inert dust, tobacco smoke)

Particles of inert dust and tobacco smoke are almost ubiquitously present in the air of workplaces. Therefore, their effect on the respiratory morbidity is frequently discussed.

Inert dust is formed of material which does not cause any specific respiratory pathology. It has been shown that high concentrations of such dust can cause acute bronchial catarrh and bronchoconstriction by mechanical irritation. However, the important question is, whether long-term dust exposure can cause disabling and irreversible bronchopulmonary disease. Some specialists believe in this association, and a few countries have even included the so-called "industrial bronchitis" in their lists of occupational diseases which could lead to compensation entitlement. However, more generally it is thought that although there is no doubt that chronic simple bronchitis (i.e. persistent phlegm production without impairment of lung functions) is caused by the inhalation of dusts, there is, at present, no convincing evidence to show that chronic obstructive bronchitis (i.e. persistent phlegm production with lung ventilation impairment) nor associated respiratory disability are directly and consistently attributable to such exposure. The principal cause of obstructive bronchitis appears to be smoking. Whereas some 6 per cent of non-smoking adult men have chronic coughs and phlegm, there is a twofold increase of chronic bronchitis in
ex-smokers, a fourfold increase in those smoking 20 cigarettes or less per day, and a sixfold increase in heavy smokers (more than 20 cigarettes daily). It is an important task of health promotion to discourage people from tobacco smoking.

The causal association between cigarette smoking and bronchogenic cancer has been firmly established since 1955. The synergistic effect of cigarette smoking on the incidence rate of lung cancer in asbestos exposure has been mentioned in section 4.1.4. Lately, the risk associated with passive smoking, i.e. sharing the same workplace with smokers and breathing the air polluted by tobacco smoke, has also come under discussion.

Whereas there is no evidence of causality relation between passive smoking and chronic bronchitis, there is some indication of its association with the risk of lung cancer. Although the causality of this relationship is at present a matter of controversy, the possibility should be given serious consideration and further research.

Notes


3. APPROACHES TO THE PREVENTION AND CONTROL
OF OCCUPATIONAL LUNG DISEASES

3.1 Harmful substances at work: Controlled use

The problems of working with hazardous substances are infinitely complex. The advantages and disadvantages of a substance for society spring from the same inherent properties.

The use of agro-chemicals has helped to increase crops and eliminate famine in many countries of the world. On the other hand, there is growing public concern about the negative consequences for agricultural workers and the environment of the indiscriminate use of chemicals in agriculture.

It has been predicted that world demand for coal will rise from today's 3,000 million tons to over 5,000 million tons by the year 2000 and the number of people exposed to risks in coal mines will increase from 8 to over 10 million.

At the International Labour Conference in 1986, when discussing safety in the use of asbestos, a representative of a developing country stated that many countries have a growing need for asbestos products and that these products will play a part in improving the living conditions of many communities around the world. This is a classical example of a harmful substance providing social benefits through improvements in housing, clean water supply and sanitation. For centuries the scourge of silicosis has accompanied mining and is still present despite technological and medical progress. The demand for more coal and minerals in an energy and consumer-products hungry world has led to an acceptance of risk for those who mine coal and minerals for the benefit of society as a whole. At the same time, the parameters of concern for safety and health are increasing.

There are two principal solutions to the dilemma in which society finds itself when dealing with harmful substances. The first is to abandon the use of the harmful substance. The second is to use it in such a way that the potential hazards are minimised.

The use of harmful substances may be abandoned voluntarily. Very often society, or groups within society, consider compulsory banning of the use of a substance as the only acceptable solution and demand governmental intervention.
In practice the abandonment of the use of a harmful substance is only possible if a satisfactory substitute becomes available. A substitute must possess the same or similar technical properties and must present no hazard or at least less hazard than the original substance. Here lies the main difficulty of substitution from the health and safety point of view. As the substitute is usually a new product, there is no experience with its potential to cause some delayed harmful effects. The hazard to the health of future generations and the risk of cancer are the most frequent concern. Pre-marketing laboratory testing on animals is able to reveal most of the harmful properties, but there may be a long latency period before a disease caused by a harmful substance becomes apparent in humans.

When the replacement of a substance is not possible, principles of the controlled use are to be applied.

The first principle of managing the problem of harmful substances is that those who are exposed to the substance must be aware of its dangerous potential. Knowledge is the key to all future action and those who may have to bear the consequences of exposure to harmful substances have a right to be kept informed. Only unrestricted access to all kinds of information essential to the safe use of harmful substances will create confidence and the willingness to comply with restrictions on working which may be cumbersome and uncomfortable.

The second principle of managing the problem of harmful substances is to apply effective engineering and process controls. This includes the right choice of technologies, proper design and maintenance of equipment and machinery, the application of engineering control measures, the monitoring of the working environment, good housekeeping and personal protective equipment and clothing.

The third principle of the management of the problem of harmful substances is the education and training of those exposed to risk, whether they be management or workers. The elements of a proper education and training programme must begin with the manufacturer or supplier of the substance who have the duty to establish the harmful potential of their products. They have also a natural interest that a harmful substance is used in such a way as not to give rise to public concern and discussion. Normally the user of a product would face difficulties in preparing an adequate training programme without assistance. Training modules on harmful substances or products containing harmful substances should originate with the manufacturers or suppliers.

The previous chapters provided elementary information concerning occupational hazards to the respiratory system.
following text deals with basic principles of prevention and control in the light of the provisions of relevant Conventions and Recommendations and other guidance provided by the ILO.

3.2 Hazard evaluation and exposure limits

The first step in the control of any hazard is its identification. The work process should be carefully investigated in order to detect in advance the potential health hazards. A useful scheme to this end is suggested in figure 4. Regrettably, there are still workplaces where the existence of an occupational hazard is not known from the examination of the work, but becomes evident only from the appearance of some work-related disease among the exposed workers.

Once the presence of a harmful agent has been recognised, the degree of danger to health shall be evaluated. Obvious danger resulting from apparent neglect of safety and hygiene measures can be revealed by simple observation, but more subtle danger may need to be detected by instrumental measurement. This is also necessary when comparison with officially established exposure limits is required according to the Working Environment (Air Pollution, Noise and Vibration) Convention, 1977 (No. 148). Article 8 of this Convention provides that the competent authority shall establish criteria for determining the hazards of exposure to air pollution in the working environment and specify exposure limits on the basis of these criteria. The type of preventive and control measures (see section 3.3) will largely depend on the level of workers' exposure in relation to the exposure limit.

The term exposure limit has been used to embrace the various expressions referring to standards of the quality of the working environment, such as maximum allowable concentration, permissible concentration, threshold limit value, etc.

The concept of exposure limits is based on the fact that for the majority of health effects there is a correlation between the amount of the harmful agent to which the worker is exposed and the resulting health impairment. In general, the higher the exposure (e.g. concentration of a pollutant in the air and the duration of employment) the more liable it is to cause some adverse health effect. A knowledge of this relationship makes it possible to evaluate the risk of such impairment. More specifically, it can be used for the establishment of a safe limit below which there should be no health hazard to the average worker. However, there are theoretical considerations that this may not be fully accurate as regards carcinogenic agents.
Preliminary Evaluation of Hazard

Questions
What is material?
What is process?
What are intermediate products?
What are final products?
What are wastes?

Action
Trace related published literature.
Trace direct or related industrial experience.

Does this suggest problem?
No
Yes

Is the factor
Chemical?
Physical?
Biological?

Follow similar route to chemical

Is the hazard
Immediately dangerous to life or limb?
Dangerous over a long period?
Not dangerous but having ill-effects?
Only undesirable physiologically or psychologically?

Can process be allowed to continue in present form for short period?
Yes
No

Can effective temporary control be applied immediately?
Yes
No

Stop Process
Stage 5
Apply Control
Stage 2
Appraisal

First consider economic & social aspects
Consider long-term strategy
Is systemic evaluation needed?
No
Yes
Exposure limits to airborne harmful substances at the workplace are concentrations in the air of harmful substances which do not, it is believed in the light of present scientific knowledge, cause adverse health effects — including long-term effects and effects on future generations — in workers exposed for eight to ten hours per day and 40 hours per week. However, concentrations below the exposure limit may not necessarily completely guarantee protection of the health of all workers. Therefore, the exposure limit does not constitute an absolute dividing line between harmless and harmful concentrations but shall be regarded as a guide for the prevention of the hazard. In other words, one should not be fully satisfied if the concentrations of air pollutants at the workplace are at the level of exposure limit but it should be attempted to keep the exposure of the workers below this limit. In some countries, the competent authorities prescribe the so-called action levels corresponding to about one-half to one-third of the exposure limit approved by the national legislation. No specific preventive action is required at such exposure.

The most extensive lists of exposure limits for air pollutants have been issued in the USSR, United States and the Federal Republic of Germany. As the establishment of an exposure limit requires a rather complex procedure, many countries tend to take over the limits already established elsewhere. However, it should always be remembered that the industrial, climatic, social and ethnic conditions in the country where an exposure limit originated may largely differ from those in another country and that therefore the exposure limit should not be used elsewhere without modification. This is particularly true in view of the duration of the working hours. If an exposure limit has been settled for a 40-hour working week, it has to be adapted to other work schedules.

The exposure limits for air pollutants are expressed in principle in mass concentration, i.e. in mg per m³. As regards dust, the exposure limit is set either for the total amount of dust, or its respirable fraction (mostly particles of a diameter below 10 μm), or both. As for mineral fibres such as asbestos or man-made mineral fibres, the exposure limit is expressed in most countries as the number of respirable fibres in ml air, although some countries still use the mass concentration (mg/m³), as with other types of dust. (Note: The respirable fibre is defined by the Asbestos Convention, 1986 (No. 162), as a fibre having a diameter of less than 3 μm and a length-to-diameter ratio greater than 3:1. Only fibres of a length greater than 5 μm shall be taken into account for purposes of measurement.)

The most general trend today is to relate the exposure limits of air pollutants to a time-weighted average.
concentration, usually the mean concentration over the working day, sampled in the breathing zone of the worker. Thus, the excursions of the actual concentration above the limit must be compensated during the shift by equivalent excursions below the limit.

For many substances, such as fast-acting irritant gases and vapours, it would be unsafe to allow unlimited peak excursions above the time-weighted average. For this reason, ceiling or short-term exposure limits have been established for such substances. Their duration may be limited according to their effects and the possibility of the measurement of their concentration in the air by available analytical methods, but usually it is in the range of from five to 15 minutes.

3.3 Principles of preventive and control measures

The objective of preventive and control measures is to eliminate or reduce the exposure of the workers to airborne harmful substances or agents to such a level that adverse health effects are prevented.

The potential of exposure can be eliminated by the abandoning of the use of a hazardous substance, or if this is not possible, by its substitution by another substance not hazardous to health. The tools, machines, plants, work processes and practices should be designed in such a way that the formation of gas, vapour or aerosol be prevented or suppressed.

Where elimination, substitution, prevention or suppression are not feasible, hazardous substances should be contained in closed systems.

Where total enclosure is not possible, effective local exhaust ventilation complemented by a good standard of general ventilation with supply of fresh air to the breathing zone of the workers shall be operated.

If the above measures are not in a position to restrict the exposure of the workers at or below the established exposure limits, appropriate protective equipment such as protective clothes and respirators should be provided and used.

Control measures shall also aim at restricting the quantity of the hazardous substance used, limiting the area in which it is used, the number of people exposed, and the duration of exposure.

Measures should be taken to secure the cleanliness of workplaces to avoid settled dust becoming airborne, and to avoid the spread of contamination throughout the enterprise as well as
outside, in particular in the workers' homes. Adequate washing facilities and changing rooms for work clothes should be made available to this end.\textsuperscript{5}

3.3.1 Elimination of the hazard

The most efficient means of elimination is the discontinuation of the use of a substance or of a work process which may expose the workers to a particularly high health risk. Thus, Convention No. 162 provides for the banning of the use of crocidolite in view of the risk of mesothelioma connected with the exposure to this type of asbestos, and for the prohibition of spraying of all types of asbestos, because it is extremely difficult to keep airborne asbestos fibre concentrations fully under control and at or below the established exposure limits during this process.

The hazard can also be eliminated by the replacement of the noxious material by a non-hazardous one. As mentioned in section 3.1, there are many obstacles to overcome in using substitute materials. Available substitute materials must satisfy technological and economical requirements, and they shall not pose a new or unrecognised health risk. Careful preliminary investigation is needed and the exposure of the workers and their health status systematically monitored for a sufficiently long period to ensure the absence of any adverse health effects, under current working conditions. Nevertheless, a number of examples of successful prevention of occupational lung diseases by the use of substitute materials can be given:\textsuperscript{6}

- use of synthetic grinding wheels (e.g. silicon carbide) instead of sandstone wheels;
- steel shot instead of quartz sand for abrasive blasting;
- non-silica moulding aggregates instead of quartz sand in foundries;
- substitute materials for asbestos;
- magnesite bricks instead of silica-based refractory bricks for furnaces;
- removal of beryllium phosphors from fluorescent tubes;
- replacement of arsenicals by arsenic-free pesticides.
3.3.2 Prevention of the formation of air pollutants

Gases and vapours are kept in enclosed systems for obvious technological reasons. Appropriate work practice and maintenance should prevent their uncontrolled release in the working environment; however, accidents due to technical failure or human error cannot be excluded.

Occasionally, the workers need to enter the containers, e.g. during maintenance work. It should always be checked that the containers have been emptied and well ventilated and do not contain hazardous gases (lung irritants etc.) in dangerous concentrations. The same caution is necessary before entering forage silos in which oxides of nitrogen may have accumulated and cause the so-called silo-filler's disease (see section 4.8).

To prevent the formation of chromium- or nickel-containing mists in electroplating, the surface of the galvanic baths can be covered by means of plastic floating bodies or artificial foam.

As most of the occupational lung diseases are dust-related, major interest lies in methods for dust supression.

Wet control methods are the most widespread. Water is applied at the point of dust generation so that dust particles become wetted and attach together. In mines, the rock (e.g. the coal-face) is either previously infiltrated with water, or water is applied during the drilling of holes. Water spraying is frequently used at such places where dust may become airborne (e.g. transport of mined rocks by conveyor belts). Wet abrasive blasting is another good example. Water should be used for cleaning dusty workplaces when vacuum cleaning is not feasible.

Dust formation can be largely diminished by the use of specially designed tools, particularly in work with asbestos cement products.

Extrinsic allergic alveolitis (see section 4.5) is mostly caused by saprophytic fungi (moulds) and can be in many instances prevented by appropriate technologies which avoid the vegetable material becoming mouldy. Where the fermentation is a component of the technology such as in cheese production, work practices should be used which reduce the generation of aerosols containing yeasts or moulds.

3.3.3 Isolation of the source of air pollution

There are many processes which by nature require a closed system. Whenever this is not the case but there is a choice, closed systems should be preferred.
If the evolution of the aerosol cannot be prevented, then segregation or enclosure of the process should be considered. Segregation of the dusty procedures in one area facilitates the installation of any necessary control system. Complete enclosure is the best form of segregation. An example of this is the blasting cabinet where the operator controls the abrasive blasting from outside the enclosure. In the removal of friable asbestos from the buildings, enclosure around the worksite is necessary in order to prevent the dispersion of asbestos-containing dust in the environment (figure 5). However, this target can be achieved only if the enclosure is kept under negative pressure. As a matter of fact, enclosures are fully reliable only if they are operated in combination with local exhaust ventilation systems.

![Figure 5. Complete enclosure of the worksite during removal of friable asbestos](image)

It should be remembered that whereas the ventilated enclosure protects the workers who are outside the enclosure, the persons working inside may be exposed to high concentrations of the harmful agents and must be protected by the use of respirators or other types of personal protective equipment (e.g. during the removal of friable asbestos insulation). 9

3.3.4 Ventilation 6,10,11

Natural ventilation may be efficient enough to take the contaminated air away from the breathing zone of the workers and replace it by fresh air if the source of air pollution is not too excessive and the work is carried out in the open air, or in rooms with large openings. The latter situation is very frequent in countries with hot climates where the natural ventilation of buildings is used to keep inside temperatures within a tolerable range. Nevertheless, the amount of natural ventilation is...
difficult to predict because of its variations and it is rarely possible to fully rely on it. It should also be kept in mind that the air pollutant is emitted into the general environment in an uncontrolled way.

With high emissions of air contaminants, it is necessary to arrange either for local exhaust ventilation or for a fresh air supply to the breathing zone of the workers. The latter can be achieved e.g. by air-fed helmets. Local exhaust ventilation is the method of choice in most instances.

Local exhaust ventilation placed close enough to the source of air pollution can remove the contaminant before it reaches the breathing zone of the worker. The principle of local exhaust ventilation\textsuperscript{10} is shown in figure 6. In combination with full or partial enclosure (hoods), it is the most efficient engineering control measure for the removal of the contaminated air (figure 6).\textsuperscript{10} Local exhaust ventilation can be installed either at fixed sites near the machines\textsuperscript{13} generating air pollutants (figure 8), or as a portable device when the worker has to move around during his work, e.g. in welding or grinding large pieces (figure 7). However, the former arrangement is to be preferred as it provides protection independently of the performance of the worker.

Figure 6. Principles of local exhaust ventilation\textsuperscript{10}
Figure 7. Dust control by local exhaust ventilation.
As local exhaust ventilation removes quite considerable amounts of air from workplaces, an adequate amount of fresh, non-contaminated air has to be supplied. This is best achieved by the combination of local exhaust ventilation with forced (powered) general ventilation and distribution of the supplied air into the breathing zone of the workers. At low external temperature the air may need to be preheated. The air extracted from the workplace must be cleaned (filtered etc.) before it is ejected, in order to remove the pollutant and prevent it from contaminating the environment (figure 9). \[11\]

The design and construction of the ventilation system requires special knowledge and has to be carried out by specialists.
It should be stressed that the efficiency of the ventilation depends on the perfect state of the equipment. Regular cleaning and maintenance play a principal role.

**Figure 9. Schematic diagram of a basic ventilation system**
3.3.5 Personal protective equipment

To do their work in safety, many workers need some form of protective equipment. The purpose of the protective equipment is to create a barrier between the workers and hazards in the workplace. In the case of airborne hazards, the respiratory protective equipment, eye protection and protective clothing should be used, as appropriate.

It should be remembered that even the best designed equipment is only the last line of defence and will not eliminate hazards; controlling them at their source must always be the method of choice.

To be effective, protective equipment must be of the right type for the job and hazard, it must fit properly, be used in the correct way, and be regularly maintained. It should create as little discomfort as possible (although some discomfort is unavoidable, particularly in the use of respiratory equipment), and no safety problems: for example, masks can cause sight difficulties.

The Working Environment (Air Pollution, Noise and Vibration) Convention, 1977 (No. 148), states that when technical or organisational measures do not bring air pollution in the working environment within the exposure limits, the employer shall provide and maintain suitable protective equipment.

(i) Respiratory protective equipment

The use of respiratory equipment causes discomfort and full protection is secured only by the selection of the appropriate type and its flawless use. Because of these problems, respirators should only be used as a temporary aid, and not be considered a permanent remedy.

(a) Respirators

Respirators purify the air by filtering out harmful gases, dust and other air pollutants. Filters designed for dusts will not protect against gases and vice versa, so it is very important to make sure that the right filter is used.

Respirators can have full facepieces or half-masks that cover nose and mouth only (figure 10). Full facepieces providing eye protection may be needed in exposure to respiratory irritants (figure 11). If all the inspired air is to pass through the filter, the mask must fit the face tightly; even a small beard can prevent a perfect fit.
Aerosol filters provide protection against dusts, mists, mists, fumes and particles. They cannot stop gases and vapours. The purifying activity is accomplished mainly by mechanical filtration. The filter must trap the size of particles which are responsible for the effect (see section 2.2). Although none of the filters will retain 100 per cent of the aerosol – if they did, not much air would get through – the filtration efficiency of approved commercial filters is well over 95 per cent for most industrial dusts. The most important concern is leakage between the facepiece and the face when the respirator has not been well matched to the individual facial configuration.

As the filter becomes loaded with dust the breathing resistance increases, consequently the filter has to be changed at intervals depending on the dust concentration in the air.

Figure 10. An aerosol filter respirator providing protection against dusts, mists, fumes and toxic particles.
Gases and vapours can be filtered by chemical cartridge or canister respirators which react by absorption, chemical reaction or catalysis. They will not trap dust particles unless a special filter has been added. These respirators do not provide protection against contaminant concentrations usually exceeding 1 or 2 per cent by volume, and protect during a limited time period only. The filters are designed to give protection against single substances (e.g. ammonia), groups of substances (e.g. acid gases), or certain combinations of them (figure 12). It is essential that filters are properly selected in accordance with the envisaged exposure. In these circumstances, they can provide effective protection for routine and emergency applications.
Figure 12. A universal gas mask canister

A universal gas mask canister can protect the hopocite from moisture. Anhydrous calcium chloride can protect the hopocite from moisture. A filter pod for removing smoke from ordinary fires can be used. Hopocite, an activated mixture of oxides of manganese and copper, for promoting the conversion of carbon monoxide (CO) to carbon dioxide (CO₂). Filter pod for removing smoke from ordinary fires.*

*(For the removal of toxic smoke, a special filter is located at the bottom of some canisters. The acid gas and the organic vapor sorbents may be present as single or multiple layers or as a mixture.)

(b) Air-supplied respiratory protective equipment

If the conditions of exposure exceed the limitation of the respirators (as regards the concentration of air pollutants or duration of exposure), air-supplied equipment may be necessary. The worker is supplied with air through a hose connected to an uncontaminated source. Self-containing apparatus that have built-in supplies of oxygen or air permit free movement of the worker but have a limited capacity ranging from 25 minutes to two hours. Therefore, they find use mainly in emergency situations.

Compressed air-line apparatus supplies air by a compressor fitted with filters to remove oil and toxic gases, in particular carbon monoxide. When fitted with either a full or half facepiece, it is comfortable enough to be worn over long periods and offers efficient protection. Positive pressure suits, fed by an air-line, can give all-over protection, e.g. in abrasive blasting or removal of friable asbestos (figure 13).

All persons expected to wear respiratory protective equipment should be trained in its correct use and declared medically fit to wear it.
All respiratory equipment should be cleaned and examined after each use, and inspected at least once a month. After cleaning, a respirator should be kept in a dust-proof container. Chemical filters have a limited shelf-life and should be disposed of and replaced as soon as their indicated expiration date is reached.

(ii) **Eye protection**

A full facepiece respiratory protective equipment will provide satisfactory eye protection in exposure to airborne irritants.

(iii) **Protective clothing**

The matter of protective clothing is specifically dealt with in the Asbestos Convention, 1986 (No. 162). The main purpose of protective clothing is to prevent excessive exposure to airborne asbestos fibres arising from dust which settles on the work clothing. The Code of Practice *Safety in the use of asbestos*\(^9\) provides that where personal clothing may become contaminated with asbestos dust, the employer, in accordance with national regulations and in consultation with workers' representatives, should provide appropriate work clothing. Where airborne asbestos levels necessitate the use of respirators, special protective clothing should also be provided and worn. Such special protective clothing should cover completely all work clothing so that no asbestos dust is retained when the protective clothing is removed. Suitable head covering should also be provided. When reusable protective or work clothing is provided,
separate locker rooms should be available so that contaminated clothing can be stored separately from personal clothing.

3.3.6 **Housekeeping and maintenance**

In any enterprise, good housekeeping and maintenance are the essential routine support to preventive and control measures. Housekeeping includes day-to-day cleanliness, tidiness and good order in all parts of the enterprise. Maintenance covers the work done to keep building, plant, equipment and machinery in safe and efficient working order and in good repair, and the regular cleaning of walls, ceilings and fixtures.17

The basic elements of good housekeeping in relation to the control of respiratory hazards at the workplace are: adequate and immediate disposal of the waste liable to become airborne; periodic cleaning of the workplace with vacuum cleaners or water in order to avoid accumulation of dust; keeping all containers with volatile irritant chemicals tightly closed; and organisation and general cleanliness. The implementation of good housekeeping should be facilitated by measures taken at the design and construction of the enterprise, and should include smooth surfaces of walls and floors which prevent settling of dust and simplify their cleaning, facilities for adequate cleaning such as vacuum cleaning and water supply, and arrangements for waste disposal.6

Badly maintained machinery and engineering controls (e.g. ventilation systems) which allow hazardous substances to escape into the air are an insidious danger to health.

It should be remembered that cleaning and maintenance personnel may be exposed to excessive concentrations of harmful agents during their work. Consequently, all preventive and control measures which apply to production workers should be fully applied also to cleaning and maintenance staff.

3.3.7 **Personal hygiene**

Personal hygiene at work is always necessary, but it is essential for workers exposed to toxic substances, to dirty work or the risk of infection. It is difficult to practice personal hygiene unless adequate sanitary facilities are provided. On the other hand, the most scrupulous personal hygiene will be of little avail against an environment contaminated with dangerous gases, dust or fumes.18

Washing facilities should be provided at all enterprises; in dirty and dusty occupations, showers are required.
Cloakroom facilities should ensure that no contamination of outside clothes by hazardous agents is possible. Special lockers may need to be provided for work or special protective clothes, and for outside clothes. After taking off their work or protective clothes, the workers should pass through the washroom before reaching their clean outside clothes.

In dusty workplaces, the work and protective clothes as well as the personal protective equipment may need to be cleaned by a vacuum cleaner (e.g. in exposure to asbestos dust); shaking and brushing should be prohibited.

Arrangements should be made to launder working clothes separately from other textiles, in order to avoid the contamination of the latter by hazardous agents, and their transmission outside the working environment.

3.4 Surveillance of the working environment

The quality of the working environment through compliance with safety and health standards has to be ensured by surveillance at the workplace. Systematic surveillance of the hazards to which workers are potentially exposed is called monitoring. According to the Occupational Health Services Convention, 1985 (No. 161), surveillance of the working environment is one of the main tasks of occupational health services.

The scope of the surveillance has been defined in Article 5 of the Occupational Health Services Recommendation, 1985 (No. 171), as follows:

The surveillance of the working environment should include -

(a) identification and evaluation of the environmental factors which may affect workers' health;

(b) assessment of conditions of occupational hygiene and factors in the organisation of work which may give rise to risks for the health of workers;

(c) assessment of collective and personal protective equipment;

(d) assessment where appropriate of exposure of workers to hazardous agents by valid and generally accepted monitoring methods;

(e) assessment of control systems designed to eliminate or reduce exposure.
Although the Occupational Health Services Convention, 1985 (No. 161), assigns the surveillance of the working environment to the occupational health services as one of its functions, it should be understood that these services are not solely responsible for surveillance. As a matter of fact, the surveillance should be a continuous daily activity of everyone involved, from the worker to the employer. Basic surveillance is carried out by simple observation and every worker should be trained to be able to identify those factors which may affect workers' health. This view is supported by Article 19 of the Occupational Safety and Health Convention, 1981 (No. 155), which provides that a worker should report forthwith to his immediate superior any situation which he has reasonable justification to believe presents an imminent and serious danger to his life or health. As regards occupational lung diseases, this applies in particular to the exposure to irritant substances or infectious agents. However, as the same provision appears in Article 9 of the Asbestos Recommendation, 1986 (No. 172), it is evident that it has been applied also to the exposure to substances which can have a serious delayed effect.

The obligation of the employer to measure concentrations of airborne dust in workplaces is directly expressed only in the Asbestos Convention, 1986 (No. 162) (Article 20). However, without measuring concentrations of air pollutants at workplaces the employer may frequently not be in a position to comply with his responsibility for appropriate protective and control measures, as provided for by the Occupational Safety and Health Convention, 1981, as well as by the Working Environment (Air Pollution, Noise and Vibration) Convention, 1977 (No. 148).

Simple observation of work processes and the working environment is the first step in any surveillance. The observation may be sufficient in cases of definite lack of adequate control measures and of overexposure of workers, and the evaluation based on such observation may justify the recommendation of control measures. No expensive determination of the level of overexposure may be needed. Visits to the workplace and observation are also necessary to provide assurance that no deterioration has occurred at workplaces initially evaluated as satisfactory.

Measurement of concentrations of air pollutants may be necessary for the following main purposes:

- to assess the compliance of the concentration of air pollutants with the established exposure limits;
- to assess the possible effect of technological changes or control measures on the concentrations;
- to identify the main sources of air pollution;
to assess the level of exposure of the workers.

Each of the purposes requires a different measurement strategy.

The exposure limits are currently expressed as time-weighted, whole-shift concentrations and short-term peak concentrations, where necessary. In practice, the concentration of air pollutants cannot be measured at all workstations and at all times. A limited number of air samples are usually taken in order to estimate the average concentration of the workplace and compare it with the exposure limit. The sampling site and duration are selected so as to ensure the representativeness of the result. The sampling is carried out at fixed sites (area sampling). Unless self-reading instruments are used, the samples are later analysed by appropriate methods.

The same sampling strategy can be applied to following up the possible effects of technological modifications, to engineering control measures, or to inadequate maintenance and housekeeping in an ageing plant.

Should the sources of the air pollution be identified, sampling takes place close to the source of the emission. Spot samples of short duration (a few minutes) may be adequate, in particular for high concentrations of chemicals, and less precise methods of detection used (e.g. indicator tubes).

The actual level of exposure of the workers can be assessed by sampling the air in the vicinity of the workers at their breathing level. The most suitable sampling devices are personal samplers, carried by the worker during the representative period with the sampling head as close to the worker's nose as possible (e.g. attached to the hard hat).

Some chemicals that are absorbed into the body can be detected in urine, blood or other media and may provide useful information about the level of exposure of the individual. This type of surveillance of exposure is called biological monitoring of exposure. However, only few substances causing occupational lung diseases can be so monitored (e.g. cobalt in exposure to hard metal dust, vanadium in exposure to vanadium pentoxide).

The evaluation of the level of air pollution and of the exposure of the workers requires special knowledge, and shall be carried out by, or in close co-operation with, an experienced industrial hygienist. The results of the measurements should be recorded. The files should be kept for a sufficiently long period in order to enable the assessment of a possible relation between later health impairment and the exposure. In exposure to silice, coal, mine dust, asbestos or carcinogenic substances, several decades may be necessary.
3.5 Surveillance of the health of workers

The fundamental international instruments in the field of occupational safety and health, such as the Occupational Safety and Health Convention, 1981, the Occupational Health Services Convention, 1985, and the Working Environment (Air Pollution, Noise and Vibration) Convention, 1977, and the relevant Recommendations underline the preventive aspects in the protection of workers' health. All these and other instruments contain provisions concerning the duties of authorities, such as the prescription of measures to be taken for the protection of workers' health and the establishment of inspection systems, the duties of employers and workers in the implementation of these measures, provisions concerning surveillance of the working environment and workers' health and education and training. It is evident that medical surveillance of the health of workers represents only a part in a comprehensive system of activities aimed at the protection of workers' health. The first priority should be given to engineering control measures at the workplace and appropriate work practices. These measures may be complemented by the use of personal protective equipment as well as by administrative control such as schedules where workers spend limited amounts of time in areas with potential exposure, with the overall result that the average exposure is at or below the recommended exposure limit. This whole complex of measures shall keep the worker free from significant exposure to health hazards.

Unfortunately, due to limitations of a technological and economic nature, it is not always possible to eliminate all health hazards from the workplace. It is in these instances that the health examinations of workers start to play a major role. The main purpose of health examinations is to assess the ability of a worker to carry out certain jobs from the medical point of view, to assess any health impairment which may be related to the exposure to harmful agents inherent in the work process, and to identify cases of occupational diseases in accordance with national legislation.

Medical examinations cannot protect workers against health hazards and they cannot substitute appropriate control measures which have first priority in the hierarchy of actions. Medical examinations can only help to identify health conditions which may make an individual more susceptible to the effect of hazardous agents or detect early signs of health impairment caused by these agents. In some instances, e.g. in cases of pneumoconioses or cancer, even this early detection may be too late to prevent further deterioration of the disease.

Details about health surveillance are given in Convention No. 161 and Recommendation No. 171, 1985. Accordingly, member States of the International Labour Organisation shall progressively develop occupational health services for all
workers in all branches of economic activity and all enterprises. The functions of occupational health services shall be essentially preventive, and one of their basic tasks is surveillance of workers' health in relation to work.

Surveillance of workers' health shall include, in the cases and under the conditions specified by the competent authority, all assessment necessary to protect the health of workers, which may include:

(a) health assessment of workers before their assignment to specific tasks which may involve a danger to their health or that of others;

(b) health assessment at periodic intervals during employment which involves exposure to a particular hazard to health;

(c) health assessment on resumption of work after a prolonged absence for health reasons for the purpose of determining its possible occupational causes, of recommending appropriate action to protect the workers and of determining the workers' suitability for the job and needs for reassignment and rehabilitation;

(d) health assessment on and after termination of assignment involving hazards which might cause or contribute to future health impairment.

Provisions should be adopted to protect the privacy of the workers and to ensure that health surveillance is not used for discriminatory purposes or in any other manner prejudicial to their interest.

Medical examinations should be provided free of charge to the workers and, as far as possible carried out in their regular working hours, without loss of earnings.

The practical implementation of these provisions should consider the following questions:

- Under which conditions shall medical examinations be prescribed?
- What type of examination is required?
- Who carries out the examination?
- How often would examinations take place?
- What type of intervention shall follow after the examination?
The international labour Conventions and Recommendations do not provide that every worker shall undergo a health assessment. Health examinations apply only to those workers who are potentially exposed to certain occupational health hazards, or who are liable to put the health of others in danger.

The latter provision is a clear indication to professions such as airline pilots, bus drivers or crane operators. As a rule, competent authorities prescribe the obligation for health examinations of workers in such professions, the type of examination and the level of health required, e.g. minimum visual or hearing acuity, physical and mental ability.

The implementation of the former provision - examinations in case of potential exposure to certain occupational health hazards - requires more consideration. It must be decided whether a certain occupational health hazard exists in a workplace at such a level that it may impair the health of the worker and that for this reason a health examination of the worker is necessary.

As a basic check-list for the identification of potential health hazards, the List of Occupational Diseases (amended 1980) appended to the Employment Injury Benefits Convention, 1964 (No. 121), can be recommended (Appendix 1). It lists the major known causes of occupational diseases and although its main purpose is to provide guidance for compensation of occupational diseases, it can also serve for their prevention. Additional hazards, not mentioned in the list, can be added according to national conditions.

If the presence of an agent mentioned in the list is identified in the workplace, the level of exposure to it should first be assessed. The decision whether health examinations shall take place or not can be based on comparison of the actual level of the hazardous agent in the workplace within a well-established exposure limit or prescribed action level.

As regards airborne mineral dust, a WHO expert group considered as health-based exposure limits 40 ug of silica per m³, and 0.5 to 4.0 mg per m³ of mixed dust in coal mines, expressed as time-weighted average whole-shift concentrations of respirable fractions of dust in the breathing zone. Similar approaches can be applied in the case of toxic chemicals and other agents.

The assessment of health is based on a full medical examination, i.e. taking the history of the individual, his clinical examination by a physician, taking blood pressure and routine urinalysis. Special attention is to be given to the status of the possible target organ or system susceptible to be damaged by the hazardous agents, and special examination or laboratory tests may be required for this purpose.
In exposure to air pollutants damaging lungs, a chest X-ray and elementary lung function tests are the basic complementary examinations. X-ray changes in pneumoconioses should be evaluated according to the ILO International Classification of Radiographs of Pneumoconioses. Reference values for lung functions have been established by the Commission of the European Communities. However, it should be kept in mind that they may not always be valid for populations in other parts of the world.

It is evident that the decision about the type of examination requires special knowledge and so does the interpretation of the results of examinations. For this reason, preventive examinations should be confined to occupational health services which ensure a good quality of health assessment. Only qualified specialists in occupational medicine should be authorised for the interpretation of abnormal results, health intervention and notification of occupational diseases. It is highly recommendable that health assessment of workers is concentrated to a limited number of specialised occupational health clinics or departments in a country.

The frequency of examinations shall correspond to the nature of the hazard and probable development of the related health impairment. Even at high-level exposure to silica dust, the first signs of dust-related lung disease - silicosis - rarely appear earlier (in present industrial conditions) than after a few years of work. It would be superfluous to request health examinations at intervals of several months, except in the case where the physician finds particular reason for concern in the individual's health condition. Other types of occupational diseases, in particular those caused by irritant or toxic chemicals, may develop in a short period, particularly during high intensity of work. Preventive examinations of potentially exposed workers should be concentrated around such periods with higher probability of development of respiratory signs and symptoms and not be planned in advance for a fixed date.

These two borderline examples indicate that the decision of the frequency of preventive examinations should not be an administrative question, but an operational decision by the responsible occupational health physician in co-operation with an occupational hygienist and other responsible people.

If there exists a reliable indicator of the status of the target organ, appropriate screening tests can be inserted between the full medical examinations which can then be carried out at longer intervals. Examples of screening tests are chest X-rays in exposure to silica or asbestos and lung function tests in exposure to vegetable dust.

Occupational health services should record data on workers' health in personal confidential health files. These files should
also contain information on jobs held by the workers, on exposure to occupational hazards involved in their work, and on the results of any assessment of workers' exposure to these hazards. Where the files contain personal information covered by medical confidentiality, access to the files should be restricted to medical personnel. The files should be kept for a sufficiently long period in order to enable the assessment of a possible relation between late health impairment and the baseline status (see also section 3.4).

On completing a prescribed medical examination for the purpose of determining fitness for work involving exposure to a particular hazard, the physician who has carried out the examination should communicate his conclusions in writing to both the workers and the employer. These conclusions should contain no information of a medical nature; they might, as appropriate, indicate fitness for the proposed or held assignment and specify the kinds of jobs and the conditions of work which are medically contra-indicated either temporarily or permanently.

Where an occupational disease has been detected through surveillance of a worker's health, it should be notified to the competent authority in accordance with national law and practice. The employer, the worker affected and workers' representatives should be informed that this notification has been carried out.

3.6 Employer's responsibility

Work with hazardous materials can be done with a very high standard of safety. To achieve this status is the duty of the employer or the management of the enterprise.

Article 16 of the Occupational Safety and Health Convention, 1981 (No. 155), provides that employers shall be required to ensure that, so far as is reasonably practicable, the workplaces, machinery, equipment and processes under their control are safe, and that also the chemical, physical and biological substances and agents under their control are without risk to health when appropriate measures of protection are taken. The same obligation is expressed in other international instruments.

In order to comply with their responsibility, employers must be aware of the nature of the hazard, of the ways by which the hazardous material may become airborne, of the potential health consequences of the exposure to the agent, and of the preventive and control measures.

In practice, the management of the enterprise should start with the identification of the potential health hazards inherent in the work process and, with the support of occupational health
services, to assess the degree of the health risk and its causes. The simple presence of a hazardous substance does not pose any special health risk if it is kept under control. The causes of the risk may be the inappropriate design of the plant or of the production process, incorrect work practices, failures in repairs, maintenance or housekeeping, or lack of appropriate engineering control measures. According to the identified causes, the employer should take measures to eliminate the hazard or to put it fully under control, and to improve the working environment so that the exposure of the workers is at or below the established exposure limits.

When it is not possible to achieve this by means of appropriate work practices, engineering control and administrative measures, the employer should provide personal protective equipment.

When required by national legislation, the employer should notify certain types of work to the competent authority.

Employers should ensure that workers are familiar with the hazards inherent in their work, the prevention and control of these hazards through appropriate work practices, engineering control measures and use of personal protective equipment. Workers should be instructed on and trained in normal working operations so that each worker is competent to carry out these duties correctly.

Finally, the employer is responsible for making arrangements for the surveillance of the working environment and the health of the workers.

3.7 Workers' participation

Co-operation between management and workers and their representatives should be an essential element in all preventive and control measures within the enterprise. According to Article 6 of the Working Environment (Air Pollution, Noise and Vibration) Convention, 1977 (No. 148), workers shall be required to comply with safety procedures relating to the prevention and control of, and protection against, occupational hazards in the working environment. Article 19 of the Occupational Safety and Health Convention, 1981 (No. 155), provides that workers in the course of performing their work, co-operate in the fulfilment by their employer of the obligations placed upon him.

Workers can play an extremely active role not only by fully complying with correct work practices and applying all safety and health instructions pertaining to their work, but by constantly watching over the safety of their workplaces. Any defects found in the course of work should be reported without delay to a
A competent supervisor. Workers should always make proper use of protective equipment made available for their protection, and strictly observe personal hygiene.

In order to be able to fully co-operate with the management in matters of occupational health and safety, each worker should be fully informed in an adequate manner of the hazards involved in their work and trained in appropriate work practices and preventive and control measures.

At national and enterprise levels, workers' organisations can significantly contribute to the establishment and implementation of preventive and control measures. There should be regular consultations between employers' and workers' organisations; all the important instruments in the field of occupational health and safety provide for consultations with the employers' and workers' organisations.

Given adequate information, workers' organisations can participate in examining factors related to health risk at the workplace and propose measures to counteract such risks. Workers' organisations should continue to develop and promote awareness and expertise among their members in matters of occupational health and safety. Training activities organised by workers' organisations are especially important.

Notes


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4. OCCUPATIONAL LUNG DISEASES: THEIR CAUSES AND MANAGEMENT

4.1 Pneumoconioses caused by sclerogenic mineral dust

The ILO list of occupational diseases mentions silicosis, anthraco-silicosis, asbestosis and silico-tuberculosis.

4.1.1 Silicosis

Silicosis is a pneumoconiosis (pulmonary fibrosis, scars of the lungs) caused by inhalation of particles of crystalline free silica (silicon dioxide, SiO₂). The main crystalline mineral forms are quartz, tridymite and cristobalite.

(i) Potential exposure risk

Most rocks contain silica. Airborne particles of free silica are produced by blasting, grinding, crushing, drilling, and milling of rocks. Dusts from commercial workings of granite, sandstone, milling sand (silica flour) and heat-treated diatomite are particularly dangerous.

Workers in the following occupations are potentially exposed to the risk: hard rock mining and extraction; civil engineering work with hard rock; stone dressing and polishing; casting, fettling, and sandblasting in foundries and in cleaning buildings; preparation and removal of refractory linings to furnaces, etc., and boiler scaling; ceramic, porcelain and enamel manufacture; and the occupations in which sand is used as an abrasive.

(ii) Effects

The dust particles of 5-15 μm diameter deposited in the airways are cleared by mucociliary movement, but particles of 0.5-5 μm diameter landing in terminal airways or beyond may be retained. Most particles below 0.5 μm diameter remain suspended in the air and are breathed out.

The dust particles retained in the lungs are taken up by macrophages (mononuclear phagocytes) and transported either to the airways and cleared, or to the lung parenchyma. When the
dust-containing cells die, other cells take up the released particles, but these too are killed, creating a continuous low-grade reaction leading to the formation of localised scars (nodules), which mostly occur around the terminal airways (figures 14 and 15). In advanced stages, the nodules grow and merge into large fibrous masses.

Figure 14. Histological structure of a silicotic nodule. The central, hyaline part is composed of concentric rings containing carbon particles, surrounded by cellular halo and emphysematous pulmonary parenchyma (Van Gieson × 50)²
The early stages of silicosis are as a rule not accompanied by any symptoms or signs of respiratory disease. Also, the basic ventilatory lung function tests remain within the normal physiological range; X-rays are therefore the method of detection (figures 16 and 17). In more advanced stages, dyspnoea on exercise develops during exercise. The coalescence of nodules is accompanied by more rapidly progressive breathlessness during exercise and by impaired respiratory function. Bronchitic symptoms, e.g. cough and phlegm due to the deposition of larger dust particles in the airways are less important and sometimes reversible.
The rate of progression of the disease is usually slow. It tends to slow down after exposure ceases, but even when all exposure has ceased, the disease continues to progress, leading occasionally to respiratory or heart failure.

Exposure to 1-2 mg quartz/m$^3$ may cause detectable disease in 5 to 15 years with X-ray changes starting first, followed by lung function abnormalities and the appearance of symptoms. At lower levels of exposure, there is a longer delay in the development of the disease, with symptoms often not appearing until after the exposure has ceased.
Health surveillance

The preplacement health examination before assignment should include a medical history of the employee and a physical examination, with special emphasis on the respiratory system. A chest X-ray should be taken to see if the individual has had pulmonary tuberculosis or any other lung disease. Basic lung function tests should be carried out, including measurement of the vital capacity (VC) and forced expiratory volume in one second (FEV1.0).

The frequency of the periodic examinations depends on the level of dust exposure. If control measures are adequate, three years' interval seems appropriate. However, periodic examination
may not help in preventing the development of silicosis, sometimes long after retirement.

The radiographs shall be interpreted according to the ILO International Classification of Radiographs of Pneumoconioses.3

(iv) Control measures

The suppression of dust by technical control measures (pre-wetting, wet drilling, etc.) should be rigidly enforced and any residual dust should be controlled by proper ventilation. Respirable dust levels and free silica content of dust should be monitored regularly. Whenever explosives are used, workers should be prevented from entering the dusty area until the dust is cleared by ventilation. Dust should be filtered out of exhaust air.

Workers should wear masks, pressure hoods, etc., during breakdown of normal technical dust control measures or in emergency situations. Air-conditioned cabs should be provided for truck drivers and operators of excavators, cranes, etc., during opencast operations in dry climates where water sprinkling is impossible.

There is no uniformity in exposure limits for silica dust in different countries. Exposure limits for total dust are mostly between 0.5 mg/m³ (dust with high silica content - i.e. above 70 per cent) and 5 mg/m³ (dust with silica content less than 10 per cent). For respirable dust the limits range from 0.1 mg/m³ to 0.2 mg/m³. The limits for cristobalite and tridymite are usually half of those for quartz. The tentative health-based exposure limit for free crystalline silica recommended by the WHO4 is 40 ug respirable dust per m³.

4.1.2 Silico-tuberculosis

The term refers to the disease resulting from the interaction of silicosis with tuberculosis of the lungs.

Workers exposed to silica are at an increased risk of tuberculosis. The infectious agent is usually Mycobacterium tuberculosis but other types (e.g. M. marinum and M. kansasii) may also be responsible. The risk increases with the severity of silicosis, and factors favourable for spreading tuberculosis include, for example, crowded working conditions, poor nutrition, and a high prevalence of infection in the community. The suspicion of tuberculosis in cases of silicosis should arise whenever there is a sudden increase in symptoms or X-ray changes, fever, loss of weight, or haemoptysis. The progression of X-ray changes continues to be more rapid even when the infection is
controlled. The most reliable index of diagnosis or cure is the culture of mycobacteria in sputum; other indices are less reliable. Previous tuberculosis, treated or not, probably increases the risk and severity of silicosis.

It is important to prevent tuberculosis in silicosis patients. When the incidence of tuberculosis in the community is high, vaccination and chemoprophylaxis should be considered, although their value has not been proved with certainty. Tuberculosis patients should be treated early. The results of antituberculosis treatment are considered, in general, to be less favourable when silicosis is also present.

4.1.3 Anthraco-silicosis

The other frequently used (but also rather outdated) term is black lung. Recently, both have been replaced by the expression coalworker's pneumoconiosis. Anthraco-silicosis (coalworker's pneumoconiosis) is a pneumoconiosis caused by exposure to mixed coalmine dust in which free silica is not the dominant fibrogenic component. In most situations, the relative importance of the components of the mixed dust in coalmines is not known.

(i) Potential exposure risk

Coal contains mostly carbon and some hydrogen, sulphur, and phosphorus, and a variety of rocks, some containing free silica. The composition varies from mine to mine and seam to seam. Mixed dust in coalmines may be derived from shale, oölite, millstone grit, kaolinite, slate, limestone, etc., all of which are frequently present within, above, and below coal seams. Coal itself may be the least important component of mineral dust.

Mixed dust formed during coalmining is most liable to accumulate in deep mines, especially where mechanical cutting of rock above and below narrow seams occurs. Opencast workings are less dependent on artificial ventilation. Once separated from rock, the distribution and use of coal creates less harmful dust.

All workers in deep mines (especially those involved in development work and those heavily exposed in opencast mines), workers in loading operations (e.g. on ships) and those in industries using coal (e.g. the steel industry) are at greatest risk of exposure.

(ii) Effects

The effects are very similar to those described in silicosis (see section 4.1.1).
Clinical disease is preceded for many years by X-ray signs of simple pneumoconiosis, which by itself leads only to a slight acceleration in the normal rate of lung function deterioration. The deterioration of lung function is associated with the formation of large rounded masses containing small amounts of collagen. This condition is known as progressive massive fibrosis. In most coalworkers, it develops only when the dust burden is very high.

If dust exposure is stopped at the early stage of simple pneumoconiosis (usually at <2/2 p or q in the ILO International Classification of Radiographs of Pneumoconioses), little disabling disease occurs unless the miner's work had involved exposure to a high proportion of dust from mine development (rock) work away from the coal-face; or the worker develops rheumatoid disease with synergistic lung changes (Caplan syndrome).

In anthraco-silicosis, serious disability occurs after exposure to much higher dust levels than is the case in silicosis. Furthermore, the disease is less likely to progress.

There are great differences in the incidence of the disease and its course among different coalfields. The reasons for this are not well known.

(iii) Health surveillance

Health surveillance should follow the same principles as those adopted for silicosis (see 4.1.1).

(iv) Control measures

The control measures recommended for silicosis also apply to anthraco-silicosis (see 4.1.1).

Exposure limits in most countries are related to the free silica content of coalmine dust.

The health-based exposure limit tentatively recommended by the WHO for coalmine dust with a free silica content equal to or less than 7 per cent (mass) of the respirable mixed dust fraction, is in the range of 0.5 to 4.0 mg/m³. Limits above 0.5 mg/m³ should be applied only when there is sufficient epidemiological evidence to show that the particular coalmine dust is relatively harmless, and there is a limited risk of developing simple pneumoconiosis radiographic category 1 during the whole working life. For coalmine dust with a free crystalline silica content higher than 7 per cent (mass), the limit for free crystalline silica should be applied.
Asbestos-related diseases: Asbestosis, lung cancer, mesothelioma

Asbestos is a generic term for a group of naturally occurring fibrous mineral silicates. They are crystalline in form and are capable of splitting longitudinally into single fibrils or fibre bundles. Two groups and six mineral types are recognised: serpentine group - chrysotile; amphibole group - crocidolite, amosite, anthophyllite, tremolite, actinolite. All are chain hydrated silicates of magnesium, except crocidolite, which is a silicate of sodium and iron. Crocidolite and amosite have a large iron content. The amphiboles split into straight fibres of variable but mostly very narrow diameter (about 0.1 \( \mu \)m). Chrysotile occurs in sheets that curl up, producing hollow, tube-like fibrils of about 0.03 \( \mu \)m in diameter. The properties that make asbestos unique are: relative insolubility, high tensile strength, and resistance to heat and acids (amphiboles only).

Most of the commercially exploited asbestos is used in making asbestos cement products such as pipe and roofing sheets. Further uses are for friction materials such as brake linings, floor coverings, plastics, insulation material, fireproof textiles and many others.

(i) Potential exposure risk

Workers in the following occupations are at greatest risk: mining, milling, and processing of asbestos; transport of mined or milled asbestos; manufacture of asbestos products; disposal of waste material from asbestos mining, milling, etc.; any use or dismantling of asbestos products that causes airborne asbestos dust.

(ii) Effects

Inhaled airborne fibres of less than about 3 \( \mu \)m diameter penetrate the airways. Since fibres of chrysotile are curly, they enter the lungs less easily than the amphiboles. A substantial proportion of the fibres entering the lungs is cleared from the respiratory tract. Of the fibres retained in the small airways and alveoli, some short fibres are engulfed by macrophages and carried to the lymph nodes and other tissues. Some of the fibres (especially the amphiboles) are coated with an iron protein complex and become "asbestos" or "ferruginous" bodies. It is thought that chrysotile gradually disappears from the body, but evidence of this is scanty.

After long or heavy exposure, there is substantial retention of asbestos fibres. This gradually leads to progressive, diffuse interstitial pulmonary fibrosis (scarring of the lungs) called...
asbestosis. Variable degrees of pleural fibrosis are often found, and sometimes hyaline or calcified pleural plaques appear, which may not necessarily be asbestos-related.

Under current industrial conditions, asbestosis is rarely seen in workers with less than ten years' exposure. Advanced asbestosis may cause dyspnoea and respiratory insufficiency.

After a latent period, rarely less than 20 years and up to 40 years or more after first occupational exposure, lung cancer, or malignant mesothelioma of the pleura, may develop.

The mechanisms of carcinogenesis are unknown. In the case of mesothelioma, animal experiments suggest that very fine fibres of about 0.1 μm diameter with a length of 8 μm or more are responsible. Mesotheliomas have rarely been reported after pure chrysotile exposure, but following exposure to crocidolite, amosite, or mixtures containing these minerals, mesotheliomas have accounted for some 2 to 16 per cent of all deaths. Lung cancer occurs with all fibre types. These malignant diseases are clinically indistinguishable from cancer unrelated to asbestos exposure. There is clear evidence that the morbidity on lung cancer is much higher in cigarette smokers exposed to asbestos dust than in non-smokers. The cumulative risk of lung cancer in workers who smoke and who are exposed to asbestos is greater than the sum of the risks of lung cancer from smoking and from asbestos exposure separately.

(iii) Health surveillance

The preplacement examination should include a medical history, a physical examination, chest X-ray, and lung function tests in order to establish baseline data for surveillance and to prevent persons with respiratory diseases from being exposed to asbestos.

In medical terms the periodic examination is the same as the preplacement one. It should be carried out at intervals depending on the level of exposure at the workplace, age of the worker, and the results of previous health examinations. Chest X-rays shall be evaluated according to the ILO International Classification of Radiographs of Pneumoconioses.

Since it takes a long time for asbestosis to develop, five-year intervals between health examinations may be appropriate during the first ten years of work. Thereafter, the examinations should be more frequent.

The benefit to be derived from the early treatment of lung cancer is probably insufficient to warrant less than one-year intervals between routine X-rays. Because of the possibility of
late development of asbestos-related diseases, supervision should continue even after the cessation of work involving exposure to asbestos.

(iv) Control measures

Most industrialised countries have adopted an exposure limit of one or two fibres of chrysotile asbestos per ml air. The exposure limits for amosite and crocidolite are as a rule much lower — up to one-tenth of the chrysotile values.5

Concern remains that the hazard is not adequately reflected by fibre counts done with an optical microscope since by this method finer fibres are not detected. As a result, the hazard may vary considerably in different industrial processes even when the optically assessed environmental concentration of asbestos is the same.

The spraying of asbestos is now widely banned, and the use of crocidolite is also banned or severely restricted in many countries, as provided for by the Asbestos Convention, 1986.

Successful dust control involves engineering control measures, appropriate work practices, provision of protective equipment (respirators, protective clothing), good housekeeping and the use of vacuum cleaning of the premises, provision of showers and laundry facilities. All workers should be informed of the nature of the hazard and of methods of protection.

Engineering controls should include mechanical handling, ventilation and redesign of the process to eliminate, contain or collect asbestos dust emissions by such means as process separation, automation or enclosure, bonding asbestos fibres with other materials to prevent dust generation, general ventilation of the working areas with clean air, local ventilation of processes, operations, equipment and tools for the prevention of dust dissemination, use of wet methods where appropriate and separate workplaces for certain processes.

Detailed information on preventive and control measures are to be found in the ILO code of practice Safety in the use of asbestos.5

4.2 Bronchopulmonary diseases caused by hard-metal dust

Hard metal is a term used for extremely hard sintered metal carbides of tungsten (to which small amounts of titanium, tantalum, vanadium, molybdenum, or chromium carbide have been added) bonded together by cobalt (also iron and nickel). The
pulverised and compressed constituents are heated to a high
temperature (1,500°C) and cooled abruptly.

Many metals are usually present in the dust: tungsten
(67-90 per cent), cobalt (6-20 per cent), and tantalum, titanium,
vанадий, iron, and niobium in amounts not exceeding 2 per cent.

Hard or sintered metals are used in the manufacture of
tools, drills, and metal parts of particular hardness (90-95 per
cent that of diamond).

(i) Potential exposure risk

Workers at greatest risk are those engaged in the production
of sintered carbides (mixing, pulverising, forming, furnace
heating, machining, precision grinding) and in the manufacture of
tools and machine parts, as well as those responsible for
sharpening the tools produced. Although those engaged in
grinding and sharpening are the most exposed to the hazard,
workers engaged in other tasks in the immediate vicinity in the
same workshop may also run a high risk of exposure.

(ii) Effects

In the majority of those exposed, a variety of irritative
symptoms are seen, including cough, allergic rhinitis,
asthma-like dyspnoea, and shortness of breath on exertion. The
symptoms improve after the cessation of exposure. Diffuse
interstitial pulmonary fibrosis is far less common and affects
1-4 per cent of workers.

It is generally agreed that the association of tungsten with
cobalt is responsible for the disease.

(iii) Health surveillance

The preplacement examination should include a medical history
and a physical examination in order to identify persons with
allergic skin and respiratory diseases. A chest X-ray should be
taken and basic pulmonary function tests should be carried out.
Periodic examinations may need to be carried out annually.

(iv) Control measures

The exposure of workers must be controlled by appropriate
technical measures (enclosing machines and applying local exhaust
ventilation, etc.) in order to reduce dust concentrations below
the recommended exposure limits. The use of personal protective
devices (respirators) may be necessary during work operations involving exposure to very high concentrations of dust.

The exposure limit adopted in most countries for soluble compounds of tungsten is 1 mg/m³ of air and for insoluble compounds 5 mg/m³ of air (as tungsten); for cobalt metal fumes and dust, the exposure limit is 0.1-0.5 mg/m³ of air.

4.3 Bronchopulmonary diseases caused by cotton, flax, hemp or sisal dust (byssinosis)

Byssinosis is caused by exposure to airborne dusts of cotton, flax, soft hemp or sisal. The dust is composed of cellulose fibres, plant debris (broken leaves and bracts), earthy matter (soil), and saprophytic micro-organisms that grow on the material during storing or retting (bacteria, fungi).

(i) Potential exposure risk

Industrial processing of cotton involves various operations: (a) ginning, in which cotton seeds are separated from lint; (b) bale pressing (in exporting countries); and (c) cotton manufacturing, which involves bale opening, blowing, carding, spinning, preparation of yarn, and weaving. All these operations are usually dusty. Airborne dust from weaving differs from that produced in earlier processes and contains particles of the sizing material.

In the processing of flax, the first operation is the retting of the dried flax plant in water tanks. Then, the flax fibres are dried and broken in order to separate the fibres for spinning and weaving. The last three operations are usually dusty.

Workers in the textile industry carrying out dusty operations are at greatest risk. In some developing countries, flax breaking and spinning are performed at home, causing dust exposure to the workers and their families.

(ii) Effects

Dusts that cause byssinosis give rise to two main types of clinical response: chest tightness (which is characteristic for the disease), and respiratory tract irritation.

The chest tightness or shortness of breath occurs on the first day of work (usually towards the end of the workshift) after the weekend break or after holidays. There is often a temporary post-shift decline of ventilatory capacity of the lungs. Within one or two days, the symptoms tend to disappear,
but as the disease progresses (if work in unfavourable conditions continues) the worker may become more severely affected with symptoms extending to the whole shift, the next day and then to every working day.

The irritation of the respiratory tract manifests itself by a cough related to dust exposure, which may eventually be associated with persistent phlegm.

Subjects with either the characteristic symptoms of byssinosis or of respiratory tract irritation may eventually develop chronic obstructive pulmonary disease (chronic bronchitis, emphysema) indistinguishable from that unrelated to occupational dust exposure. Chest X-rays do not show any typical changes. The decline in ventilatory capacity may become permanent.

Prevalence rates of byssinosis varying from 20 per cent to 50 per cent have been reported in cotton carding rooms with respirable dust concentrations between 0.35 mg/m$^3$ and 0.60 mg/m$^3$. Prevalences of less than 10 per cent occur only in workrooms with respirable dust concentrations of less than 0.1 mg/m$^3$.

(iii) **Health surveillance**

The preplacement health examination should include a medical history and a physical examination, with special attention to allergic and respiratory diseases. Chest X-ray and basic pulmonary function tests should also be carried out.

The periodic examination is repeated usually once a year. A questionnaire for byssinosis should be included in the examination.

A worker with an early case of byssinosis should be moved to work involving less exposure. In these cases, the symptoms usually disappear. Advanced cases with obstructive pulmonary disease should be removed from dust exposure altogether and given appropriate therapy.

(iv) **Control measures**

These mainly involve dust suppression by enclosing dusty operations (gins) and providing appropriate ventilation (e.g. suction-stripping equipment together with downwards exhaust ventilation).

There have been trials of washing and steaming the cotton before processing, but these have not proved to be very effective in eliminating the diseases.
Personal protective equipment, such as filter masks, can be useful if checked regularly for effective and complete air filtration. Unfortunately, the wearing of masks is inconvenient, particularly in hot climates.

The exposure limits for total airborne dust vary in different countries from 2 mg/m³ to 6 mg/m³ of air. The recommended health-based occupational exposure limits (time-weighted averages for inhalable dust, as measured by the vertical elutriator with a cut-off point of 15 μm are: for cotton ginning 0.5 mg/m³; for cotton yarn processing, carding, spinning, etc., 0.2 mg/m³; and for cotton weaving 0.75 mg/m³. For flax, a tentative value of 2 mg/m³, and in workplaces with green or chemically-retted (not dew- or water-retted) flax, a value of 5 mg/m³ have been recommended. For soft hemp, a tentative value of 2 mg/m³ has been recommended.6

4.4 Occupational asthma caused by sensitising agents or irritants

Occupational asthma is caused by the inhalation of sensitising agents or irritants present in the working environment, such as dusts, droplets and gases. The list of causal agents is very long and only the major classes are mentioned here.

Sensitising agents cause bronchial hyper-reaction. They include: material of plant origin (e.g. grain flour, coffee beans, castor beans, colophony (rosin), tea fluff, tobacco, woods such as red cedar, and mansonia); dusts from shellfish and laboratory animals (e.g. rats, mice, guinea-pigs) and from mites, silkworms and other insects; metals (particularly their salts) such as platinum, chromium, and nickel; organic compounds (formaldehyde, phenylenediamine, isocyanates, particularly toluene diisocyanate, trimellitic anhydride, phthalic anhydride, epoxy resins, reactive dyes, and many others); and drugs (particularly antibiotics) and enzymes (detergents derived from Bacillus subtilis, pepsin, papain, etc.).

Irritants cause chemical asthma. They include: strong alkalis, acids, oxidizing agents (ammonia, chlorine, hydrogen chloride, phosgene, hydrogen fluoride, oxides of nitrogen or sulphur, zinc chloride, etc.); and inert dust (non-fibrous and non-toxic dust) in extreme concentrations.

(i) Potential exposure risk

Workers at greatest risk include: those who handle grains and cereals (silo workers, millers, bakers); grain storage workers exposed to mites; workers exposed to dusts from castor
or coffee beans and those involved in tea sifting and packing; woodworkers, sawmill operators, and workers in the furniture industry; printing workers; laboratory workers handling animals; manufacturers of detergent enzymes, platinum refiners (rarely chromium or nickel platers); workers in the chemical and pharmaceutical industries; manufacturers of polyurethane foam using isocyanates; painters and insulation workers; meat wrappers exposed to fumes of polyvinyl chloride (PVC) soft-wrap film; and health personnel.

(ii) Effects

Occupational asthma caused by sensitising agents and irritants is characterised by: (a) acute reversible obstruction of the airways caused by bronchoconstriction, airway oedema or inflammation; and (b) mucous excretion induced by exposure to agents inherent in the work processes. Clinically, these disorders do not differ from other types of asthma. In some circumstances the same agents may cause allergic alveolitis (see section 4.5).

Most of the sensitising agents stimulate, after repeated exposure, the production of specific antibodies which trigger the asthmatic reaction. This condition is characterised by transitory chest tightness, shortness of breath, wheezing and lung function impairment. In an already sensitised individual, the asthmatic reaction usually starts within a few minutes after exposure, but delayed reaction some four to eight hours after exposure - i.e. after the shift or at night - may also occur, sometimes in combination with the immediate reaction. Recovery may take more than 24 hours.

Irritants act by causing direct tissue injury. No previous sensitisation is needed. Asthmatic attacks usually develop during or immediately after the exposure, however some of the irritants (e.g. phosgene or oxides of nitrogen) may induce effects after a latency of several hours (see also section 4.8).

(iii) Health surveillance

The medical examination should include a medical history (with special attention to possible skin and respiratory allergy), a physical examination, and simple lung function tests.

(iv) Control measures

Technical measures should be applied to control the concentration of air pollutants in the working environment. Personal protective devices (respirators, etc.) may be necessary
in some operations. Safe alternatives to irritants and sensitising agents should be sought.

4.5 Extrinsic allergic alveolitis

Extrinsic allergic alveolitis is a general term for a group of disorders caused by a hypersensitivity reaction to inhaled dust, especially dust containing material of fungal origin.

Dust containing organic antigens (particularly thermophilic micro-organisms and moulds) or animal proteins is the main cause of this disease. The same agents may also cause bronchial hyper-reaction (see section 4.4).

The most common types of dust, responsible antigens, and the resulting clinical conditions are summarised in table 2.

Table 2. Causal agents of extrinsic allergic alveolitis

<table>
<thead>
<tr>
<th>Type of dust</th>
<th>Responsible antigen</th>
<th>Disease (common denomination)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouldy hay</td>
<td>Micropolyspora faeni</td>
<td>Farmer's lung</td>
</tr>
<tr>
<td></td>
<td>Thermoactinomyces vulgaris</td>
<td></td>
</tr>
<tr>
<td>Mouldy bagasse</td>
<td>Thermoactinomyces vulgaris</td>
<td>Bagassosis</td>
</tr>
<tr>
<td>Mushroom compost</td>
<td>Micropolyspora faeni</td>
<td>Mushroom picker's lung</td>
</tr>
<tr>
<td></td>
<td>Thermoactinomyces vulgaris</td>
<td></td>
</tr>
<tr>
<td>Cork dust</td>
<td>Cork dust</td>
<td>Suberosis</td>
</tr>
<tr>
<td></td>
<td>Penicillium</td>
<td></td>
</tr>
<tr>
<td>Maple bark</td>
<td>Cryptostroma corticale</td>
<td>Maple bark disease</td>
</tr>
<tr>
<td></td>
<td>(pneumonitis)</td>
<td></td>
</tr>
<tr>
<td>Redwood sawdust</td>
<td>Graphium</td>
<td>Sequoiosis</td>
</tr>
<tr>
<td></td>
<td>Pullaria</td>
<td></td>
</tr>
<tr>
<td>Wood pulp</td>
<td>Alternaria</td>
<td>Wood dust pneumonia</td>
</tr>
<tr>
<td>Mouldy barley</td>
<td>Aspergillus clavatus</td>
<td>Malt worker's lung</td>
</tr>
<tr>
<td></td>
<td>Aspergillus fumigatus</td>
<td>Aspergillos</td>
</tr>
<tr>
<td>Type of dust</td>
<td>Responsible antigen</td>
<td>Disease (common denomination)</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------------------------------------------</td>
<td>----------------------------------------</td>
</tr>
<tr>
<td>Mouldy straw</td>
<td>Aspergillus versicolor</td>
<td>Aspergillosis</td>
</tr>
<tr>
<td>Pigeon, parrot and other bird droppings</td>
<td>Sera, protein and droppings</td>
<td>Bird breeder's lung</td>
</tr>
<tr>
<td>Animal hairs</td>
<td></td>
<td>Animal hair pneumonitis</td>
</tr>
<tr>
<td>Coffee bean</td>
<td>Coffee bean dust</td>
<td>Coffee-bean dust pneumonitis</td>
</tr>
<tr>
<td>Paprika</td>
<td>Mucor stolonifer</td>
<td>Paprika splitter's lung</td>
</tr>
<tr>
<td>Cheese particles</td>
<td>Penicillium caseli</td>
<td>Cheese washer's lung</td>
</tr>
</tbody>
</table>

(i) Potential exposure risk

Workers in agriculture, food and beverage industries (e.g. cheese dairies, breweries), wood and furniture industries, and animal keepers are at greatest risk.

(ii) Effects

Inhaled dust particles that are small enough to be retained in the alveoli and small airways and that contain antigenic material are responsible for the disease. In the early acute phase, the walls of the alveoli and small airways are infiltrated with lymphocytes and there is the formation of characteristic granulomata. The chronic phase is characterised by diffuse interstitial pulmonary fibrosis.

In the acute and subacute disease, symptoms develop about four to eight hours after heavy exposure to dust containing the antigen. They include: headache, fever, nausea, vomiting, chest tightness, breathlessness and coughing. In advanced cases, chest X-rays show small opacities distributed throughout the middle and lower areas of the lungs. The diminution of ventilatory capacity and gas transfer are the main lung function impairments.
Chronic disease may develop after repeated acute episodes, and, occasionally, without an antecedent acute episode. Recurrent exposures to low concentrations of antigenic dust gradually lead to dyspnoea and lung function impairment. Chest X-rays correspond to the development of diffuse interstitial pulmonary fibrosis.

(iii) **Health surveillance**

Preplacement and periodical preventive examinations are of limited value, as the disease is usually caused by high-level accidental exposure to the aerosol of the causal agent.

(iv) **Control measures**

Changes should be made in work practices in order to avoid the multiplication of moulds and bacteria. Technical measures should be applied to control dust in the air of workplaces. Personal protective devices (respirators, etc.) may be necessary in particularly dusty operations.

4.6 **Diseases caused by beryllium and its toxic compounds**

Beryllium is a metal which is used frequently in alloys, in the aerospace industry and in the manufacture of precision instruments and computers, non-arcing cutters for the petroleum industry, X-ray tubes, fluorescent tubes, vacuum electrodes, heater cathodes, and moderators for use in nuclear reactors.

(i) **Potential exposure risk**

Workers exposed to airborne beryllium, its alloys and compounds in the above-mentioned industries are at greatest risk of contacting berylliosis.

(ii) **Effects**

Soluble beryllium compounds (chloride, sulfate, fluoride) may cause acute intoxication, while relatively insoluble forms (metallic beryllium and beryllium oxide) cause chronic disease.

Acute berylliosis arises from accidental inhalation of high amounts of soluble beryllium compounds. It manifests itself as nasopharyngitis, tracheobronchitis, bronchiolitis, or pulmonary oedema, appearing from a few hours to one to two days after exposure.
Chronic berylliosis is caused by slightly soluble beryllium compounds. The allergenic effect of beryllium has a major role in the pathogenesis of berylliosis: it causes the disease by inducing an immunopathological process in susceptible individuals. The disease may develop many years after the cessation of beryllium exposure. A distinctive granulomatous process, which is mainly confined to the lungs, but which may also be found in other organs (liver, spleen, etc.), is typical. The usual early signs and symptoms are breathlessness on exertion, coughing and fever; later on there may be asthenia and rapid weight loss.

The disease may progress rapidly and respiratory insufficiency may develop. Chest X-rays show changes corresponding to the pathological process in the lungs. The lung function tests are impaired.

Although berylliosis usually results from high exposure to beryllium and its compounds, sometimes even brief contact with low concentrations is sufficient to bring on the disease; it may even develop after the cessation of exposure.

(iii) Health surveillance

The health examinations should include a medical history and a physical examination, with special attention to allergic skin and respiratory diseases. Chest X-ray and basic pulmonary function tests are also essential.

In berylliosis, exposure to beryllium must be discontinued.

(iv) Control measures

The exposure of workers to beryllium must be restricted by appropriate technical control measures as well as by personal protection measures including: wearing a respirator in areas with high beryllium content; working in a pressurised suit in particularly hazardous places; compulsory changing of working clothing; and wearing goggles, gloves, etc.

Exposure limits for beryllium in air in different countries range from 1 ug/m$^3$ to 2 ug/m$^3$.

4.7 Lung cancer

The association of environmental factors with lung cancer (precisely, bronchogenic cancer) is well recognised. Among those factors cigarette smoking, air pollution and occupational exposures to specific industrial agents are of major importance.
(i) Potential exposure risk

The following agents and industrial processes have been evaluated by the International Agency for Research on Cancer (IARC) to be known causes of lung cancer in humans:7

- aluminium production;
- arsenic and its compounds (production and use of arsenical pesticides, mining, copper smelting);
- asbestos (see section 3.1.4);
- bis (chloromethyl) ether and chloromethyl ether, technical grade (production),
- chromium compounds, hexavalent (chromium pigments production and use, chromium plating, chromium alloy production, stainless steel welding);
- coal gasification;
- coal-tar pitches/coal-tars (coal distillation, patent fuel, roofing and asphalting);
- coke production;
- haematite mining, underground, with exposure to radon;
- iron and steel founding;
- mustard gas (sulphur mustard) production;
- nickel refining;
- radon (underground mining);
- talc containing asbestiform minerals (production and use).

Workers exposed to these agents and processes are at the greatest risk.

Most of the single agents (but not all of them) are given in the list of occupational diseases (amended 1980) appended to the Employment Injury Convention, 1964 (No. 121) (see Appendix 1).

According to the IARC,7 probably carcinogenic to humans (as causes of lung cancer) are the following:

- acrylonitrile (production);
- beryllium (refining);
- cadmium (smelting, battery production, electroplating, alloy production);
- diethyl sulphate (production);
- epichlorhydrin (production);
- formaldehyde (production, manufacture of resins, use as disinfectant, fumigant and preservative);
- silica, crystalline (see section 3.1.1).

The definite evaluation of the carcinogenic potential for humans of these agents as well as of a number of possible carcinogens will need further research.

(ii) Effects

Biologically and clinically, cancer due to occupation is at present indistinguishable from cancer due to other (mostly unknown) causes. The carcinogenic process which results in the transformation of a normal cell (of the bronchial mucosa in the case of bronchogenic cancer) into a cancer (malignant) cell is still very incompletely understood. There is evidence that the process starts with an irreversible "initiation" stage inducing changes in the genetic information of the cell carried by the DNA (desoxyribonucleic acid), followed by a "promotion" stage which propagates the cell into a malignant growth. By reference to this model, carcinogens can be divided into complete carcinogens (capable both of initiating and promoting), and incomplete carcinogens, only capable of initiation or of promotion. This difference may have practical implications for the establishment of exposure limits for carcinogens; however, the distinction proves very unclear.

For practical consideration of the risk the epidemiological evidence of the incidence rate of lung cancer in the exposed workers in comparison with a non-exposed population remains the most important indicator of the efficiency of preventive and control measures.

(iii) Health surveillance

Preplacement examination shall establish the worker's health status and fitness for the assignment in the exposure to potential carcinogens, particularly with regard to the respiratory system. Unfortunately, there are no reliable methods available that would permit identification of individual susceptibility to the agent. Cigarette smoking should be taken into account because the
The carcinogenic effect of tobacco smoking may be additive or even multiplicative to that of occupational carcinogens.

Periodic examinations aim at the detection of ill-health at the earliest and reversible level. Three methods are available for screening lung cancer: chest X-ray, sputum cytology (to detect cancer cells in the phlegm), and bronchoscopy (direct observation of the bronchi through a fibroscope inserted in the respiratory ways). Only the first two non-invasive methods are suitable for mass screening. Unfortunately, none of them have been able to detect lung cancer in early enough stages for the improvement of the prognosis of the disease and the survival of the patients.

Nevertheless, periodic medical examinations are required by law in most countries where health surveillance of the workers is compulsory.

Health surveillance should continue also after the termination of the employment involving exposure to carcinogens as lung cancer may develop after many years of latency.

(iv) Control measures

In view of the unsatisfactory results of health examinations in the prevention of the irreversible stages of the disease, effort should mainly be concentrated on the implementation of control measures, based on the provisions of the Occupational Cancer Convention, 1974 (No. 139), and Recommendation (No. 147), and the Asbestos Convention, 1986 (No. 162) and Recommendation (No. 172) as well as other sources.5,7,8

Every effort should be made to replace carcinogenic substances and agents by non-carcinogenic ones, or by less harmful substances and agents or technologies, and to use work processes which do not cause the formation, and particularly the emission in the working environment of carcinogenic substances or agents.

Where complete elimination of the carcinogenic hazards is not possible, the exposure of the workers to them should be eliminated or reduced to a minimum in terms of numbers exposed, duration of exposure and degree of exposure. Arrangements should be made for the monitoring of the exposure.

Where engineering control measures cannot decrease the exposure to an acceptable level, personal protective equipment, in particular protective clothes and respiratory protective devices, should be provided and used.

Measures should be taken to prevent the transfer of the carcinogenic substances or agents from the workplace, particularly
on to work or protective clothing. Appropriate washing and showering facilities and changing rooms for the workers should be provided as well as facilities for the laundering of work or protective clothing separately from non-contaminated clothing.

Most countries do not establish exposure limits for carcinogenic substances in view of the uncertainty as to whether a ceiling concentration exists below which there is no increased danger of cancer induction. Nevertheless, some of the carcinogens, e.g. arsenic, chromium, nickel, asbestos, may have an assigned exposure limit with regard to the prevention of other (toxic) effects of the substances, but not with regard to cancer. In some countries, there is a strong belief in a factual threshold for the effect of carcinogenic substances, and exposure limits have been established (e.g. in the USSR). In the Federal Republic of Germany, technical-guiding concentrations for carcinogenic substances are assigned as action levels for protective measures and surveillance by screening techniques. In general, they are considered to be the lowest technologically feasible values.

Regardless of the scientific uncertainty of the threshold effect level, it is generally agreed that there is a relationship between the level of exposure and the incidence of cancer, and that at sufficiently low exposure no excess of cancer incidence may be detectable by current epidemiological methods.

4.8 Bronchopulmonary diseases caused by irritant gases and vapours

The generic term "irritant gases and vapours" covers a whole series of chemicals whose common characteristic is the irritant action they exert on the respiratory system and conjunctivas.

Many irritant gases and vapours may also have a pronounced general toxic action; in some substances the irritant action is the fundamental toxicological characteristic whereas in others it is of only subordinate significance.

The most important gases whose action is primarily irritant include: chlorine and derivatives (hydrochloric acid, phosgene, chlorine dioxide, etc.); fluorine and derivatives (hydrofluoric acid, silicon tetrafluoride, fluosilicic acid, etc.); bromine and iodine; sulphur dioxide and sulphuric acid; nitrogen oxides (nitrous oxide, nitric oxide, dinitrogen tetroxide, nitrogen dioxide), nitrogen dioxide being the most dangerous; ozone; ammonia; acrolein; acetic acid; and formaldehyde.

Other toxic substances which also may have an irritant action include: benzene and its homologues; bromoethane; bromomethane; butadienes; diolefins; ketones; methyl-chloroacrylate (methyl
ester of 2-chloroacrylic acid); phosphine; hydrogen selenide; acrylonitrile; dimethyl sulphate; tetrachloroethylene; carbon tetrachloride; trichloroethylene; metal mercury vapour.

A number of fumes and aerosols, also acting on inhalation, have strong irritant properties on airways; these include those of antimony compounds, beryllium and compounds, cadmium, hexavalent chromium compounds, molybdenum trioxide, nickel carbonyl, osmium tetroxide, vanadium pentoxide and zinc chloride. This explains in particular the health hazards involved in the large inhalation of welding and metal cutting fumes.

(i) Potential exposure risk

The workers exposed to the following are at greatest risk:

- ammonia: refrigeration, manufacture of nitrogen-containing chemicals, soil fertilising, petroleum refining;
- chlorine: paper and textile bleaching, water disinfection, chemical manufacturing, metal fluxing;
- hydrochloric acid: chemical manufacturing, electroplating, metal pickling, in various industries;
- hydrofluoric acid: chemical manufacturing, aqueous solution for frosting, etching and polishing glass;
- nitrogen oxides: manufacturing of acids, nitrogen-containing chemicals, explosives: by-product in many industrial processes and anaerobic fermentation of green silage in silos;
- ozone: arc welding, water and air purification, food and textile bleaching;
- phosgene: manufacturing of organic chemicals, pyrolysis, decomposition product of chlorinated hydrocarbons (e.g. tetrachloroethylene);
- sulphur dioxide: manufacturing of sulphur-containing chemicals, food and textile bleaching, fumigation; by-product of coal burning, smelting and in paper industry.

(ii) Effects

The irritant effect is perceived as a burning sensation in the eyes, nose, throat, or on the chest, which may be accompanied by dry cough and shortness of breath due to bronchoconstriction (see in section 4.4, asthma caused by irritants). The intensity
of the irritant action depends on the chemical structure of the substance, its concentration in the air and solubility in water, duration of the exposure and the ventilation rate: during sedentary work, higher concentrations of irritants are tolerated than during physical exercise.

Gases with high water solubility act on upper respiratory airways within seconds and may be fully absorbed there, with the result that they do not reach the deeper bronchi. Highly water soluble gases, such as ammonia, hydrogen chloride or hydrogen fluoride affect primarily the conjunctivas and the upper sections of the respiratory tract: the irritation is incisive and there is a burning sensation in the eyes, profuse lacrimation and coughing. This acts as a warning to the subject who can withdraw from exposure before the irritant substance penetrates further along the respiratory tract.

The moderately soluble gases, such as chlorine or fluorine gas can also reach the lower respiratory tract, and act within minutes. They produce upper respiratory irritation as well as bronchospasm.

The most insidious gases and vapours are those which have low solubility (nitrogen oxides, phosgene) and no warning odour. They penetrate into the deep portions of the respiratory tract without causing obvious immediate distress, they act predominantly on the alveoli some six to 24 hours after exposure, producing lung oedema without preliminary warning.

Nevertheless, the severity of the lesions depends fundamentally on the concentration and length of exposure. At high concentrations, ammonia may also cause acute pulmonary oedema and nitrogen oxides may irritate the upper respiratory tract. However, high concentrations of irritant gases and vapours do serious damage even when exposure is of only very short duration. Long-term and repeated exposure to excessive concentrations of irritants can cause chronic conjunctivitis and inflammation of the mucosae of the respiratory ways.

(iii) Health surveillance

Preplacement medical examinations (medical history, physical examination) should aim at detecting disorders which may potentiate the action of irritant substances (in particular inflammatory and allergic processes of the respiratory system). Periodical medical examinations shall also be offered. First-aid personnel should be prepared and trained in advance in all workplaces where there exists a potential risk of high-level exposure to respiratory irritants. The victim of the over-exposure should be immediately removed to the fresh air; all muscular effort on the part of the victim should be avoided.
The victim should be conveyed to the hospital promptly, or a doctor should be called immediately and informed of the substance responsible. Medical supervision and bed rest may be necessary up to 48 hours after the exposure, even in apparently benign cases of exposure, particularly to oxides of nitrogen or phosgene, so as to avoid acute pulmonary oedema.

(iv) Control measures

These consist primarily in the enclosure of industrial process equipment, widespread process mechanisation, capturing of gases at the point of emission, local exhaust ventilation of processes and general ventilation of premises, automatic gas-leakage or hazardous-concentration monitoring devices which actuate an alarm when a danger level is reached, etc.

It may also be necessary to provide workers with respiratory protective equipment. Exposure limits are in general established for the time-weighted average exposure and short-term peak exposures.9

4.9 Infectious and parasitic diseases of the lungs1

Exposure to live infective micro-organisms or parasites is encountered in many occupations. Although a great number of infectious diseases can have some respiratory symptoms and signs, only those which attack the lungs as the major target organ will be discussed.

(i) Potential exposure risk

Work-related infectious and parasitic diseases are mostly encountered in:

- agricultural work;
- hospitals, laboratories, clinics, autopsy rooms, etc.;
- work involving handling of animals and their products (veterinary clinics, slaughterhouses, meat and fish markets, etc.);
- outdoor work where animal excreta may be encountered (work in canals, river, ditches, sewers, docks, farmyards, construction sites, etc.).
(ii) **Effects**

Infection occurs when an unimmunised or non-resistant person comes into contact with an infective agent. In the case of lung diseases, the agent enters the respiratory system usually carried on droplets or contaminated dust.

Early signs of infections are seldom specific, but are usually sufficiently definite (mostly fever with respiratory problems) to warrant suspicion of disease if appropriate occupational factors are present. For more clinical details, reference is made to standard medical textbooks.

Table 3 summarises infectious and parasitic diseases of the lungs encountered in different occupations.

**Table 3. Summary of occupations and associated infectious (and parasitic) diseases of the lungs**

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agriculture, animal husbandry, forestry, trapping and hunting</td>
<td>Anthrax, arthropod-borne viral diseases (e.g. plague), coccidiomycosis, fungal infections, histoplasmosis, Q fever, tuberculosis</td>
</tr>
<tr>
<td>Construction work, land excavation, sewer work, ditching, mining</td>
<td>Coccidiomycosis, histoplasmosis</td>
</tr>
<tr>
<td>Meat and fish handling and packing</td>
<td>Bovine tuberculosis, fungal infections, Q fever</td>
</tr>
<tr>
<td>Poultry and bird handling</td>
<td>Fungal infections, ornithosis</td>
</tr>
<tr>
<td>Work with hair, hides, wool</td>
<td>Anthrax, Q fever</td>
</tr>
<tr>
<td>Veterinarians</td>
<td>Tuberculosis, brucellosis, fungal infections, ornithosis, Q fever</td>
</tr>
<tr>
<td>Physicians, nurses, dentists, laboratory technicians</td>
<td>Tuberculosis, other communicable infections</td>
</tr>
<tr>
<td>Work in fully air-conditioned rooms</td>
<td>Legionnaires' disease</td>
</tr>
</tbody>
</table>
(iii) **Health surveillance**

The vast majority of persons exposed to infectious and parasitic diseases are self-employed rural workers in developing countries. Since they mostly live and work in areas that are not readily accessible, health examinations are rarely, if ever, done. However, certain groups of workers in rural areas (health workers, municipal workers, construction and mining workers, etc.) can be reached more easily and regular health examinations of such workers should be encouraged.

The preplacement examinations should include a medical history and a physical examination. The main objectives of this examination are: (a) to determine and record the initial health status of the worker; (b) to identify susceptible persons; and (c) to diagnose and treat latent and active cases of infectious diseases. In occupations involving the risk of tuberculosis (e.g. health-service workers), the tuberculin test should also be done along with a chest X-ray. Depending on the geographical area and occupation, selected serological and microbiological tests may also be necessary in order to detect past or current infection. Whenever possible, all workers should be immunised against locally prevalent diseases for which vaccinations exist.

Individuals considered as being susceptible to infectious and parasitic diseases include: unimmunised persons; those recovering from serious systemic infections; those suffering from immunosuppression; and persons whose nutritional and general health status is poor.

In medical terms, the periodic examination is the same as the preplacement one. It involves the maintenance of medical records of febrile or infective illnesses by systematic inquiry and by repetition of previously conducted serological tests; in tuberculin-positive workers, a chest X-ray should be repeated. Cases of clinical disease require specific treatment.

(iv) **Control measures**

Some zoonotic diseases (transmitted from animals) can be eliminated by the control of the carrier, e.g. by immunisation or slaughtering of cattle and domestic animals.

By controlling and restricting the import of birds, domestic mammals, hides, wool and products made from animal bone it may be possible to prevent ornithosis, psittacosis (parrot fever), and anthrax. In certain workplaces, aerosol suppression by exhaust ventilation may also prevent airborne anthrax and ornithosis. Appropriate maintenance of air-conditioning systems is necessary to prevent Legionnaires' disease. Exposed workers should be informed about the nature of the hazard and the appropriate
protection and personal hygiene. Vaccination should be offered: BCG for tuberculin-negative health workers, anthrax vaccines for veterinarians, Q fever vaccines for laboratory workers.

4.10 Pneumoconioses not included in the list of occupational diseases

Dust of a number of minerals may cause respiratory disorders which are not necessarily classified as occupational diseases. The reason for this may be the benign character of the condition, its rare occurrence, or a pathology which is not clearly and universally established. If silica or asbestos are components of the dust, the resulting lung fibrosis is usually classified as silicosis or asbestosis.

4.10.1 Pneumoconioses without fibrosis

A number of pneumoconiosis result from a simple accumulation of mineral dust in the lungs which does not result in lung fibrosis or significant alteration of lung functions. When the dust contains elements which absorb X-rays, it may cause radiographic appearances similar to silicosis. The best known examples are baritosis, siderosis and stannosis.

(i) Potential exposure risk

Baritosis: mining, grinding and bagging of barite (barium sulphate), manufacturing of lithopone.

Siderosis: mining and crushing iron ores (magnetite, hematite), electric arc and oxyacetylene welding, metal polishing.

Stannosis: milling and grinding tin ore, smelting and casting of tin.

(ii) Effects

With the exception of the impressive radiographic lung image, there is no health impairment. The X-ray changes develop only after many years of employment involving exposure to barium sulphate dust, or iron and tin dust or fumes, respectively, and may gradually disappear after the end of the exposure as the dust continues to be eliminated.

If there is evidence of concomitant lung fibrosis, the effect of combined exposure to quartz is mostly supposed, in particular in work with the ore which is frequently accompanied by quartz-containing rocks. In welders, the complex effect of
welding fumes containing irritant gases (oxides of nitrogen, ozone) and metallic and mineral components of the electrodes and of the flux exist. There is increasing evidence that welding over a long period in unfavourable conditions may result in some degree of lung fibrosis.

(iii) Health surveillance

Health surveillance shall follow the same principles as for other pneumoconioses (see section 4.1).

(iv) Control measures

The control measures recommended for other pneumoconioses (section 4.1) are valid.

4.10.2 Pneumoconioses caused by silicates

Silicates are minerals that comprise in their structure other forms of silicon oxides than silicon dioxide (silica), usually in combination with other elements, in particular aluminium, magnesium, iron and calcium. The asbestos group of silicates has been discussed in section 4.1.4. Among the many other silicates in industrial use, only those of major importance will be discussed: clay, kaolin, mica, slate and talc.

(i) Potential exposure risk

Clay is a hydrous aluminium silicate; it also contains varying amounts of feldspar, mica and quartz. It is extracted in opencast or underground mines, dried, crushed and mixed, and is used in the manufacture of pottery, bricks, tiles and refractories.

Kaolin is also a hydrous aluminium silicate. Although commercial kaolins contain very little quartz, the raw material from which they are produced may contain more than 50 per cent of quartz sand. Kaolin is used for the manufacture of china, as a filler in many applications (paper, rubber, paints, plastics), and for many other purposes.

Mica is a potassium- or magnesium-containing aluminium silicate. It is mined in opencast or underground mines. Large transparent sheets are used as high-temperature resistant window panes (e.g. in stoves or furnaces) and mica powder is used in the manufacture of electric cables, pneumatic tyres, dielectric dressings, flameproof insultators, or as filler in paints and plastics, etc.
Slate is a sedimentary rock containing silicates and calcium carbonate, but also quartz. Slate is extracted in quarries and used for roofing and for other construction elements. Powdered slate is used as filler in paints and mastics.

Talc is a hydrous magnesium silicate; however, the term "talc" applies to a group of minerals which are essentially a mixture of various silicates, including asbestos (tremolite, anthophyllite, serpentines) and may contain an appreciable amount of quartz. It is obtained from opencast or underground mines and used in cosmetics and toiletry preparations, as filler in paints and ceramic products, and in the rubber industry.

(ii) Effects

Cases of pneumoconioses have been reported after long-lasting, high-level exposure to all types of the silicates. The degree of lung fibrosis reached from minimum changes up to large massive fibrosis resulting in respiratory failure.

There is no consensus as regards the cause of the fibrosis: whether pure silicates alone are fibrogenous, or whether the presence of quartz and in the case of talc also of asbestos is the decisive pathogenetic factor.

(iii) Health surveillance

Health surveillance shall follow the same principles as for other pneumoconioses (see section 4.1).

(iv) Control measures

The control measures recommended for other pneumoconioses (see section 4.1) are valid.

4.10.3 Effects of man-made mineral fibres

Man-made mineral fibres (MMMF), most of which are referred to as man-made vitreous fibres, or synthetic mineral/vitreous fibres, have been manufactured and used for over 100 years. There are different types of MMMF such as insulation wool (including glass wool, mineral wool, rock-wool and slag-wool), refractory (including ceramic) fibres, continuous filament and special-purpose fibres (figure 18).
Figure 18. Classification, methods of manufacture and nominal diameters of MMMF

<table>
<thead>
<tr>
<th>Man-made mineral fibres</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous filament</td>
</tr>
<tr>
<td>Insulation wool</td>
</tr>
<tr>
<td>Refractory fibres</td>
</tr>
<tr>
<td>Special-purpose fibres</td>
</tr>
<tr>
<td>(1) Glass</td>
</tr>
<tr>
<td>(1) Glass wool</td>
</tr>
<tr>
<td>(1) Ceramic</td>
</tr>
<tr>
<td>(1) Glass microfibres</td>
</tr>
<tr>
<td>(2) Rockwool</td>
</tr>
<tr>
<td>(2) Others</td>
</tr>
<tr>
<td>(3) Slagwool</td>
</tr>
</tbody>
</table>

Nominal fibre diameters

- Continuous: 6-15 μm
- Insulation: 2-9 μm
- Refractory: 1.2-3 μm
- Special-purpose: 0.1-3 μm

Method of manufacture

- Drawn
- Centrifuged
- Blown
- Centrifuged/blown
- Drawn/blown
- Flame attenuated

^ Approximate range of mean diameters.

The different types of MMMF have provided great benefits to society through use in homes, offices and factories for thermal insulation, energy conservation, acoustic insulation, fireproofing and fire protection, domestic appliance insulation, aerospace insulation, and as reinforcing materials in plastics, plasters, cement and textiles.

On the other hand, the exposure of workers to excessive concentrations of airborne MMMF dust has raised serious concern about possible harmful effects on their health, concern supported by results of animal experiments and some epidemiological evidence. Skin, eye and upper respiratory tract irritation in people exposed to high concentrations of MMMF has been known for decades. Only more recently has it been realised that airborne MMMF can be of such small dimension as to become respirable and be deposited in the lung tissue. Therefore, it is debatable whether MMMF might not have effects similar to those caused by excessive exposure to asbestos dust, i.e. causing lung fibrosis, bronchial cancer or mesothelioma of the pleura and peritoneum.

(i) Potential exposure risk

MMMF have a wide range of uses, which means that many people are exposed to them at work. Continuous filament glass fibres are used in the reinforcement of cement, plaster and plastic materials, in paper and rubber products, in industrial textiles and in electrical insulation. Insulation wools are used in thermal and acoustic insulation (domestic and industrial), in acoustic ceiling tiles and panels, in ventilation and air-conditioning ducts and in fireproofing and fire protection.
Refractory fibres are used in high temperature insulation; in fireproofing and fire protection; and in interpenetrations in buildings. Both insulation wools and refractory fibres are used as components in friction materials. Special-purpose glass fibres are used in high performance insulation, particularly in the aerospace industry, in high-performance acoustic protection, and in high-efficiency filtration.

(ii) **Effects**

Skin, eye, and upper respiratory tract irritation can be caused by fibres with diameters greater than about 5 μm.

No consistent pattern of MMMF-related effects on the respiratory functions has emerged, to date, from epidemiological surveys: whereas some of the studies suggest that there may be some adverse effects, others do not.

As regards cancer, evaluation of studies on both humans and experimental animals concluded that rock-wool, slag-wool and glass wool are possibly carcinogenic to humans, whereas continuous (glass) filaments are not classifiable as to their carcinogenicity to humans due to the lack of reliable data.

(iii) **Health surveillance**

Regular health surveillance should be offered to all those who are exposed to dust in the production or use of the fibres. A medical examination complemented by basic pulmonary function tests and the baseline chest X-ray should be carried out before employment. Five-year intervals between periodical examinations may be appropriate during the first ten years of work, thereafter, the examinations may be more frequent.

(iv) **Control measures**

The control measures recommended for pneumoconioses (see section 4.1) should be applied.

Exposure limits established in a number of countries vary between 3 to 8 mg per m$^3$ of total dust. However, there is a tendency of transition to fibre counts between one to three respirable fibres per ml.
Notes

1 WHO: Early detection of occupational diseases (Geneva, 1986).


3 ILO: Guidelines for the use of ILO international classification of radiographs of pneumoconiosis, Occupational Safety and Health Series No. 22 (revised) (Geneva, 1980).


11 A.T. Doig: "Barium and compounds", in ILO: Encyclopaedia of occupational health and safety, op. cit., pp. 242-244.


19 ILO: Safety in the use of mineral and synthetic fibres, Occupational Safety and Health Series No. 64 (Geneva, 1990).
APPENDIX I

EMPLOYMENT INJURY BENEFITS CONVENTION, 1964 (No. 121)

Schedule I. List of occupational diseases
(amended 1980)

<table>
<thead>
<tr>
<th>Occupational diseases</th>
<th>Work involving exposure to risk*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pneumoconioses caused by sclerogenic mineral dust (silicosis, anthraco-silicosis, asbestosis) and silico-tuberculosis, provided that silicosis is an essential factor in causing the resultant incapacity or death.</td>
<td>All work involving exposure to the risk concerned.</td>
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<tr>
<td>2. Bronchopulmonary diseases caused by hard-metal dust.</td>
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<tr>
<td>3. Bronchopulmonary diseases caused by cotton dust (byssinosis), or flax, hemp or sisal dust.</td>
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<tr>
<td>4. Occupational asthma caused by sensitising agents or irritants, both recognised in this regard and inherent in the work process.</td>
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<tr>
<td>5. Extrinsic allergic alveolitis and its sequelae caused by the inhalation of organic dusts, as prescribed by national legislation.</td>
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<tr>
<td>6. Diseases caused by beryllium or its toxic compounds.</td>
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<tr>
<td>7. Diseases caused by cadmium or its toxic compounds.</td>
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<tr>
<td>8. Diseases caused by phosphorus or its toxic compounds.</td>
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<td>9. Diseases caused by chromium or its toxic compounds.</td>
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<tr>
<td>10. Diseases caused by manganese or its toxic compounds.</td>
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</tr>
<tr>
<td>Occupational diseases</td>
<td>Work involving exposure to risk*</td>
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</tr>
<tr>
<td>11. Diseases caused by arsenic or its toxic compounds.</td>
<td>All work involving exposure to the risk concerned.</td>
</tr>
<tr>
<td>12. Diseases caused by mercury or its toxic compounds.</td>
<td>&quot;</td>
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<tr>
<td>13. Diseases caused by lead or its toxic compounds.</td>
<td>&quot;</td>
</tr>
<tr>
<td>14. Diseases caused by fluorine or its toxic compounds.</td>
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<tr>
<td>15. Diseases caused by carbon disulfide.</td>
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</tr>
<tr>
<td>16. Diseases caused by the toxic halogen derivatives of aliphatic or aromatic hydrocarbons.</td>
<td>&quot;</td>
</tr>
<tr>
<td>17. Diseases caused by benzene or its toxic homologues.</td>
<td>&quot;</td>
</tr>
<tr>
<td>18. Diseases caused by toxic nitro- and amino-derivatives of benzene or its homologues.</td>
<td>&quot;</td>
</tr>
<tr>
<td>19. Diseases caused by nitroglycerin or other nitric acid esters.</td>
<td>&quot;</td>
</tr>
<tr>
<td>20. Diseases caused by alcohols, glycols or ketones.</td>
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</tr>
<tr>
<td>21. Diseases caused by asphyxiants: carbon monoxide, hydrogen cyanide or its toxic derivatives, hydrogen sulfide.</td>
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</tr>
<tr>
<td>22. Hearing impairment caused by noise.</td>
<td>&quot;</td>
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<tr>
<td>23. Diseases caused by vibration (disorders of muscles, tendons, bones, joints, peripheral blood vessels or peripheral nerves).</td>
<td>&quot;</td>
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<tr>
<td>24. Diseases caused by work in compressed air.</td>
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</tr>
</tbody>
</table>
### Occupational diseases

<table>
<thead>
<tr>
<th>Work involving exposure to risk*</th>
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</thead>
<tbody>
<tr>
<td><strong>25. Diseases caused by ionising radiations.</strong></td>
</tr>
<tr>
<td><strong>26. Skin diseases caused by physical, chemical or biological agents not included under other items.</strong></td>
</tr>
<tr>
<td><strong>27. Primary epitheliomatous cancer of the skin caused by tar, pitch, bitumen, mineral oil, anthracene, or the compounds, products or residues of these substances.</strong></td>
</tr>
<tr>
<td><strong>28. Lung cancer or mesotheliomas caused by asbestos.</strong></td>
</tr>
<tr>
<td><strong>29. Infectious or parasitic diseases contracted in an occupation where there is a particular risk of contamination.</strong></td>
</tr>
</tbody>
</table>

* In the application of this Schedule the degree and type of exposure should be taken into account when appropriate.