Annex I 301.21,
301.22,
302,
306,
308

Asbestosis (301.21)

Mesothelioma following the inhalation of asbestos dust (301.22)

Complication of asbestos in the form of bronchial cancer (302)

Fibrotic diseases of the pleura, with respiratory restriction, caused by asbestos (306)

Lung cancer following the inhalation of asbestos dust (308)

Definition of causal agent

Asbestos is a fibrous silicate which exists in various forms:

- Serpentines: chrysotile
- Amphiboles: crocidolite, amosite, actinolite, tremolite, anthophyllite.

All these fibres are capable of causing the diseases mentioned below, although their biological activities are different.

Main occupational uses and sources of exposure:

Exposure sources and levels have evolved substantially in recent decades. In Europe, exposure levels have fallen significantly, and some types of exposure have disappeared from many European countries (extraction and handling of asbestos-bearing rock, carding, spinning and weaving the fibres, manufacture of asbestos cement, sprayed coatings, manufacture of vehicle brakes, etc). Asbestos has been used in many applications, and exposure can still occur in connection with coatings remaining in place, insulation, ovens, construction materials containing asbestos etc.

Certain working operations dealing with asbestos still in place may involve significant exposure (asbestos removal, building maintenance, dismantling/refurbishment of ships, etc.).

Asbestos Exposure Assessment

Diseases linked to asbestos exposure develop very slowly; usually several decades after exposure. Mineralogical analysis of biological samples for asbestos fibres and bodies can provide information additional to a person's work history. The presence of asbestos bodies or fibres does not prove the existence of an asbestos-related disease but, in cases of doubt, may confirm exposure to asbestos. Guidance on identifying subjects with a high probability of asbestos exposure is available from the Helsinki Consensus Report 2005 which recommends values for asbestos bodies/fibres in biological samples.

Each laboratory must establish its own reference values. The median values for occupationally exposed groups should be well above the reference values.

☐ Adverse effects

The following are distinct clinical entities associated with asbestos exposure. The presence of one does not imply the existence of other asbestos related diseases.

☐ Asbestosis (Annex I nr. 301.21)

Bilateral, diffuse, interstitial pulmonary fibrosis caused by exposure to asbestos.

Asbestosis is similar to many other fibroses and the diagnostic criteria below must be used with a history suggestive of asbestos exposure.

Diagnostic criteria:

There are no specific anatomo-pathological criteria for the diagnosis of asbestosis. The following criteria, together with a history of asbestos exposure, suggest the diagnosis of asbestosis and provide a basis for assessing its severity:

- *Symptoms and signs*: breathlessness; persistent bilateral late inspiratory basal crepitations; clubbing
- *Chest X-ray*: diffuse interstitial opacities (usually reticular or reticulonodular), mainly in the lower lung fields
- *Computerized tomography*: diffuse interstitial opacities mainly in the lower lung fields
- *Lung function tests*: restriction, reduction in gas transfer, decrease of the flow rates at low volume (flow-volume curve).

These features do not necessarily appear simultaneously, and the order in which they occur may differ from one subject to another. At present in industrialised countries, most cases of asbestosis show up only on radiological examinations without progression to respiratory insufficiency. Early disease that is only visible on CT scanning requires expert radiological assessment.

Exposure criteria:

Minimum intensity of exposure: confirmed occupational exposure, assessed by history and study of working conditions, providing evidence of prolonged and repeated heavy exposure to asbestos, and by (where feasible):

- Estimation of a cumulative exposure index from exposure times, type of occupational activity and concentrations in the air which might have been measured at the place of work. There is evidence that the risk of developing asbestosis at cumulative exposures of <25 fibres.ml^{-1}.year is low.
- significant concentrations of asbestos bodies or fibres in the sputum, fluid from bronchoalveolar lavage or lung parenchyma.

Minimum duration of exposure: 5 years. This may be shorter in the event of heavy exposure.

Maximum Latent Period: not applicable

Minimum induction period: 5 years

These are localised, usually focal, bilateral hyaline thickenings (fibrosis) of the parietal pleura; they are sometimes (partially) calcified. Their presence does not imply the existence of other asbestos related diseases. On their own they do not usually cause symptoms or deficits in lung function.

Exposure criteria:

Minimum intensity of exposure: confirmed occupational exposure, assessed by history and study of working conditions providing evidence of exposure to asbestos. This exposure may be confirmed by the presence of asbestos bodies or fibres in biological samples (sputum, fluid from bronchoalveolar lavage or lung biopsy)

Minimum duration of exposure: unknown

Maximum latent period: not applicable.

Minimum induction period: usually more than 10 years. The onset of pleural plaques is related to the time since first exposure.

☐ Other benign lung diseases

- ***Asbestos pleural effusions***

Diffuse exudative pleural reaction, with or without symptoms and often recurrent.

- ***Diffuse pleural thickening***

Diffuse thickening mainly of the visceral pleura, accompanied by parenchymal strips or atelectasis caused by twisting or deterioration of the bottom of the ipsilateral pleural sac. It often follows asbestos pleurisy. It may be accompanied by a restrictive syndrome or a decline in total lung capacity.

- ***Rounded atelectasis***

Twisting of a segment of lung parenchyma in contact with an area of visceral pleural fibrosis.

Exposure criteria

Minimum intensity of exposure: confirmed occupational exposure, if possible assessed by history and study of working conditions providing evidence of prolonged or repeated exposure to asbestos

Minimum duration of exposure: unknown

Maximum latent period: not applicable.

Minimum induction period: usually more than 10 years. With high exposures it may be less.

☐ Malignant mesotheliomas (Annex I nr. 301.22)

Primary malignant tumour of the pleura
Primary malignant tumour of the peritoneum
Primary malignant tumour of the pericardium.

80-90% of pleural mesotheliomas are attributable to occupational exposure to asbestos. Smoking does not increase the risk. The risk of mesotheliomas increases considerably in relation to time since first exposure. Exposure to amphibole asbestos fibres carries a far higher risk of mesothelioma than does chrysotile asbestos exposure.

Diagnostic Criteria

The diagnosis of mesothelioma is a pathological diagnosis. Its presence may be suggested by:

- Characteristic clinical features including chest pain, pleural effusion, breathlessness and weight loss
- Standard radiology and computed tomography
- Histological examination of biopsy specimen
- Immunocytochemistry may be helpful in distinguishing the chief differential diagnosis of secondary adenocarcinoma.

Exposure criteria:

Minimum intensity of exposure: confirmed occupational exposure, if possible assessed by history and study of working conditions providing evidence of exposure to asbestos. Some occupations (for example those involved with the refurbishment of office buildings) may incur unrecognised exposure to asbestos, in which case a history of occupational exposure may be unreliable.

Minimum duration of exposure: usually a few years but shorter exposures (as low as 3 months) have been described.

Minimum induction period: usually more than 20 years but rarely, cases associated with high exposure have been described with shorter induction periods

– See section on ***Occupational cancers*** in the **Preface**

☐ Primary bronchial cancer (Annex I nr. 302)

Asbestos may cause a primary bronchial cancer. The presence of asbestosis increases the likelihood of causal association between asbestos and primary bronchial cancer. However, asbestosis is not essential for the development of primary bronchial cancer arising from asbestos exposure.

The risk is increased considerably by smoking. Since tobacco smoke is the main risk factor for bronchial cancer, it must be considered carefully alongside workplace exposures in attributing an occupational cause.

Diagnostic Criteria:

All histological types of bronchial cancer have been linked to asbestos exposure. The diagnosis is pathological. Its presence may be suggested by:

- Characteristic clinical features including haemoptysis, cough, weight loss, and pleural effusion.
- Standard radiography and computed tomography. PET scanning may be helpful.
- Cytological examination of sputum, bronchial aspiration or bronchial lavage
- Histological examination of biopsy specimen

Exposure criteria:

Minimum intensity of exposure: confirmed occupational exposure, assessed by history and study of working conditions, providing evidence of prolonged and repeated heavy exposure to asbestos, and by (where feasible):

- Estimation of a cumulative exposure index from exposure times, type of occupational activity and concentrations in the air which might have been measured at the place of work.

There is evidence that the risk of developing bronchial cancer at cumulative exposures of <25 fibres.ml^{-1}.year is low.

- significant concentrations of asbestos bodies or fibres in the sputum, fluid from bronchoalveolar lavage or lung parenchyma.
- the presence of asbestosis (the presence of pleural plaques suggests exposure to asbestos but does not reflect the exposure level).

Minimum duration of exposure: usually a few years.

Minimum induction period: usually more than 15 years.

See section on ***Occupational cancers*** in the **Preface**.

☐ Asbestos warts

Pronounced thickening and hyperkeratosis on the dorsal and palmar surfaces of the hands and forearms caused by minute asbestos fibres penetrating the skin. A cure can be effected by removing the fibres.

Exposure criteria:

Minimum intensity of exposure: occupational exposure confirmed by history, evidence of which is provided by the subcutaneous presence of asbestos fibres. A single contact is enough for fibres to penetrate the skin.