Definition of work – related diseases and occupational diseases

The Godfather of Occupational Diseases Bernard Ramazzini (1633-1714):

“When you come to a patient’s house, you should ask him what sort of pains he has, what caused them, how many days he has been ill, whether the bowels are working and what sort of food he eats. So says Hippocrates.

I may venture to add one more question: what occupation does he follow?”

Ramazzini wrote De Morbis Artificum Diatriba (Diseases of Workers), the first comprehensive work on occupational diseases, outlining the health hazards of irritating chemicals, dust, metals, and other abrasive agents encountered by workers in 52 occupations.

Figure 1 Bernard Ramazzini
http://www.corbisimages.com/images/Corbis-PG4631.jpg?size=67&uid=7cfc2775-02d3-4f03-8a98-4ab5269c822a

Occupational diseases are illnesses that are associated with a particular occupation or industry. They result from chemical, physical, biological or psychological hazards or factors in the work environment and although they may occur due to other causes, are not a special risk in the workplace if there is no such exposure. These diseases are preventable if the hazardous exposure is eliminated or reduced to a level that protects workers. The pattern of occupational disorders is constantly changing due to changes in the economy, in industrial processes, and in the workforce. However, these changing patterns are often overlooked because work-related diseases are rarely monitored. Occupational diseases often go unrecognized, known occupational diseases are consistently underreported in every country and new occupational diseases are introduced from by changing technology. The list of
notifiable occupational conditions recognised at any one time by governments, insurance companies, or compensation agencies is never complete.

In general, occupational diseases cost more than injuries to workers and to the economy because they tend to be chronic and often lead to permanent disability. Occupational diseases, therefore, produce a significant cost burden for workers in medical expenses, loss of earnings, reduced life expectancy, physical or psychological suffering, loss of future earnings, and financial and social difficulties. Employers also face costs in compensation for lost earnings, disability and death, medical costs, time lost, decreased productivity, decreased employee morale, and unfavourable public relations. The prevention and control of occupational hazards decreases the risk of developing both occupational and work-related diseases.

For workers, occupational diseases may be catastrophic, resulting in loss of income for the family, expenses for medical care, and severe disability that may make it impossible to hold a well-paying job in the future. Some occupational diseases lead to early death, and most lead to significant impairments in daily life.

In the third edition of the ILO’s (International Labour Organization) Encyclopedia of Occupational Health and Safety, a distinction was made among the pathological conditions that could affect workers in which diseases due to occupation (occupational diseases) and diseases aggravated by work or having a higher incidence owing to conditions of work (work-related diseases) were separated from conditions having no connection with work. In some countries work-related diseases are treated the same as work-caused diseases, which are in fact occupational diseases.

According to OSHA (Occupational Health and Safety Administration) an occupational illness (or disease) is, "any abnormal condition or disorder, other than one resulting from an occupational injury, caused by exposure to factors associated with employment."

Nevertheless, it is not always that easy to designate a disease as being work-related. In fact, there is a wide range of diseases that could be related in one way or another to occupation or working conditions. On the one hand, there are the classical diseases that are occupational in nature, generally related to one causal agent and relatively easy to identify. On the other hand, there are all sorts of disorders without strong or specific connections to occupation and with numerous possible causal agents. Many of these diseases with a multifactorial aetiology may be work-related only under certain conditions. The relationship between work and disease could be identified in the following categories:
- *occupational diseases*, having a specific or a strong relation to occupation, generally with only one causal agent, and recognised as such. The risk to workers in a certain occupation is substantially greater than the risk to the general population.

- *work-related diseases*, with multiple causal agents, where factors in the work environment may play a role, together with other risk factors, in the development of such diseases, which have a complex aetiology.

**Recognition of occupational diseases**

„Legal“occupational diseases are those that are included in the valid national list of occupational diseases in particular country, are compensable and recordable. The causal relationship to a particular workplace is established on the basis of clinical data, occupational history and identification of occupational risk factors (chemicals, biological agents, physical factors, adverse ergonomic conditions, allergens and psychosocial risk factors). After the completion and confirmation of clinical diagnosis an objective evidence of a hazardous workplace has to be provided. This complex assessment is completed by the public health authorities (occupational or public health physicians, occupational health specialists). In connection to the assessment and confirmation of occupational disease, irrespective of the particular diagnosis, only occupational physicians (e.g. in Slovak Republic, Czech Republic) are allowed to provide and complete whole final process of recognition including the notification of the disease and a proposal for compensation. The occupational diseases notification is sent to the patient, employer, occupational health service, public health authority, the National Registry of Occupational Diseases and Health Insurance Company, where every employer must be insured for the case of occupational disease development. According to the Slovak legislation, the last employer for whom the patient worked under risky occupational conditions is responsible for the development of an occupational disease and therefore is obliged to cover compensations.

**Taking the occupational history**

Occupational history is the cornerstone for early diagnosis of occupational disease. An occupational history is a chronological list of all the patient’s employment, with dates, expanded as necessary to detail any evidence of occupational exposure to potentially hazardous agents.
The occupational history or exposure history seeks to define possible exposure to hazards to health and links with actual ill-health outcomes. The hazard must be confirmed by public health authorities:

Figure 2 The occupational and exposure history
www.fom.ac.uk/wp-content/uploads/comppgm4.ppt

The patient with a possibly work-related illness frequently seeks care initially from a family physician. The physician's recognition of a possible link between work and disease often determines the diagnostic tests that are performed and the treatment that is recommended. Early diagnosis of an occupational illness may prevent progressive morbidity and disability from conditions such as occupational asthma and may facilitate the reversal of adverse effects from exposures to substances such as lead. The family physician is in a crucial position to contribute new information about occupational disease. History taking is a time consuming complex which involve more than merely recording the job title. What is necessary is to obtain information on what the patient does at work. The key questions are:

- do you have a job?
- what do you do at work?
- do you have any problems at work?
- do you think that your work is affecting your health?
- what kind of work do you do?
- how long?
- what exposures are involved?
- what personal protective equipment do you use?
- list all other jobs in chronologic order?
- military exposures?
- symptoms or illness related to work?
- timing of symptoms?
- other workers have symptoms?
- non occupational exposures-smoking, alcohol, geographic history, family exposure, hobbies?
- ventilation at work?
- preemployment examination?
- days missed from work and reason?
- secondary jobs?
- past history-noise, chemicals, heat, vibration, asbestos, radiation, toxic agents, etc?
- animals in or around the workplace?
- how many hours a week do you work?
- how many years have you worked at what jobs?
Finally, it is often sensible to ask:
- do you have any other jobs?
Questions about hobbies are very relevant but very often provide no reliable information. It is important to know how long the patient has had the job. If it has been for a relatively short period, it is sensible to ask about the main work the patient had carried out previously. In the case, when a patient presents with a chronic disease or with a malignancy, it may be useful to know about jobs many years before.

**Specific disorders when you should probe more precise:**

anaemia
asthma, acute bronchitis, lung inflammation
chronic lung disease
pneumoconiosis (dust in lungs)
dermatitis
headaches
hepatitis
injury
nausea and vomiting of unknown origin
new onset of depression/irritability
neuropathy
kidney failure
reproductive anomalies

1. **Occupational diseases caused by exposure to agents arising from work activities**

   1.1. *Diseases caused by chemical agents*

      1.1.1. Diseases caused by beryllium or its compounds
      1.1.2. Diseases caused by cadmium or its compounds
      1.1.3. Diseases caused by phosphorus or its compounds
      1.1.4. Diseases caused by chromium or its compounds
      1.1.5. Diseases caused by manganese or its compounds
      1.1.6. Diseases caused by arsenic or its compounds
      1.1.7. Diseases caused by mercury or its compounds
      1.1.8. Diseases caused by lead or its compounds
      1.1.9. Diseases caused by fluorine or its compounds
      1.1.10. Diseases caused by carbon disulfide
      1.1.11. Diseases caused by halogen derivatives of aliphatic or aromatic hydrocarbons
      1.1.12. Diseases caused by benzene or its homologues
      1.1.13. Diseases caused by nitro- and amino-derivatives of benzene or its homologues
      1.1.14. Diseases caused by nitroglycerine or other nitric acid esters
      1.1.15. Diseases caused by alcohols, glycols or ketones
      1.1.16. Diseases caused by asphyxiants like carbon monoxide, hydrogen sulfide, hydrogen cyanide or its derivatives
      1.1.17. Diseases caused by acrylonitrile
      1.1.18. Diseases caused by oxides of nitrogen
      1.1.19. Diseases caused by vanadium or its compounds
      1.1.20. Diseases caused by antimony or its compounds
      1.1.21. Diseases caused by hexane
      1.1.22. Diseases caused by mineral acids
      1.1.23. Diseases caused by pharmaceutical agents
      1.1.24. Diseases caused by nickel or its compounds
      1.1.25. Diseases caused by thallium or its compounds
      1.1.26. Diseases caused by osmium or its compounds
      1.1.27. Diseases caused by selenium or its compounds
      1.1.28. Diseases caused by copper or its compounds
1.1.29. Diseases caused by platinum or its compounds
1.1.30. Diseases caused by tin or its compounds
1.1.31. Diseases caused by zinc or its compounds
1.1.32. Diseases caused by phosgene
1.1.33. Diseases caused by corneal irritants like benzoquinone
1.1.34. Diseases caused by ammonia
1.1.35. Diseases caused by isocyanates
1.1.36. Diseases caused by pesticides
1.1.37. Diseases caused by sulphur oxides
1.1.38. Diseases caused by organic solvents
1.1.39. Diseases caused by latex or latex-containing products
1.1.40. Diseases caused by chlorine
1.1.41. Diseases caused by other chemical agents at work not mentioned in the preceding items where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to these chemical agents arising from work activities and the disease(s) contracted by the worker

1.2. Diseases caused by physical agents
1.2.1. Hearing impairment caused by noise
1.2.2. Diseases caused by vibration (disorders of muscles, tendons, bones, joints, peripheral blood vessels or peripheral nerves)
1.2.3. Diseases caused by compressed or decompressed air
1.2.4. Diseases caused by ionising radiations
1.2.5. Diseases caused by optical (ultraviolet, visible light, infrared) radiations including laser
1.2.6. Diseases caused by exposure to extreme temperatures
1.2.7. Diseases caused by other physical agents at work not mentioned in the preceding items where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to these physical agents arising from work activities and the disease(s) contracted by the worker

1.3. Biological agents and infectious or parasitic diseases
1.3.1. Brucellosis
1.3.2. Hepatitis viruses
1.3.3. Human immunodeficiency virus (HIV)
1.3.4. Tetanus
1.3.5. Tuberculosis
1.3.6. Toxic or inflammatory syndromes associated with bacterial or fungal contaminants
1.3.7. Anthrax
1.3.8. Leptospirosis
1.3.9. Diseases caused by other biological agents at work not mentioned in the preceding items where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to these biological agents arising from work activities and the disease(s) contracted by the worker

2. Occupational diseases by target organ systems

2.1. Respiratory diseases
2.1.1. Pneumoconioses caused by fibrogenic mineral dust (silicosis, anthraco-silicosis, asbestosis)
2.1.2. Silicotuberculosis
2.1.3. Pneumoconioses caused by non-fibrogenic mineral dust
2.1.4. Siderosis
2.1.5. Bronchopulmonary diseases caused by hard-metal dust
2.1.6. Bronchopulmonary diseases caused by dust of cotton (byssinosis), flax, hemp, sisal or sugar cane (bagassosis)
2.1.7. Asthma caused by recognised sensitising agents or irritants inherent to the work process
2.1.8. Extrinsic allergic alveolitis caused by the inhalation of organic dusts or microbially contaminated aerosols, arising from work activities
2.1.9. Chronic obstructive pulmonary diseases caused by inhalation of coal dust, dust from stone quarries, wood dust, dust from cereals and agricultural work, dust in animal stables, dust from textiles, and paper dust, arising from work activities
2.1.10. Diseases of the lung caused by aluminium
2.1.11. Upper airways disorders caused by recognized sensitising agents or irritants inherent to the work process
2.1.12. Other respiratory diseases not mentioned in the preceding items where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to risk factors arising from work activities and the disease(s) contracted by the worker

2.2. Skin diseases
2.2.1. Allergic contact dermatoses and contact urticaria caused by other recognized allergy-provoking agents arising from work activities not included in other items
2.2.2. Irritant contact dermatoses caused by other recognized irritant agents arising from work activities not included in other items
2.2.3. Vitiligo caused by other recognized agents arising from work activities not included in other items
2.2.4. Other skin diseases caused by physical, chemical or biological agents at work not included under other items where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to risk factors arising from work activities and the skin disease(s) contracted by the worker

2.3. Musculoskeletal disorders
2.3.1. Radial styloid tenosynovitis due to repetitive movements, forceful exertions and extreme postures of the wrist
2.3.2. Chronic tenosynovitis of hand and wrist due to repetitive movements, forceful exertions and extreme postures of the wrist
2.3.3. Olecranon bursitis due to prolonged pressure of the elbow region
2.3.4. Prepatellar bursitis due to prolonged stay in kneeling position
2.3.5. Epicondylitis due to repetitive forceful work
2.3.6. Meniscus lesions following extended periods of work in a kneeling or squatting position
2.3.7. Carpal tunnel syndrome due to extended periods of repetitive forceful work, work involving vibration, extreme postures of the wrist, or a combination of the three
2.3.8. Other musculoskeletal disorders not mentioned in the preceding items where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to risk factors arising from work activities and the musculoskeletal disorder(s) contracted by the worker

2.4. Mental and behavioural disorders
2.4.1. Post-traumatic stress disorder
2.4.2. Other mental or behavioural disorders not mentioned in the preceding item where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to risk factors arising from work activities and the mental and behavioural disorder(s) contracted by the worker.

3. Occupational cancer
3.1. Cancer caused by the following agents
3.1.1. Asbestos
3.1.2. Benzidine and its salts
3.1.3. Bis-chloromethyl ether (BCME)
3.1.4. Chromium VI compounds
3.1.5. Coal tars, coal tar pitches or soots
3.1.6. Beta-naphthylamine
3.1.7. Vinyl chloride
3.1.8. Benzene
3.1.9. Toxic nitro- and amino-derivatives of benzene or its homologues
3.1.10. Ionising radiations
3.1.11. Tar, pitch, bitumen, mineral oil, anthracene, or the compounds, products or residues of these substances
3.1.12. Coke oven emissions
3.1.13. Nickel compounds
3.1.14. Wood dust
3.1.15. Arsenic and its compounds
3.1.16. Beryllium and its compounds
3.1.17. Cadmium and its compounds
3.1.18. Erionite
3.1.19. Ethylene oxide
3.1.20. Hepatitis B virus (HBV) and hepatitis C virus (HCV)
3.1.21. Cancers caused by other agents at work not mentioned in the preceding items where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure to these agents arising from work activities and the cancer(s) contracted by the worker

4. Other diseases
4.1. Miners’ nystagmus
4.2. Other specific diseases caused by occupations or processes not mentioned in this list where a direct link is established scientifically, or determined by methods appropriate to national conditions and practice, between the exposure arising from work activities and the disease(s) contracted by the worker.

References:


As in the previous years, the diseases of bones, joints, tendons and nerves caused by long-term, excessive and unilateral exertion of limbs represented the largest proportion of occupational diseases (144 cases out of all 301 reported cases). The second most commonly reported occupational disease was a disease of bones, joints, muscles, and nerves of the limbs caused by vibrating tools usage (58 cases).

The severity of the persisting problem of high proportion of occupational disease incidence affecting the musculoskeletal, vascular and nervous system among workers exposed to long-term, unilateral exertion of limbs and harmful effects of vibration underlines the fact that they accounted for 66.1% out of the total number of occupational diseases. We can thus observe an upward trend in comparison with 2012 (63.1%).

The third most common occupational disease, apart from musculoskeletal disorders, was hearing loss from noise (33 reported cases).
Figure 4 Trend in number of professional diseases of limbs from longterm, inordinate, one-sided workload

Figure 5 Trend in number of diseases of bones, joints, muscles, vessels and nerves of limbs caused by work with vibrating tools and devices
List of Occupational Diseases and Threats of Occupational Diseases
(National Health Information Centre (NHIC) 2014

1 Diseases from lead and its alloys and compounds
1-1 Poisonings with lead and its inorganic compounds and alloys
1-2 Poisonings with organic compounds of lead

2 Diseases from phosphor and its compounds
2-1 Poisonings with phosphor and its inorganic compounds
2-2 Poisonings with organic compounds of phosphor

3 Diseases from fluorine and its compounds
3-1 Poisonings with fluorine and its compounds
3-2 Fluorosis

4 Diseases from mercury and from its amalgams and compounds
4-1 Acute and chronic poisoning with mercury
4-2 Acute and chronic poisonings with compounds of mercury

5 Diseases from arsenic and its compounds

6 Diseases from manganese and its compounds

7 Diseases from cadmium and its compounds

8 Diseases from vanadium and its compounds

9 Diseases from chrome and its compounds
9-1 Harm of nasal partition from chrome
9-2 Other injuries from chrome

10 Diseases from carbon sulphide

11 Diseases from hydrogen sulphide

12 Diseases from carbon dioxide
12-1 Acute poisonings with carbon dioxide
12-2 Chronic poisonings and conditions after poisoning with carbon dioxide
12-3 Poisonings with explosion's combustion residues

13 Diseases from cyan compounds
13-1 Poisonings with hydrocyanic and its compounds
13-2 Poisonings with calcium cyanamid

14 Diseases from benzene and its homologies
14-1 Acute and chronic poisonings with benzene
14-2 Acute and chronic poisonings with toluene
14-3 Acute and chronic poisonings with xylene
14-4  Acute and chronic poisonings with homologies of benzene
15  Illnesses from nitro- and amino compounds of benzene or its homologies
16  Diseases from halogenated carbohydrates
17  Diseases from nitrate esters of glycerin
18  Diseases from warfare agents or chemical materials with same impact look like warfare agents
19  Diseases from ionising radiation and from radiation with similar biological effect
19-1  Harm of blood-forming from ionising radiation
19-2  Radio- and rtg dermatitis
19-3  Skin cancer from ionising radiation
19-4  Cataract from ionising radiation
20  Diseases from electromagnetic radiation including laser
21  Diseases on skin cancer
22  Skin diseases apart from skin cancer and communicable skin diseases
22-1  Professional dermatoses from alkalies
22-2  Professional dermatoses from cement
22-3  Professional dermatoses from organic and inorganic acids
22-4  Professional dermatoses from detergents
22-5  Professional dermatoses from organic solvents
22-6  Professional dermatoses from mineral oil products (oils from mineral oil)
22-7  Professional dermatoses from chrome and its compounds
22-8  Professional dermatoses from nickel and its alloys
22-9  Professional dermatoses from metals and metalloids and their compounds
22-10  Professional dermatoses from synthetic materials
22-11  Professional dermatoses from gum and gum processing chemicals
22-12  Professional dermatoses from tar and its derivatives
22-13  Professional dermatoses from of organic dye stuffs
22-14  Professional dermatoses from remedies
22-15  Professional dermatoses from disinfectants
22-16  Professional dermatoses from insecticideous substances and agrochemicals
22-17  Professional dermatoses from other chemicals (organic and inorganic)
22-18  Professional dermatoses from plants
22-19  Professional dermatoses from other biological factors
22-20  Professional dermatoses from physical factors (except of ionising radiation)
Diseases on pulmonary cancer from radioactive substances
Diseases on communicable and parasitic illnesses apart from tropical communicable and parasitic diseases and illnesses communicable from animals on people
Tropical communicable and parasitic diseases
Illnesses communicable from animals on people directly or by means of communicants
Diseases evoked with work in compressed air
Acute form of Kesón’s illness
Chronic form of Kesón’s illness
Diseases of bones, joints, muscles, vessels and nerves limbs caused at work with vibrating tools and devices
Injuries from vibrations mostly of vessels and nerves
Injuries from vibrations mostly of bends, of bones, of tendons and muscles
Other injuries from vibrations and combined injuries from vibrations
Diseases of bones, joints, tendons and nerves of limbs from longterm, inordinate, one-sided workload
Illnesses of lubrication sacs from still lasting local pressure
Illnesses of tendons, tendonous sheath and muscle insertions from inordinate overloading
Impairment of meniscuses
Diseases of peripheral nerves of limbs
Diseases of elbow nerve from mechanical influences
Diseases of lower respiratory tract and lung caused with aluminium dust from aluminium alloys (dusting of lung with aluminium – fibrosis of lung)
Diseases from beryllium and its compounds
Diseases on dusting of lung with dust containing silicon oxide (silicosis, silicotuberculosis) including (miner pneumoconiosis):
  a) with typical x-ray signs take account on dynamics of diseases,
  b) in connection with active tuberculosis
Silicosis simple
Silicosis complicated
Silicotuberculosis
Miner pneumoconiosis
Diseases on dusting of lung with asbestos dust (Asbestosis):
  a) with typical x-ray signs,
b) in connection with lungs cancer

34-1 Asbestosis
34-2 Asbestosis with pulmonary cancer
35 Diseases at production of heavy metals
36 Diseases of lower respiratory tract and lung on harmful effects of Thomas’s flour
37 Asthma bronchiale
37-1 Asthma bronchiale – sensitivity on flour, mill dust
37-2 Asthma bronchiale – sensitivity on straw, hay
37-3 Asthma bronchiale – sensitivity on dust from cereals, agricultural plants
37-4 Asthma bronchiale – sensitivity on pelt of animals
37-5 Asthma bronchiale – sensitivity on colophonic evaporations
37-6 Asthma bronchiale – sensitivity on disinfectants
37-7 Other causes of accruement of asthma bronchiale
38 Hearing defect from noise by which is reached loss hearing according to Fowler with harm younger as 30 years at least 40 %. Harm older as 30 years then presented level is increased by 1 % each two years till 50 years age of harm person and since that time loss hearing must exceeded 50 %
39 Diseases on cataract
40 Diseases on nystagmus
41 Bloating of lung with of glassblowers and musicians on wind instruments
42-1 Heavy hyperkinetical dysphony, lumps on vocal chords or heavy nonclosing of vocal chords, which are permanent and which forbid a performance of occupation which takes increased requirements on voice
42-2 Heavy phonasthenia
43 Bronchopulmonar diseases caused with dust from cotton, flax, hank or sisal
44 Outside allergic alveolitidis and their consequences caused with breathing in of organic dusts of type of farmer’s lung
45 Allergic diseases of upper respiratory tract with proven susceptibility on allergens from the working environment of the patient
46 Tumour diseases emergent due to work with settled chemical carcinogens in damaged working environment and demonstrative in particular targeted organs, which are not involved in this list
47 Other harms of health from work. It is dealt of damaged health from work which is not occupational diseases and also occupational disease involved in this list
Reference

Occurrence and type of health problem:
In 2007, persons aged 15 to 64 years that work or worked previously were asked whether they suffered from one or more health problems caused or made worse by work in the past 12 months. In total, 8.6% of the respondents in the EU27 had a work-related health problem. This corresponds to approximately 23 million persons in the EU27. In total, 2.1% of these persons had two or more work-related health problems.

About 60% of the respondents with work-related health problems identified musculoskeletal problems as their most serious work-related health problem (Fig. 29, Fig. 30, Table 3)

Table 1 Type of work-related health problems indicated as the most serious among persons with a work-related health problem in the EU27 (%)

<table>
<thead>
<tr>
<th>Type of work-related health problem</th>
<th>Persons that work, or worked previously</th>
<th>Persons that work</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone, joint or muscle problem which mainly affects back</td>
<td>28.4</td>
<td>20.5</td>
</tr>
<tr>
<td>Bone, joint or muscle problem which mainly affects neck, shoulders, arms or hands</td>
<td>18.8</td>
<td>20.1</td>
</tr>
<tr>
<td>Stress, depression or anxiety</td>
<td>13.7</td>
<td>14.5</td>
</tr>
<tr>
<td>Bone, joint or muscle problem which mainly affects hips, legs or feet</td>
<td>12.5</td>
<td>11.3</td>
</tr>
<tr>
<td>Breathing or lung problem</td>
<td>5.2</td>
<td>4.8</td>
</tr>
<tr>
<td>Heart disease or attack, or other problems in the circulatory system</td>
<td>5.0</td>
<td>3.8</td>
</tr>
<tr>
<td>Headache and/or eyestrain</td>
<td>4.4</td>
<td>4.9</td>
</tr>
<tr>
<td>Infectious disease (virus, bacteria or other type of infection)</td>
<td>2.5</td>
<td>3.1</td>
</tr>
<tr>
<td>Hearing problem</td>
<td>1.4</td>
<td>1.3</td>
</tr>
<tr>
<td>Skin problem</td>
<td>1.3</td>
<td>1.4</td>
</tr>
<tr>
<td>Other types of complaint</td>
<td>5.8</td>
<td>5.3</td>
</tr>
</tbody>
</table>
Figure 6 Relative occurrence of different health problems as the most serious work related health problem in the past 12 months in employed persons in the EU27

Figure 7 Employed persons with one or more work-related health problems in the past 12 months in different sectors in the EU27 (%) (LFS: Labour Force Survey; EWCS: European Survey on Working Conditions)

The diseases most prominently registered between 2001 and 2007 in EU:
- Carpal tunnel syndrome
- Diseases due to overstraining of the muscular and tendinous insertions
- Hypoacousis or deafness caused by noise
- Diseases due to overstraining of the tendon sheaths
- Occupational skin ailments caused by scientifically recognised allergy provoking or irritative substances not included under other headings
- Pleural fibrosis, with respiratory restriction, caused by asbestos
- Mesothelioma following the inhalation of asbestos dust
- Chronic obstructive bronchitis or emphysema in miners working in underground coal mines
- Angioneurotic diseases caused by mechanical vibration
- Asbestosis

Reference:
## COMMON HEALTH CONDITIONS ASSOCIATED WITH OCCUPATIONAL EXPOSURE

Igor Bátor

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Causal agent</th>
<th>Occupations/exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Musculoskeletal</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carpal tunnel syndrome</td>
<td>Repetitive wrist motions</td>
<td>Letter and parcels sorting, meat cutting</td>
</tr>
<tr>
<td></td>
<td>Hand-arm vibration</td>
<td>Drilling, sawing</td>
</tr>
<tr>
<td></td>
<td>Awkward postures</td>
<td>Computer work</td>
</tr>
<tr>
<td></td>
<td>Cold temperature</td>
<td>Food processing</td>
</tr>
<tr>
<td>Tennis, golf elbow</td>
<td>Repetitive stress of forearm muscles</td>
<td>Computer work, driving</td>
</tr>
<tr>
<td>De Quervain’s tendonitis</td>
<td>Repetitive hand twisting and forceful gripping</td>
<td>Meatpacking, manufacturing</td>
</tr>
<tr>
<td>Cervical strain</td>
<td>Static posture</td>
<td>Computer work</td>
</tr>
<tr>
<td>Low back pain</td>
<td>Lifting and handling of loads, awkward postures, whole body vibration</td>
<td>Construction, agriculture, office work, truck driving, teaching and nursing</td>
</tr>
<tr>
<td>Articular pain</td>
<td>Atmospheric decompression, Bacteria Borellia burgdorferi</td>
<td>Professional divers, forestry work</td>
</tr>
<tr>
<td>Impingement syndrome</td>
<td>Overhead activities, repetitive impingement of subacromial structures</td>
<td>Slaughter house work, painting, decorating, nursing, fruit picking</td>
</tr>
<tr>
<td>Thoracic outlet syndrome</td>
<td>Static posture, repetition</td>
<td>Assembly work, carrying loads on the shoulder</td>
</tr>
<tr>
<td><strong>Respiratory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intestinal fibrosis</td>
<td>Silica</td>
<td>Mining, tunnelling, foundry work</td>
</tr>
<tr>
<td></td>
<td>Asbestos</td>
<td>Mining, building maintenance</td>
</tr>
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<td></td>
<td>Coal</td>
<td>Mining</td>
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<tr>
<td>Conditions</td>
<td>Causal agent</td>
<td>Occupations/exposure</td>
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<tr>
<td><strong>Asthma/rhinitis</strong></td>
<td>Animal products</td>
<td>Laboratory work</td>
</tr>
<tr>
<td></td>
<td>Plant products</td>
<td>Baking</td>
</tr>
<tr>
<td></td>
<td>Plant pollens</td>
<td>Gardering</td>
</tr>
<tr>
<td></td>
<td>Wood dust</td>
<td>Furniture making, wood working</td>
</tr>
<tr>
<td></td>
<td>Isocyanates</td>
<td>Plastics manufacturing, building insulation, autobody repair</td>
</tr>
<tr>
<td></td>
<td>Perfumes, dyes</td>
<td>Hairdressing</td>
</tr>
<tr>
<td></td>
<td>Heavy metals</td>
<td>Metal refining</td>
</tr>
<tr>
<td></td>
<td>Irritants (e.g. sulfur dioxide, smoke, fumes)</td>
<td>Various occupations</td>
</tr>
<tr>
<td><strong>Bronchitis</strong></td>
<td>Smoke</td>
<td>Fire fighting</td>
</tr>
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<td></td>
<td>Coal</td>
<td>Mining, tunnelling</td>
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<td></td>
<td>Nitrogen oxides</td>
<td>Welding</td>
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<td></td>
<td>Acids</td>
<td>Planting</td>
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<tr>
<td><strong>Hypersensitivity pneumonitis</strong></td>
<td>Moldy hay</td>
<td>Farming</td>
</tr>
<tr>
<td>(farmer’s lung)</td>
<td>Indoor air pollution (bacteria, mold, pollen, viruses; i.e. Sick building syndrome)</td>
<td>Office work</td>
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<td></td>
<td>Sulphur dioxide</td>
<td>Combustion</td>
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<td></td>
<td>Nitrogen dioxide, Ozone</td>
<td>Arc welding</td>
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<td></td>
<td>Ammonia, Chlorine</td>
<td>Industrial accidents</td>
</tr>
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<td>Fluorides</td>
<td>Soldering</td>
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<tr>
<td><strong>Neurologic</strong></td>
<td></td>
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<tr>
<td>Chronic encephalopathy</td>
<td>Organic solvents</td>
<td>Painting, autobody repair</td>
</tr>
<tr>
<td>(memory impairment)</td>
<td>Lead</td>
<td>Bridge work, painting, metal recycling</td>
</tr>
<tr>
<td></td>
<td>Carbon disulfide</td>
<td>Viscose rayon work</td>
</tr>
<tr>
<td>Neurobehavioral disorders</td>
<td>Manganese</td>
<td>Welding, mining, ore crushing, dry cell batteries manufacturing</td>
</tr>
<tr>
<td>Peripheral polyneuropathy</td>
<td>Organophosphate pesticides</td>
<td>Pesticide application</td>
</tr>
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<td></td>
<td>Methyl butyl ketone</td>
<td>Fabric coating</td>
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<td></td>
<td>Ethylene oxide</td>
<td>Manufacture of mono ethylene glycol</td>
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<tr>
<td>Conditions</td>
<td>Causal agent</td>
<td>Occupations/exposure</td>
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<tr>
<td>Hexane</td>
<td>glues, cleaning fluids</td>
<td>sterilisation</td>
</tr>
<tr>
<td>Mercury</td>
<td>Amalgamation industry, dentistry,</td>
<td></td>
</tr>
<tr>
<td>Lead</td>
<td>Bridge work, painting, metal recycling</td>
<td></td>
</tr>
<tr>
<td>Hearing loss</td>
<td>Noise</td>
<td>Many occupations</td>
</tr>
<tr>
<td>Vertigo</td>
<td>Carbon monoxide, Organic solvents</td>
<td>Garage personnel, fire fighters</td>
</tr>
<tr>
<td><strong>Infectious</strong></td>
<td></td>
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<tr>
<td>Bloodborne infections</td>
<td>HIV, hepatitis B and C, syphilis</td>
<td>Health care work, prison work</td>
</tr>
<tr>
<td>Airborne infections</td>
<td>Tuberculosis, Chicken pox, Measles,</td>
<td>Health care work, prison work</td>
</tr>
<tr>
<td>Infections transmitted faecally/orally</td>
<td>Hepatitis A</td>
<td>Health care work, animal care, laundry work, sewage work</td>
</tr>
<tr>
<td>Zoonoses</td>
<td>Lyme diseases</td>
<td>Forestry and other outdoor work</td>
</tr>
<tr>
<td></td>
<td>Brucellosis, Tetanus, Q-fever, Leptospirosis, Tularaemia, Psittacosis, Ornithosis</td>
<td>Animal husbandry</td>
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<tr>
<td><strong>Cancer</strong></td>
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<td>Lung</td>
<td>Asbestos</td>
<td>Construction trades</td>
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<td></td>
<td>Chromium</td>
<td>Welding, planting</td>
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<td></td>
<td>Coal tar, pitch</td>
<td>Steelworking</td>
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<td></td>
<td>Silica</td>
<td>Mining, tunnelling, foundry work processing or handling of wood (cutting, sanding, or milling)</td>
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<td>Wood dust</td>
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<tr>
<td>Liver</td>
<td>Vinyl chloride</td>
<td>Plastic manufacturing</td>
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<td></td>
<td>Chlorinated hydrocarbon solvents</td>
<td>Many occupations</td>
</tr>
<tr>
<td>Bladder</td>
<td>Benzidine</td>
<td>Plastic and chemical manufacturing</td>
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<td></td>
<td>Polycyclic aromatic hydrocarbons (PAH)</td>
<td>Combustion processes</td>
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<td>Diesel engine exhaust</td>
<td>Many occupations</td>
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<td>Conditions</td>
<td>Causal agent</td>
<td>Occupations/exposure</td>
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<td><strong>Skin</strong></td>
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<td>Contact dermatitis</td>
<td>Organic solvents</td>
<td>Many occupations</td>
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<td></td>
<td>Epoxy resin</td>
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<td></td>
<td>Nickel</td>
<td>Hairdressing</td>
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<td>Latex</td>
<td>Health care work</td>
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<td>Hyperpigmentation</td>
<td>Arsenic</td>
<td>Coal burning</td>
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<td>Hyperkeratosis</td>
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<td><strong>Reproductive</strong></td>
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<td>Spontaneous abortion</td>
<td>Ethylene oxide</td>
<td>Manufacture of monoethylene glycol, sterilisation</td>
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<tr>
<td>Sperm abnormalities</td>
<td>Dibromochloropropane</td>
<td>Pesticide manufacturing</td>
</tr>
<tr>
<td>Birth defects</td>
<td>Ionising radiation</td>
<td>Radiographic technicians</td>
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<tr>
<td>Developmental abnormalities</td>
<td>Ionising radiation</td>
<td>Radiographic technicians</td>
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<td></td>
<td>Lead</td>
<td>Bridge work, painting, metal recycling</td>
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<tr>
<td>Erectile dysfunction</td>
<td>Carbon disulfide</td>
<td>Viscose rayon work</td>
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<td><strong>Kidney</strong></td>
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<td>Tubulointerstitial nephropathy</td>
<td>Cadmium</td>
<td>Pigment factory, construction industry (cutting, brazing, burning, grinding, welding)</td>
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<td>Lead</td>
<td>Bridge work, painting, metal recycling</td>
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<td></td>
<td>Mercury</td>
<td>Temperature and pressure measuring instruments, manufacture of fluorescent and mercury lamps</td>
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<td><strong>Cardiovascular</strong></td>
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<td>Coronary artery disease</td>
<td>Carbon monoxide</td>
<td>Working with combustion products</td>
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<td>Stress</td>
<td>Poor work organization, work design and management</td>
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<td>Conditions</td>
<td>Causal agent</td>
<td>Occupations/exposure</td>
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<td><strong>Gastrointestinal</strong></td>
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<td>Hepatitis</td>
<td>Polychlorinated biphenyls</td>
<td>Electrical equipment manufacturing and repair</td>
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<tr>
<td><strong>Others</strong></td>
<td></td>
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<tr>
<td>Metal fume fever</td>
<td>Zinc, Iron, Copper, Aluminium,</td>
<td>Welding, foundry work</td>
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<td>Tin, Chromium, Nickel</td>
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<tr>
<td>Memory impairment</td>
<td>Carbon disulfide</td>
<td>Viscose rayon work</td>
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<td></td>
<td>Organic solvents</td>
<td>Many occupations</td>
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5 MOST FREQUENT OCCUPATIONAL DISEASES
Igor Bátorá

Work-related musculoskeletal disorders (MSDs) are impairments of body structures such as muscles, joints, tendons, ligaments, nerves, bones or a localised blood circulation system caused or aggravated primarily by the performance of work and by the effects of the immediate environment where the work is carried out. Most work-related MSDs are cumulative disorders, resulting from repeated exposures to high intensity loads over a long period of time and/or awkward postures in the workplace. The symptoms may vary from discomfort and pain to decreased body function and invalidity. Most frequent are as follows:
- Lateral and medial epicondylitis
- Subacromial bursitis (impingement syndrome)
- Carpal tunnel syndrome
- Hand-arm vibration syndrome

Figure 8 Awkward hand postures in the workplace
Diseases due to overstraining of the muscular and tendonous insertions

Definition of the disease:

Lateral epicondylitis (tennis elbow, most common overuse syndrome in the elbow, occurs at least five times more often on the lateral rather than on the medial aspect of the joint): inflammation of the extensor tendons at the lateral epicondyle.

Medial epicondylitis (golf elbow): Inflammation of the flexor tendons at the medial epicondyle.

Definition of causal agent:

Prolonged periods of forceful and repetitive arm movements, repetitive stress of forearm muscles.

Main occupational exposure:

Repetitive and forceful hand use, e.g. in meat cutting, manual assembly, computer work, driving, use of hand-held tools.

Symptoms and diagnostic criteria:

Lateral epicondylitis (tennis elbow): pain and tenderness over the lateral epicondyle of the humerus, radiating into the forearm, and pain on resisted dorsiflexion of the wrist, middle finger or both. Chair test: The patient grasps the back of the chair while standing behind it and attempts to raise it by putting their hands on the top of the chair back. Pain reproduction at the lateral epicondyle is a positive test.
Medial epicondylitis (golf elbow): pain and tenderness maximal over the medial epicondyle, radiating into the forearm, which is aggravated by wrist flexion and pronation.

**Subacromial bursitis**

**Definition of the disease:**
Shoulder bursitis is inflammation of a particular area within the shoulder joint. The proper terminology is 'impingement syndrome.' Impingement syndrome occurs when there is inflammation of the rotator cuff tendons and the bursa that surrounds these tendons. Impingement syndrome occurs when there is inflammation between the top of the humerus (arm bone) and the acromion (tip of the shoulder blade). Between these bones are the tendons of the rotator cuff, and the bursa that protects these tendons. Normally, these tendons slide effortlessly within this space, called the subacromial space. In some people this space becomes too narrow for normal tendon motion, and the tendons and bursa become inflamed. Inflammation leads to thickening of the tendons and bursa, which leads to the lack of adequate room in the subacromial space. The acromion can rub against ("impinge" on) the tendon and the bursa, causing the pain.

**Definition of causal agent:**
repetitive activities with an elevated arm and big amplitude of movements in shoulder, overhead overuse activities.

**Main occupational exposure:**
slaughter house work, painting, decorating, nursing, fruit picking, blacksmiths, sawyers, earth diggers.
Symptoms and diagnostic criteria:
Difficulty reaching up behind the back, pain when the arms are extended above the head, weakness of shoulder muscles, pain while sleeping at night. When actively abducting the arm elicits a painful arc occurs between 80 and 120 degrees. X-rays may help to see calcification in the subacromial space and rotator cuff may be identified. Magnetic Resonance Imaging may be indicated to evaluate subacromial bursitis.

![Subacromial bursitis](http://www.acacpt.com/Injuries-Conditions/Shoulder/Shoulder)

Carpal tunnel syndrome
Definition of the disease:
The carpal tunnel is an anatomical compartment of the hand; it is bounded on three sides by carpal bones which form an arch and on the palmar side by the transverse carpal ligament. Carpal tunnel syndrome (CTS) results from compromise of median nerve function at the wrist, caused by increased pressure in the carpal tunnel. CTS is a relatively common condition that causes pain, numbness and a tingling sensation in the hand and fingers.

Definition of a causal agent:
in general, anything that crowds, irritates or compresses the median nerve in the carpal tunnel space can lead to carpal tunnel syndrome like repetitive hand motions, awkward hand positions, strong gripping, mechanical stress on the palm, working with vibrating tools or on an assembly line that requires prolonged or repetitive flexing of the wrist may create harmful pressure on the median nerve or worsen existing nerve damage. Other not occupational reasons are: wrist fracture, diabetes, sex (more often in women), illnesses that are characterised by inflammation, such as rheumatoid arthritis, fluid retention, common during
pregnancy or menopause, obesity, thyroid disorders and kidney failure may increase the pressure within carpal tunnel.

Main occupational exposure:
letter and parcels sorting, meat cutting, drilling, sawing, computer work, food processing, production sewer, tailor, garment worker/stitcher, musician, assembly worker, carpenter, painter.

Symptoms and diagnostic criteria:
The symptoms of CTS tend to develop gradually and usually start off being worse at night or early in the morning. The main symptoms: tingling, numbness and pain occur in the thumb, the index finger, the middle finger and half of the ring finger. Other symptoms: weakness of the hand radiated or referred pain into the arm and shoulder. Pressure on the median nerve at the wrist, produced by bending the wrist, tapping on the nerve or simply pressing on the nerve, can bring on the symptoms. Electromyography (EMG) is the “gold standard” for the detection of CTS. Nerve conduction studies use EMG to measure the speed at which impulses travel along the median nerve. Delayed responses between the time the current is delivered and the response of the muscle indicate the ischemic damage of the median nerve.

Figure 12 Carpal tunnel syndrome
http://images.ccohs.ca/oshanswers/office15c.gif
Hand-arm vibration syndrome

Definition of the disease:
Mechanical vibration is an oscillating motion about a central fixed position. Vibration frequency, expressed in Hertz (Hz), describes the cyclic nature of vibration. Vibration is separated into two subcategories: hand-arm vibration (HAV) and whole–body vibration (WBV). For HAV the relevant nominal frequency range is from 5 to 1500 Hz but frequencies usually occur between 125 till 300 Hz.

Hand-arm vibration exposure affects the blood flow (vascular effect) and causes loss of touch sensation (neurological effect) in fingers. Vibration can cause changes in tendons, muscles, bones and joints as well. These effects are known as Hand-Arm Vibration Syndrome (HAVS) or Raynaud's phenomenon of occupational origin. The symptoms are aggravated when the hands are exposed to cold. Workers affected by HAVS commonly report: attacks of whitening (blanching) of one or more fingers when exposed to cold, tingling and loss of sensation in the fingers, loss of light touch, pain and cold sensations between periodic white finger attacks, loss of grip strength, bone cysts in fingers and wrists (Fig. 36)

![Figure 13 Blanching of fingers](http://img.medscape.com/pi/emed/ckb/rheumatology/329097-1339496-331197-1696719.jpg)

Definition of a causal agent:
HAVS is caused by repeated and frequent use of hand-held vibrating tools, for example power drills, chainsaws, pneumatic drills, grinding machine, chipping hammer, spinning, fettling, using a grinding wheel, high pressure water hose, hammer drill, rammer, chisel, or other pneumatic tool etc. It may also be caused by holding or working with machinery that vibrates. It is due to slight but repeated injury to the small nerves and blood vessels in the fingers. Over time these may gradually lose some of their function and cause symptoms.
Symptoms and diagnostic criteria:
Workers affected by HAVS commonly report: attacks of whitening (blanching) of one or more fingers when exposed to cold, tingling and loss of sensation in the fingers, loss of light touch, pain and cold sensations between periodic white finger attacks, loss of grip strength, bone cysts in fingers and wrists. The symptoms are aggravated when the hands are exposed to cold.

The cold provocation test (5 min immersion of hands in water 10 C°) provides visual evidence of blanching. Neurological tests involve the determination of vibrotactile perception. Tests that measure induction of vasospasm utilise plethysmography (strain gauge or photocell) or laser Doppler techniques.

Reference:

Work-related low back pain

Definition of the disease:
Work-related low back pain correspond to intervertebral disc-related diseases of the lumbar spine from many years of carrying or lifting heavy loads, occupations in extreme postures of
full flexion or oscillation of the whole body when seated, and which compel the cessation of all activities which are or could be the cause for the origin, exacerbation or recurrence of the disease. It is commonly assumed that low back pain (LBP) is caused by work, but the relationship between the physical demands of work and LBP is complex and inconsistent. Studies suggest that between 60% and 90% of people will suffer from low back disorders at some point in their life. Data from the European survey on working conditions reveal that 30% of European workers suffer from back pain, which tops the list of all reported work-related disorders. Chronic low back pain is defined as back pain that lasts for longer than 7-12 weeks.

**Definition of a causal agent:**
Work factors that increase the risk of low back disorders:

- **physical aspects of work:**
  Heavy physical work
  Lifting and handling of loads
  Awkward postures (for example: bending; twisting; static postures)
  Whole body vibration (for example truck driving)

- **psychosocial work-related factors:**
  Low social support
  Low job satisfaction

- **work organisation factors:**
  Poor work organisation
  Low job content

**Symptoms and diagnostic criteria:**
A common feature of low back pain, that lasts for longer than 7-12 weeks, is radiculopathy which occurs when a nerve root is irritated by a herniated disc of the spine. Sciatica refers to the most common symptom of radiculopathy: a sharp or burning pain or numbness and tingling that extends down the back or side of the thigh, usually to the foot or ankle.
Imaging tests: plain x-rays, CT (computed tomography) scanning, or an MRI (magnetic resonance imaging), is recommended. However, X-rays do not usually show enough detail to diagnose a herniated disc, therefore CT scanning and MRI provide more detailed images of the soft tissues and bony structures.

Reference:

Hypoacusis or deafness caused by noise

Definition of the disease:
A sensorineural hearing loss results from damage to the cochlea itself, which is composed of sensory hair cells and nerves. Noise-induced hearing loss (NIHL) results from sensorineural damage. In NIHL, the sensory hair cells of the cochlea are damaged and die. Because of the spiral conical structure of this organ and its internal resonances, certain areas of this organ are more damaged by noise than others. The area of the cochlea that is most damaged by noise are the hair cells that detect sound frequencies around 4000 Hz.

Definition of causal agent:
Sound is a phenomenon by means of which mechanical vibration energy is propagated through an elastic medium, generally air, giving rise to auditory perception. Noise is a class of sounds that are disturbing, harmful or detrimental to hearing. The risk of noise induced chronic hearing loss depends on the cumulative cochlear noise exposure, which is determined
by daily noise exposure level, including impulsive noise, and exposure time in years. Occupational exposure assessed by history and study of working conditions providing evidence of prolonged or repeated cochlear exposure to noise of over 85 dB.

**Figure 16 Anatomy of the ear**
http://www.physlink.com/education/askexperts/ae37.cfm

**Symptoms and diagnostic criteria:**

1. **Acute effects**
Dizziness, tinnitus (noise or ringing in the ears), hypoacusis which can lead to total deafness partly reversible, depending on the energy of the sound wave and the duration of exposure.

2. **Chronic effects**
The disease develops slowly and insidiously. Hypoacusis is characterized by a quantitative reduction in auditory sensitivity, by a loss of the ability to discriminate between sounds. It is bilateral and generally symmetrical, irreversible but usually not progressive once exposure to noise ceased.

Audiogram, a graph that shows the audible threshold for standardized frequencies as measured by an audiometer, when measuring hearing loss, the speech frequencies around 4 kHz are most affected. The audiogram below shows both normal hearing (straight line) and a person with NIHL (showing a 4000 Hz “notch” typical for occupational hearing loss):
Occupational diseases of respiratory system

Silicosis

Definition of the disease:
Silicosis is a fibrotic pneumoconiosis, (the accumulation of dust in the lungs and the tissue reactions to its presence) that is caused by the inhalation of fine particles of crystalline silicon dioxide (silica). The classic form can be classified as simple or complicated, according to the radiographic findings: simple silicosis with small and round or irregular opacities (nontransparent area) and complicated silicosis with large conglomerate opacities that equate to progressive massive fibrosis (Fig. 41)
Definition of causal agent:
Tiny particles of silicon dioxide in the form of unbound (free) crystalline silica (usually quartz). Crystalline silica classified as carcinogenic to humans.

Main occupational exposure:
Workers at greatest risk are those who move or blast rock and sand (miners, quarry workers, stonecutters) or who use silica-containing rock or sand abrasives (sand blasters; glass makers; foundry, gemstone, and ceramic workers; potters), coal miners.

Symptoms and diagnostic criteria:
Chronic silicosis is often asymptomatic, but many patients eventually develop dyspnea during exertion that progresses to dyspnea at rest. Productive cough, when present, may be due to silicosis, coexisting chronic occupational (industrial) bronchitis, or smoking. Pulmonary hypertension and respiratory failure with or without right ventricular failure may develop in advanced disease. Carcinoma and tuberculosis are potential serious complications of silicosis. Diagnosis: occupational history of silica exposure; chest CT or chest x-ray; spirometry (symmetric reduction in lung volumes and reduction of the diffusing capacity), sometimes tissue biopsy for confirmation.

Asbestos induced diseases

Definition of causal agent:
Asbestos is a fibrous (Fig. 42) silicate which exists in various forms, classified as a known carcinogen:
- Serpentines: chrysotile
- Amphiboles: crocidolite, amosite, actinolite, tremolite, anthophyllite. There is sufficient evidence in humans for the carcinogenicity of all forms of asbestos.
All these fibres are capable of causing the following diseases:
1. Asbestosis - parenchymal lung disease
2. Asbestos-induced pleural diffuse benign thickening mainly of the visceral pleura
3. Asbestos-induced mesothelioma, malignant tumour of the pleura, peritoneum, pericardium
Main use and occupational exposure:
In Europe, exposure levels have fallen significantly, and some types of exposure have disappeared, but exposure can still occur in connection with coatings remaining in place, insulation, ovens, construction materials containing asbestos etc. (Fig. 43). Certain working operations dealing with asbestos still in place may involve significant exposure (asbestos removal, building maintenance, dismantling/refurbishment of ships, etc.).

Asbestosis
Definition of the disease:
Bilateral, diffuse, interstitial pulmonary fibrosis caused by large exposure to asbestos.

Symptoms and diagnostic criteria:
Breathlessness. Chest radiograph (HRCT scans more sensitive): symmetric irregular opacities in the lower parts of the lungs, relative sparing of the upper lobes. Spirometry: symmetric reduction in lung volumes – restrictive damage, reduction of the diffusing capacity (Fig. 44)
Mesothelioma
associated with asbestos exposure, is a malignancy that occurs in the lining of the lungs, abdomen, and heart.

Asbestos-related lung cancer
This cancer affects the lining of the lungs, much like mesothelioma. The latency period is the same as mesothelioma, about 20-40 years. Smoking combined with asbestos exposure does greatly increase the risk of developing lung cancer

Extrinsic allergic alveolitis
Definition of the disease:
Extrinsic allergic alveolitis EAA (also known as hypersensitivity pneumonitis HP) comprises a group of related inflammatory interstitial lung diseases that result from hypersensitivity immune reactions to the repeated inhalation of various antigens derived from fungal, bacterial, animal protein, or reactive chemical sources.

Definition of causal agent:
Antigens in: mouldy hay, mouldy pressed sugarcane, mouldy compost and mushrooms, contaminated wood pulp, contaminated humidifiers, air conditioners, heating systems, mould on tobacco, cheese or cheese casings, chicken feathers and many others.

Main use and occupational exposure:
Farmers, mushroom workers, wood workers, tobacco workers, cheese workers, chicken breeders etc.

Symptoms and diagnostic criteria:
There are acute, subacute and chronic forms. Acute and subacute forms cause a pneumonitis like symptoms, which can be recurrent. Chronic disease can cause fibrosis, emphysema and permanent lung damage. Diagnosis: history of exposure to a known offending antigen, positive antibodies to the offending antigen in serum, symptoms occurring 4-8 hours after exposure. Spirometry: reduction in lung volumes – restrictive damage, reduction of the
diffusing capacity. HRCT is the most useful in diagnosis of mid–lung zone fibrosis in chronic form.

**Occupational asthma**

**Definition of the disease:**
Asthma induced by exposure in the working environment to airborne dusts, vapours or fumes, in workers with or without pre-existing asthma. Occupational asthma can be subdivided into:

- Sensitiser-induced occupational asthma, is characterised by a latency period between first exposure to a respiratory sensitiser at work and the first presentation of symptoms.
- Irritant-induced occupational asthma, starts typically within a few hours of a high-intensity exposure to an irritant gas, fume or vapour encountered at work.

**Main use and occupational exposure:** see chapter 15.

**Symptoms and diagnostic criteria:**
wheezing, shortness of breath, often, **runny nose** (rhinorrhea), **cough** and chest tightness weeks to years after workplace exposure. Diagnostic lung function challenge test with aerosol containing a small amount of a suspected chemical to see if it triggers a reaction.

**Reference:**

**Occupational skin diseases**

**Definition of the disease:**
Skin disorders belong to the most often encountered problems in the occupational health setting and can be divided in 3 groups:

1. Dermatitis (skin inflammation) — the most common is contact dermatitis (irritant and allergic contact dermatitis), which accounts for 90 % of all occupational skin diseases.
2. Skin damage — a collection of “benign” (not malignant) conditions, such as skin infections and damage from various physical agents.
3. Skin cancer (malignancy) — the most common is squamous cell carcinoma and basal cell carcinoma and the most serious is melanoma.

**Contact dermatitis**
Inflammatory reactions in the skin (eczema) with a spectrum of clinical and histopathological characteristics. In most cases, an occupationally related dermatitis will affect the hands alone...
or there may be spread onto the forearms. Occasionally, the face may be the prime site of dermatitis (for example, with airborne agents), other sites may be affected.

**Irritant contact dermatitis**
This is initiated by direct chemical or physical damage to the skin, often seen in the finger webs and on the backs of the hands, rather than on the palms. Contact dermatitis is particularly seen in “wet work”: solvents, detergents, soluble coolants, vegetable juices, wet cement, and occlusive gloves.

**Allergic contact dermatitis**
This is a manifestation of a type IV (delayed) hypersensitivity reaction. An allergic contact dermatitis will develop at the site of skin contact with the allergen, but secondary spread may occur.

**Definition of causal agent:**
Examples of common occupational allergens: nickel, fragrances, rubber chemicals, epoxy resins, formaldehyde, hairdressing chemicals, chromate (leather, cosmetics, cement), plant allergens and many others.

Main occupational exposure:
Hairdressing/ beauty therapy, food industry, health care including dental and veterinary workers, agriculture including gardeners and florists, cleaning, painting and decorating, motor vehicle repair, construction, printing.

**Symptoms and diagnostic criteria:**
Whether it is due to an irritation (80 % of cases) or an allergy (20 % of cases), the clinical appearance is often the same. There may be areas covered with vesiculopapules (vesicle: circumscribed, fluid-containing, epidermal elevation; papule: circumscribed, solid elevation of skin with no visible fluid), erythematos or lichenified patches. Irritant contact dermatitis may occur as “epidemics” in a workplace, allergic contact dermatitis is usually sporadic. The diagnosis of irritant factors is always subjective. Diagnosis of allergic contact factors is objective and provided only by diagnostic patch test investigations.

**Reference:**

Toxicity of metals
Among broad spectrum of metals represents inconsistently defined “heavy metals” group of great concern especially due to significant toxicity (e.g., lead, cadmium, mercury, chromium). They persist in the environment and can accumulate in plant and animal tissues. Chronic occupational exposure to metal dusts has also been linked to the development of pneumoconiosis, neuropathies, hepatorenal damage and a variety of cancers. The toxicity of heavy metals depends on a number of factors: the total dose absorbed, whether the exposure was acute or chronic, the age of the person (e.g. young children absorb several times the percent ingested compared with adults). The route of exposure is also important. Elemental mercury ($\text{Hg}^0$) is relatively inert in the gastrointestinal tract and also poorly absorbed through intact skin.

Lead

Definition of causal agent
Lead (Pb) is a soft, blue-grey metal characterized by high density, ductility and corrosion resistance. Lead has affinity for bone and other calcified tissue, is readily absorbed, mobilized and cumulated in the body. About 95% of the lead body burden is located in the bones. Biological half-time of Pb in blood can be as short as 20–40 days, lead stored in bones some years. Exposure occurs through inhalation, ingestion or occasionally through breaks in the skin.

Main use and occupational exposure
High-risk occupations include blasting or scraping of lead-painted metal, brass foundry work, flame welding and cutting of lead-painted metal, indoor shooting ranges, battery storage and recycling, lead smelting, production of lead metal and its compounds and alloys. Moderate to low-risk occupations include antique restoration, car repair, lead mining, lead soldering, and porcelain glaze manufacture.

Toxic effects
Acute symptoms: GI colic, severe constipation, acute encephalopathy and nephropathy. Chronic symptoms: peripheral neuropathy, encephalopathy with psychiatric symptoms, chronic nephropathy, anemia.

Diagnosis
Pb level in blood and urine, aminolevulinic acid (ALA) and coproporphyrin (UCP) in urine.
Therapy
administration of chelating agents (antidotes) to remove heavy metals from the body.

Mercury

Definition of causal agent
Mercury (Hg) exists in three main chemical forms: elemental, inorganic mercurous and mercuric salts, and organic compounds. Elemental Hg is a silver-grey liquid at room temperature which vaporises slowly, is almost completely absorbed by the respiratory system, whereas ingested elemental Hg is not readily absorbed and is relatively harmless. Once absorbed, elemental Hg can cross the blood–brain barrier into the nervous system. Most exposure to elemental mercury is from occupational sources. The biological half time is about 15-30 days.

Main use and occupational exposure
Mining, smelting, metal refining, chlorine production, cold extraction, dentistry (amalgam), battery manufacture, fossil fuel (coal, natural gas) burning, catalysts, production of steel, cement production, laboratory usages, electrical industry, mercury in control instruments.

Toxic effects
The main toxic effects are observed in the kidney (elemental, inorganic), gastrointestinal system (inorganic), central nervous system (elemental and organic) and respiratory system (elemental mercury vapour). They include:
- kidney: tubular impairment, oliguria, proteinuria, nephrotic syndrome, renal failure;
- central nervous system: erethism, tremor, loss of memory, hallucinations, polyneuropathy;
- gastrointestinal system: gingivitis, salivation, stomatitis;
- respiratory system: inflammation, bronchitis, pulmonary edema, fibrosis.

Diagnosis
Biological monitoring: Hg in blood and urine.

Therapy
Administration of chelating agents (antidotes) to remove heavy metals from the body.

Chromium

Definition of causal agent
Chromium (Cr) is a hard blue-white to steel-grey, metal very resistant to corrosion. It is
found in nature only in the combined state and it forms a number of compounds. The most used for industrial purpose are the hexavalent Cr (VI) (chromous, Cr$^{6+}$) and trivalent Cr (III) (chromic, Cr$^{3+}$) which are of biologic significance.

**Main use and occupational exposure**
The stainless steel production and welding, manufacture of alloys, metal-plating industry, manufacture of pigments, wood preservation, tanning leather industry, toners for copying machines, magnetic tapes, lithography, photography, chromium in cement.

**Toxic effects**
Chromium compounds are both skin and mucous membrane irritants, and skin and pulmonary sensitisers. Dermatitis, chrome ulcers, corrosion and perforation of the nasal septum, conjunctivitis and lacrimation also occur. Hexavalent chromium compounds are carcinogenic (bronchogenic carcinoma).

**Diagnosis**
Cr concentrations in whole blood, plasma, serum or urine.

**Therapy**
No proven antidote is available for chromium poisoning. In cases of acute high-level Cr exposure is supportive and symptomatic.

**Other metals of concern.**
- **Beryllium (Be):** skin irritation and sensitisation, chronic granulomatous lung disease.
- **Cadmium (Cd):** „metal fume fever“, nephrotoxicity due the a tubular dysfunction.
- **Manganese (Mn):** encephalopathy and manganism - Parkinsonian syndrome.
- **Nickel (Ni):** allergic contact dermatitis (nickel itch), lung fibrosis. Human studies have reported an increased risk of lung and nasal cancers among nickel refinery workers exposed to nickel refinery dust.

**Toxicity of organic solvents**
The function of a solvent is to disperse solids, liquids or gases into solution and to separate the solute molecules. Most solvents are volatile. The property of quick evaporation makes solvents available for exposure and absorption into the body. In general, the toxicity of solvents is related to their ability to dissolve fatty materials. The main body systems affected are the lungs (irritation, allergy), the skin (irritation, inflammation), the central nervous system (narcotic effect, „solvent neurotoxic syndrome“ i.e. difficulties in concentration, forgetfulness,
headaches, irritability, insensitivity, personality disorders, mental disabilities), the liver, and the kidneys.

**Benzene**

**Definition of causal agent**
Benzene is a volatile, colourless, liquid, aromatic hydrocarbon.

**Main use and occupational exposure**
currently present in solvent mixtures in limited concentrations of < 0.1; still present in car fuel (1 to 2% in petrol). It is used in the synthesis of a variety of chemical products (styrene, cumene, cyclohexane, nitrobenzene, chlorobenzene, phenol, diphenyl), production of benzene via coal tar distillation or from petroleum. Benzene is absorbed by the skin.

**Toxic effects**
At high concentrations, benzene has a narcotic effect on the central nervous system. Acute effects: headache, excitement, confusion, euphoria, nausea, vertigo, and drowsiness, respiratory paralysis and death. Chronic benzene intoxication creates a disturbance in the hematopoietic (blood-forming) system: bone marrow depression leading to aplastic anemia. Benzene is a well-established human carcinogen and mutagenic to germ cells. Epidemiological studies demonstrated a causal relationship between benzene exposure and the development of acute myeloblastic leukemia.

**Diagnosis**
Benzene in blood, in urine (end of shift sample) metabolites of benzene: t,t-muconic acid, S-phenylmercapturic acid.

**Toluene**

**Definition of a causal agent**
Toluene (methylbenzene) is clear, colourless liquid volatile and easily flammable at ambient temperature, with a sweet, pungent odour. The technical product may contain small amounts of benzene.

**Main use and occupational exposure**
Toluene is used in the production of benzoic acid, benzaldehyde, explosives; solvent for paints and coatings, gums, resins, oils, rubber, cosmetics, pharmaceuticals.

**Toxic effects**
The CNS is the primary target organ for acute and chronic exposures: fatigue, sleepiness, headaches, and nausea. CNS depression and death have occurred at higher levels of exposure. Neurobehavioral effects have been observed in chronic occupationally exposed workers. High chronic toluene exposure may induce liver enlargement.

**Diagnosis**
Toluene in blood, metabolite o-cresol in urine

**Halogenated derivates of the hydrocarbons**

**Trichloroethylene**

**Definition of causal agent**
Trichloroethylene is a non inflammable fluid with a chloroform-like odour.

**Main use and occupational exposure**
Metal degreasing; extraction solvent for oils, fats, waxes; dry cleaning; refrigerant and heat exchange liquid; fumigant; cleaning and drying electronic parts; diluent in paints and adhesives; chemical intermediate.

**Toxic effects**
Trichloroethylene can cause irritation of the skin and mucous membranes. Acute effect: headache, dizziness, nausea, drowsiness, weakness, confusion, loss of consciousness, coma. Similarly to other organic solvents, trichloroethylene can cause, in repeated, prolonged exposures, chronic toxic encephalopathy.

**Diagnosis**
Principal metabolites of trichloroethylene: trichloroethanol in blood, trichloroacetic acid in urine.

**Other halogenated derivates of the hydrocarbons of concern**

**Methylene chloride and Tetrachloroethylene:** similar toxicity like trichloroethylene.

**Carbon tetrachloride:** extreme toxic for liver and kidney, probable human carcinogen.

**Vinyl chloride monomer:** Raynaud’s phenomenon (excessively reduced blood flow) in the hands and feet, liver fibrosis, angiosarcoma (malignant neoplasm of cells that line vessel walls) and hepatocellular carcinoma of the liver.
Carbon disulphide

Definition of causal agent
Carbon disulphide (CS₂) is a colourless, volatile liquid which yellows on exposure to air and light. It is highly reactive and very flammable. In its pure state is has a sweet, pleasing and aether-like odour.

Main use and occupational exposure
CS₂ is mainly used in the production of viscose rayon fibre, cellulose film and other viscose products. It is also used as a solvent and for the manufacture of pesticides, dyes, drugs and in rubber curing.

Toxic effects
An industrial neurotoxic chemical. Inhalation is the main route of absorption. Principal target organ: the nervous system, although cardiovascular (an increased risk of cardiovascular diseases) and other effects are also recognized. Chronic exposure can produce permanent central and peripheral nervous system damage. Symptoms: headache, dizziness, fatigue, nervousness, irritability, sleep disturbances, short memory loss, melancholia, increased suicide rate, sexual dysfunction, psychological and behavioural disorders.

Diagnosis
2-thiothiazolidine-4-carboxylic acid (TTCA) in the urine, carbon disulphide in the exhaled air.

Toxic alcohols

Ethylene glycol

Definition of causal agent
A colorless sweet-tasting viscous diol, C₂H₆O₂.

Main use and occupational exposure
Ethylene glycol is used in antifreeze (automotive antifreeze and hydraulic brake fluid) or liquid coolants.

Toxic effects
The severity of ethylene glycol toxicity is related to the metabolic acidosis resulting from the biotransformation of ethylene glycol into toxic metabolites. Glycolic acid causes severe acidosis and oxalate precipitates as calcium oxalate in the kidneys. For ethylene glycol acute systemic toxicity has only been described after ingestion. Ingestion of 1 mg/kg body weight can lead to severe intoxication starting with central nervous depression, followed by metabolic acidosis and ultimately renal failure.
Diagnosis
Severe metabolic acidosis (pH in blood). Calcium oxalate (square envelope shape) in urine (Fig. 45). Ethylene glycol concentrations in serum and urine.

Figure 22 Calcium oxalate in urine
http://3.bp.blogspot.com/_E7YrQ5zrPDA/SqaPA3wr8HI/AAAAAAAAABs/XLPP7ai4_E/s320/Calcium+Oxalate.jpg

Therapy
The toxicity of ethylene glycol comes from its metabolism to glycolic acid and oxalic acid. The goal of therapy is to prevent the formation of these metabolites by inactivation of enzyme alcohol dehydrogenase using competitive effect of ethanol. 4-Methylpyrazole (4-MP, fomepizole) more effectively inactivates alcohol dehydrogenase, therefore is the treatment of choice.

Other toxic alcohols of concern
Methanol: extremely toxic due to metabolite formic acid. Fatalities might occur from ingestion of 0.5 ml/kg of 100 percent methanol. An amount of 100 - 200 ml is fatal to most adults. Ingestion of less than 30 ml has been reported to cause death. The optic nerve is particularly sensitive to the effect of methanol and even a non-fatal dose can destroy the nerve, leading to permanent blindness. Treatment see ethyleneglycol.
Isopropyl alcohol: not as toxic as methanol or ethylene glycol, lethal dose to be 250 ml in humans.

Toxic gases
Toxic gases belong to the huge range of airborne contaminants in workplace air - vapours, fumes, mists, dusts and fibres. There is no physical barrier between the air and the various structures of respiratory system, gases can enter the body very easily and quickly. The toxic
effects of gases are manifold, as follows: asphyxiants (reduce or displace the normal oxygen concentration in breathing air), irritants, sensitisers.

**Carbon monoxide**

**Definition of causal agent**
Carbon monoxide (CO), „silent invisible killer“, is a colourless, odourless, tasteless, lighter than air non-irritant gas generated by incomplete combustion of carbon-containing organic material (coal, paper, wood, oil, gasoline). Frequent reason of non-occupational fatal deaths (Fig. 46)

**Main occupational exposure**
Working with combustion products, motor vehicle exhaust, heating facilities, incineration and manifold industrial processes. CO is non occupational problem as well (Fig. 46).

**Toxic effects**
The gas has a > 200-fold greater affinity for haemoglobin than oxygen; it combines with hemoglobin to produce carboxyhemoglobin (COHb). Inhibition of the normal oxygen-carrying capacity of the blood is the reason of tissue hypoxia. CO is a chemical asphyxiant.

<table>
<thead>
<tr>
<th>Percent COHb in blood</th>
<th>Signs and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 10</td>
<td>minimal manifestation,</td>
</tr>
<tr>
<td>10 – 20</td>
<td>moderate dyspnea, headache</td>
</tr>
<tr>
<td>20 – 30</td>
<td>headache, throbbing in temples</td>
</tr>
<tr>
<td>30 – 40</td>
<td>dizzy, weakness, nausea, vomiting</td>
</tr>
<tr>
<td>40 – 50</td>
<td>confusion, tachycardia, collapse</td>
</tr>
<tr>
<td>50 – 60</td>
<td>increased respiration, coma</td>
</tr>
<tr>
<td>60 – 70</td>
<td>coma, convulsions, heart failure</td>
</tr>
<tr>
<td>70 – 80</td>
<td>death</td>
</tr>
</tbody>
</table>

Chronic effects are not well defined, although, prolonged exposure to carbon monoxide or following severe acute CO poisoning can cause chronic detriment in neurobehavioral functioning. The long-term exposure to lower levels of CO can produce progressive symptoms like headache, dizziness, nausea and exhaustion.

**Diagnosis**
COHb concentration in the blood.

**Therapy**
Oxygen administration and severe cases may need hyperbaric oxygen therapy.
Carbon dioxide

**Definition of causal agent**
Carbon dioxide (CO₂) is a colorless, odorless, tasteless gas, about one and one-half times as dense as air. It does not burn, and under normal conditions it is stable, inert and nontoxic, but it can cause death by suffocation if inhaled in large amounts (replacing oxygen in air). CO₂ is a chemical asphyxiant. Prevention of intoxication: to enter the hazardous space (e.g. wine cellar) with a candle and watch for the candle to extinguish.

**Main use and occupational exposure**
CO₂ is produced by volcanic outgassing, the combustion of organic matter and respiration processes of living aerobic organisms, by various microorganisms from fermentation and cellular respiration. CO₂ is used as a cryogenic agent in cooling, chilling and freezing applications of food products, in the pulp and paper industry, in electric arc welding, in fire extinguishers carbonated drinks etc.

**Toxic effects**
Headache, shortness of breath, dizziness, drowsiness, ringing in the ears, death.
Diagnosis
Hypercapnia (abnormally elevated carbon dioxide level in the blood)

Therapy
Treatment for hypercapnia includes exposure to high levels of oxygen and rest.

Chlorine
Definition of causal agent
Chlorine (Cl₂) is green-yellow gas, which is heavier than air and has a pungent, irritating odour similar to bleach that is detectable at low concentrations.

Main use and occupational exposure
Cl₂ is used to disinfect water and is part of the sanitation process for sewage and industrial waste, in paper and cloth industry as bleaching agent. It is also used in cleaning products, including household bleach, in the preparation of chlorides, chlorinated solvents, pesticides, polymers, synthetic rubbers, and refrigerants or as various cleaning solutions for general disinfection of hospital wards, clinics, theatres and departments.

Toxic effects
Most Cl₂ exposures occur via inhalation. Cl₂ causes eye/skin/airway irritation, sore throat and cough. At higher levels of exposure, signs and symptoms may progress to chest tightness, wheezing, dyspnea, and bronchospasm. Severe exposures may result in pulmonary edema (fluid in the lungs). Complications such as bronchiolitis, pulmonary fibrosis, emphysema, asthma may occur.

Diagnosis
Diagnosis is primarily based upon clinical examination and patient history, not laboratory testing.

Therapy
Inhalational Cl₂ poisoning is treated with supportive care and administration of humidified oxygen and bronchodilators

Other toxic gases of concern
Nitrogen oxides (NOx): irritant to the eyes, respiratory tract and skin; severe exposures to nitric oxide (NO) may result in methaemoglobinemia (methaemoglobin is a form of haemoglobin that has a decreased ability to bind oxygen), hypoxemia, pulmonary oedema (fluid in the lungs), lung inflammation etc.
Ammonia (NH₃): severe irritation even lesions of the skin, eyes and respiratory tract. Exposures to concentrated aerosols of ammonium solution can result in tracheal burns, airway obstruction, and pulmonal oedema.

Ozone (O₃): O₃ is produced by ultraviolet light from the welding arc. O₃ can cause gaseous irritation to all mucous membranes. Excessive exposure can cause pulmonary oedema.

Phosgene (COCl₂): Phosgene is formed by decomposition of chlorinated hydrocarbon solvents by intense heat e.g. during welding operations. It reacts with moisture in the lungs to produce hydrogen chloride (HCl - acid), which in turn destroys lung tissue.

Pesticides
Pesticides are chemical compounds that are used to kill pests, including insects, rodents, fungi and unwanted plants (weeds). Pesticides are used in public health to kill vectors of disease, such as mosquitoes, and in agriculture, to kill pests that damage crops. Benefits of pesticides are control of vector-borne diseases, increased agricultural productivity, and control of urban pests. Ideally, pesticides should be highly selective, destroying target organisms while leaving non-target organisms unharmed. In reality, most pesticides are not so selective. By their nature, pesticides are potentially toxic to other organisms, including humans, and need to be used safely and disposed of properly.

Most occupational exposures are caused by absorption through exposed skin such as the face, hands, forearms, neck, and chest. This exposure is sometimes enhanced by inhalation in settings including spraying operations in greenhouses and other closed environments, tractor cabs, and the operation of rotary fan mist sprayers. Extensive occupational exposure: pesticide formulators and manufacturers, agricultural workers, retail workers in stores, fire-fighters and police officers, flight attendants at application.

Classes of pesticides by target organism:
Insecticides (kill insects; organochlorines, organophosphates, carbamates, pyrethroids);
Herbicides (kill weeds and other plants that grow where they are not wanted);
Rodenticides (kill rodents);
Fungicides (kill fungi including mildews, molds and rusts);
Molluscicides (kill snails and slugs);
Nematicides (kill nematodes, microscopic, worm-like organisms that feed on plant roots);
Algaecides (kill algae in lakes, canals, swimming pools);

Based on lethal dose (LD₅₀) most toxic pesticides for mammals and humans are organophosphates (OP) and carbamates, both may cause serious life-threatening conditions.
Organophosphates

Definition of a causal agent

Organophosphorus pesticides (Ops) are a group of chemicals that are characterised by the following general formula:

\[
\begin{array}{c}
\text{OP} \\
R_1 - \text{P} - \text{O} \text{R}_3 \\
\text{OR}_2
\end{array}
\]

Organophosphate

“insecticide”

OP are derivates of esters or thiols of phosphoric, phosphonic, phosphorothionic or phosphono thionic acids (examples: pirimiphos, chlorpyriphos, dimethoate, diazinon). Because of their relatively fast rate of degradation, they replaced long-persistenting organochlorines (DDT).

Toxic effects

The clinical picture of acute intoxication is attributable to the irreversible inhibition of enzyme Acetylcholinesterase (AChE) activity in the nervous system and to the consequent acetylcholine accumulation in the nerve synapses and neuro muscle junctions (muscarinic and nicotinic systems). AChE is an enzyme present in the synaptic cleft (Fig. 47 Fig. 48) that hydrolyses acetylcholine, a neurotransmitter released by the neuron during transmission of the action potential.
Under normal conditions, acetylcholine is released during transmission of the nerve impulse so that it carries the impulse through the synaptic junction. After its short action, acetylcholine is hydrolysed by AChE in order to avoid accumulation of the neurotransmitter. This enzyme is necessary for controlling nerve impulse transmission between nerve fibres. A loss of this enzyme function results in an accumulation of acetylcholine, which causes unregulated
nervous impulses (cholinergic crisis). Higher levels of acetylcholine result in sensory and behavioral disturbances, incoordination, and depressed motor function. Muscarinic and nicotinic symptoms of acute poisoning develop during or after exposure, within minutes to hours, depending on route of entry to body. Inhalation exposure results in the fastest appearance of symptoms, followed by the gastrointestinal route, and then the dermal (skin) route. Recovery from organophosphate exposure depends upon generation of a new enzyme. In addition to acute effects, some OP compounds have been associated with the delayed neurotoxicity, known as organophosphorus - induced delayed neuropathy (OPIDN). The characteristic clinical sign is bilateral paralysis of the distal muscles, predominantly of the lower extremities, occurring from 7 to 10 days following ingestion.

**Muscarinic** effects by organ systems include the following:
- **Cardiovascular** – Bradycardia (decreased puls rate), hypotension
- **Respiratory** - Bronchorrhea (excessive discharge of watery mucous from the lungs), bronchospasm, cough, severe respiratory distress
- **Gastrointestinal** - Hypersalivation, nausea and vomiting, abdominal pain, diarrhea, fecal incontinence
- **Genitourinary** - Incontinence
- **Ocular** - Blurred vision, miosis (constriction of the pupil of the eye)
- **Glands** - Increased lacrimation, profuse sweating

**Nicotinic** signs and symptoms include muscle fasciculations, cramping, weakness, and diaphragma (the main muscle of respiration between chest and abdomen) failure.
- **CNS effects** include anxiety, emotional lability, restlessness, confusion, ataxia, tremors, seizures, and coma.

**Diagnosis**
Typical muscarinic and nicotinic symptoms, enzymatic activity of AChE in red blood cells, metabolites of organophosphate in urine.

**Therapy**
- **Atropine** as an antidote, muscarinic antagonist, and thus blocks the action of acetylcholine in synaptic cleft peripherally in conjunction with oximes such as obidoxime or pralidoxime which are AChE reactivators used to restore AChE functionality.

**Carbamates**
**Definition of a causal agent**
The carbamate insecticides are esters of N-methyl carbamic acid (for example pirimicarb, carbofuran):

\[
\begin{align*}
\text{CH}_3 \\
\text{R} & \text{C} \text{N} \text{H} \\
\text{O} & \\
\text{Carbamate} \\
\text{“insecticide”}
\end{align*}
\]

**Toxic effects**

Toxic effects of poisoning are attributable to the reversible inhibition of AChE activity in the nervous system and to the consequent acetylcholine accumulation in the nerve synapses and neuromuscular junctions (muscarinic and nicotinic systems). Since, compared to organophosphates, carbamates are weaker AChE inhibitors (reversible inhibition), the duration of AChE inhibition is shorter and the severity of the signs and symptoms of poisoning is usually lower.

**Diagnosis**

Typical muscarinic and nicotinic symptoms, enzymatic activity of AChE in red blood cells soon after intoxication.

**Therapy**

Atropine as an antidote, to block the action of acetylcholine, but comparing to organophosphate not reactivators (short inhibition of AChE) are recommended.

**References:**

1. WHO. Pesticides. Available at: [http://www.who.int/topics/pesticides/en/](http://www.who.int/topics/pesticides/en/).
LIST OF ABBREVIATIONS

BEIs - Biological Exposure Indices
BMD - Benchmark Dose
BMDL - Benchmark Dose Lower-confidence Limit
Bq - Becquerel
CDC - Centers for Disease Control and Prevention
CLP - Classification, Labeling and Packaging
dB - Decibel
DNA - Deoxyribonucleic Acid
ED 50 - Effective Dose, 50%
EC - European Communities
EEC - European Economic Community
EMFs - Electromagnetic Fields
EU - European Union
GHS - Globally Harmonized System of Classification and Labelling of Chemicals
Gy - Gray
HBV - Hepatitis B virus
HBC - Hepatitis C virus
HEPA - High Efficiency Particulate Air
ILO - International Labour Organization
IR - Infrared radiation
IUR - Inhalation Unit Risk
LC 50 - Lethal Concentration, 50%
LD 50 - Lethal Dose, 50%
LOAEL - Lowest-Observed-Adverse-Effect Level
MSD - Musculoskeletal Disorders
NAS - National Academy of Sciences
NRC - National Research Council
NOAEL - No-Observed-Adverse-Effect Level
OECD - Organization for Economic Cooperation and Development
OELs - Occupational exposure limits
OH - Occupational Health
PCB - Polychlorinated Biphenyls
QSAR - Qualitative/Quantitative Structure-Activity Relationship
RA - Risk Assessment
REACH - Registration, Evaluation, Authorization and Restriction of Chemicals
RIC - Reference Concentration
RfD - Reference Dose
RPE - Rating of Perceived Exertion
STEL - Short Term Exposure Limit
STOT - Specific Target Organ Toxicity
Sv - Sievert
TWA - Time-Weighted Average
UFs - Uncertainty Factor
UN - United Nations
US EPA - Environmental Protection Agency of the USA

UV - Ultraviolet radiation
VCM - Vinyl Chloride Monomer
WHO - World Health Organization