THE CHEMICAL ANALYSIS OF TESLA MOTORS TOXIC BATTERY ISSUES

By Engineering Students at Stanford University and Chemix Research Group

GENERAL PROFILE

This presents a series of short discussions of the metals used in the Tesla Motors vehicles, particularly it's lithium ion batteries It contains a tabulation of major health effects, physical properties and physical and chemical hazards associated with these metals and many of their compounds (see table 63.2 and table 63.3). For additional information on the toxicity of related chemical substances and compounds see the latest OSHA and litigation filing evidence.

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ALUMINIUM

Occurrence and uses

Aluminium is the most abundant metal in the earth's crust, where it is found in combination with oxygen, fluorine, silica, etc., but never in the metallic state. Bauxite is the principal source of aluminium. It consists of a mixture of minerals formed by the weathering of aluminium-bearing rocks. Bauxites are the richest form of these weathered ores, containing up to 55% alumina. Some lateritic ores (containing higher percentages of iron) contain up to 35% Al_2O_3. Commercial deposits of bauxite are mainly gibbsite ($Al_2O_3 \cdot 3H_2O$) and boehmite ($Al_2O_3 \cdot H_2O$) and are found in Australia, Guyana, France, Brazil, Ghana, Guinea, Hungary, Jamaica and Suriname. World production of bauxite in 1995 was 111,064 million tonnes. Gibbsite is more readily soluble in sodium hydroxide solutions than boehmite and is therefore preferred for aluminium oxide production.

Aluminium is used widely throughout industry and in larger quantities than any other non-ferrous metal; worldwide primary metal production in 1995 was estimated at 20,402 million tonnes. It is alloyed with a variety of other material including copper, zinc, silicon, magnesium, manganese and nickel and may contain small amounts of chromium, lead, bismuth, titanium, zirconium and vanadium for special purposes. Aluminium and aluminium alloy ingots can be extruded or processed in rolling mills, wire-works, forges or foundries. The finished products are used in shipbuilding for internal fittings and superstructures; the electrical industry for wires and cables; the building industry for house and window frames, roofs and cladding; aircraft industry for airframes and aircraft skin and other components; automobile industry for bodywork, engine blocks and pistons; light engineering for domestic appliances and office equipment and in the jewellery industry. A major application of sheet is in beverage or food containers, while aluminium foil is used for packaging; a fine particulate form of aluminium is employed as a pigment in paints and in the pyrotechnics industry. Articles manufactured from aluminium are frequently given a protective and decorative surface finish by anodization.

Aluminium chloride is used in petroleum cracking and in the rubber industry. It fumes in air to form hydrochloric acid and combines explosively with water; consequently, containers should be kept tightly closed and protected from moisture.

Alkyl aluminium compounds. These are growing in importance as catalysts for the production of low-pressure polyethylene. They present a toxic, burn and fire hazard. They are extremely reactive with air, moisture and compounds containing active hydrogen and therefore must be kept under a blanket of inert gas.

Hazards

For the production of aluminium alloys, refined aluminium is melted in oil or gas-fired furnaces. A regulated amount of hardener containing aluminium blocks with a percentage of manganese, silicon, zinc, magnesium, etc. is added. The melt is then mixed and is passed into a holding furnace for degassing by passing either argon-chlorine or nitrogen-chlorine through the metal. The resultant gas emission (hydrochloric acid, hydrogen and chlorine) has been associated with occupational illnesses

and great care should be taken to see that appropriate engineering controls capture the emissions and also prevent it from reaching the external environment, where it can also cause damage. Dross is skimmed off the surface of the melt and placed in containers to minimize exposure to air during cooling. A flux containing fluoride and/or chloride salts is added to the furnace to assist in separation of pure aluminium from the dross. Aluminium oxide and fluoride fumes may be given off so that this aspect of production must also be carefully controlled. Personal protective equipment (PPE) may be required. The aluminium smelting process is described in the chapter Metal processing and metal working industry. In the casting shops, exposure to sulphur dioxide may also occur.

A wide range of different crystalline forms of aluminium oxide is used as smelter feed stock, abrasives, refractories and catalysts. A series of reports published in 1947 to 1949 described a progressive, non-nodular interstitial fibrosis in the aluminium abrasives industry in which aluminium oxide and silicon were processed. This condition, known as Shaver's disease, was rapidly progressive and often fatal. The exposure of the victims (workers producing alundum) was to a dense fume comprising aluminium oxide, crystalline free-silica and iron. The particulates were of a size range that made them highly respirable. It is likely that the preponderence of disease is attributable to the highly damaging lung effects of the finely divided crystalline free-silica, rather than to the inhaled aluminium oxide, although the exact aetiology of the disease is not understood. Shaver's disease is primarily of historical interest now, since no reports have been made in the second half of the 20th century.

Recent studies of the health effects of high level exposures (100 mg/m^3) to the oxides of aluminium amongst workers engaged in the Bayer process (described in the chapter Metal processing and metal working industry) have demonstrated that workers with more than twenty years of exposure can develop pulmonary alterations. These changes are clinically characterized by minor, predominantly asymptomatic degrees of restrictive pulmonary function changes. The chest x-ray examinations revealed small, scanty, irregular opacities, particularly at the lung bases. These clinical responses have been attributed to deposition of dust in the lung paraenchyma, which was the result of very high occupational exposures. These signs and symptoms cannot be compared to the extreme response of Shaver's disease. It should be noted that other epidemiological studies in the United Kingdom regarding widespread alumina exposures in the pottery industry have produced no evidence that the inhalation of alumina dust produces chemical or radiographic signs of pulmonary disease or dysfunction.

The toxicological effects of aluminium oxides remain of interest because of its commerical importance. The results of animal experiments are controversial. An especially fine (0.02 µm to 0.04 µm), catalytically active aluminium oxide, uncommonly used commercially, can cause lung changes in animals dosed by injection directly into the lung airways. Lower dose effects have not been observed.

It should also be noted that so-called "potroom asthma" which has frequently been observed among workers in aluminium processing operations, is probably attributable to the exposures to fluoride fluxes, rather than to the aluminium dust itself.

The production of aluminium has been classified as a Group 1, known human carcinogenic exposure situation, by the International Agency for Research on Cancer (IARC). As with the other diseases described above, the carcinogenicity is most likely attributable to the other substances present (e.g., polycyclic aromatic hydrocarbons (PAHs) and silica dust), although the exact role of the alumina dusts are simply not understood.

Some data on the absorption of high levels of aluminium and nervous tissue damage are found among individuals requiring kidney dialysis. These high levels of aluminium have resulted in severe, even fatal brain damage. This response, however, has also been observed in other patients undergoing dialysis but who did not have similar elevated brain aluminium level. Animal experiments have been unsuccessful in replicating this brain response, or Alzheimer's disease, which has also been postulated in the literature. Epidemiological and clinical follow-up studies on these issues have not been definitive and no evidence of such effects has been observed in the several large-scale epidemiological studies of aluminium workers.

ANTIMONY

Antimony is stable at room temperature but, when heated, burns brilliantly, giving off dense white fumes of antimony oxide (Sb_2O_3) with a garlic-like odour. It is closely related, chemically, to arsenic.

It readily forms alloys with arsenic, lead, tin, zinc, iron and bismuth.

Occurrence and Uses

In nature, antimony is found in combination with numerous elements, and the most common ores are stibnite (SbS_3), valentinite (Sb_2O_3), kermesite (Sb_2S_2O) and senarmontite (Sb_2O_3).

High-purity antimony is employed in the manufacture of semiconductors. Normal-purity antimony is used widely in the production of alloys, to which it imparts increased hardness, mechanical strength, corrosion resistance and a low coefficient of friction; alloys combining tin, lead and antimony are used in the electrical industry. Among the more important antimony alloys are babbitt, pewter, white metal, Britannia metal and bearing metal. These are used for bearing shells, storage battery plates, cable sheathing, solder, ornamental castings and ammunition. The resistance of metallic antimony to acids and bases is put to effect in the manufacture of chemical plants.

Hazards

The principal hazard of antimony is that of intoxication by ingestion, inhalation or skin absorption. The respiratory tract is the most important route of entry since antimony is so frequently encountered as a fine airborne dust. Ingestion may occur through swallowing dust or through contamination of beverages, food or tobacco. Skin absorption is less common, but may occur when antimony is in prolonged contact with skin.

The dust encountered in antimony mining may contain free silica, and cases of pneumoconiosis (termed silico-antimoniosis) have been reported among antimony miners. During processing, the antimony ore, which is extremely brittle, is converted into fine dust more rapidly than the

accompanying rock, leading to high atmospheric concentrations of fine dust during such operations as reduction and screening. Dust produced during crushing is relatively coarse, and the remaining operations—classification, flotation, filtration and so on—are wet processes and, consequently, dust free. Furnace workers who refine metallic antimony and produce antimony alloy, and workers setting type in the printing industry, are all exposed to antimony metal dust and fumes, and may present diffuse miliar opacities in the lung, with no clinical or functional signs of impairment in the absence of silica dust.

Inhalation of antimony aerosols may produce localized reactions of the mucous membrane, respiratory tract and lungs. Examination of miners and concentrator and smelter workers exposed to antimony dust and fumes has revealed dermatitis, rhinitis, inflammation of upper and lower respiratory tracts, including pneumonitis and even gastritis, conjunctivitis and perforations of the nasal septum.

Pneumoconiosis, sometimes in combination with obstructive lung changes, has been reported following long-term exposure in humans. Although antimony pneumoconiosis is regarded as benign, the chronic respiratory effects associated with heavy antimony exposure are not considered harmless. In addition, effects on the heart, even fatal, have been related to long-term occupational exposure to antimony trioxide.

Pustular skin infections are sometimes seen in persons working with antimony and antimony salts. These eruptions are transient and primarily affect the skin areas in which heat exposure or sweating has occurred.

Toxicology

In its chemical properties and metabolic action, antimony has a close resemblance to arsenic, and, since the two elements are sometimes found in association, the action of antimony may be blamed on arsenic, especially in foundry workers. However, experiments with high-purity metallic antimony have shown that this metal has a completely independent toxicology; different authors have found the average lethal dose to be between 10 and 11.2 mg/100 g.

Antimony may enter the body through the skin, but the principal route is through the lungs. From the lungs, antimony, and especially free antimony, is absorbed and taken up by the blood and tissues. Studies on workers and experiments with radioactive antimony have shown that the major part of the absorbed dose enters the metabolism within 48 hours and is eliminated in the faeces and, to a lesser extent, the urine. The remainder stays in the blood for some considerable time, with the erythrocytes containing several times more antimony than the serum. In workers exposed to pentavalent antimony, the urinary excretion of antimony is related to the intensity of exposure. It has been estimated that after 8 hours exposure to 500 µg Sb/m^3, the increase in concentration of antimony excreted in the urine at the end of a shift amounts on average to 35 µg/g creatinine.

Antimony inhibits the activity of certain enzymes, binds sulphydryl groups in the serum, and disturbs protein and carbohydrate metabolism and the production of glycogen by the liver. Prolonged animal experiments with antimony aerosols have led to the development of distinctive endogenous lipoid

pneumonia. Cardiac injury and cases of sudden death have also been reported in workers exposed to antimony. Focal fibrosis of the lung and cardiovascular effects have also been observed in animal trials.

The therapeutic use of antimonial drugs has made it possible to detect, in particular, the cumulative myocardial toxicity of the trivalent derivatives of antimony (which are excreted more slowly than pentavalent derivatives). Reduction in amplitude of T wave, increase of QT interval and arrhythmias have been observed in the electrocardiogram.

Symptoms

The symptoms of acute poisoning include violent irritation of the mouth, nose, stomach and intestines; vomiting and bloody stools; slow, shallow respiration; coma sometimes followed by death due to exhaustion and hepatic and renal complications. Those of chronic poisoning are: dryness of throat, nausea, headaches, sleeplessness, loss of appetite, and dizziness. Gender differences in the effects of antimony have been noted by some authors, but the differences are not well established.

Compounds

Stibine (SbH_3), or antimony hydride (hydrogen antimonide), is produced by dissolving zinc-antimony or magnesium-antimony alloy in dilute hydrochloric acid. However, it occurs frequently as a by-product in the processing of metals containing antimony with reducing acids or in overcharging storage batteries. Stibine has been used as a fumigating agent. High-purity stibine is used as an n-type gas-phase dopant for silicon in semiconductors. Stibine is an extremely hazardous gas. Like arsine it may destroy blood cells and cause haemoglobinuria, jaundice, anuria and death. Symptoms include headache, nausea, epigastric pain and passage of dark red urine following exposure.

Antimony trioxide (Sb_2O_3) is the most important of the antimony oxides. When airborne, it tends to remain suspended for an exceptionally long time. It is obtained from antimony ore by a roasting process or by oxidizing metallic antimony and subsequent sublimation, and is used for the manufacture of tartar emetic, as a paint pigment, in enamels and glazes, and as a flameproofing compound.

Antimony trioxide is both a systemic poison and a skin disease hazard, although its toxicity is three times less than that of the metal. In long-term animal experiments, rats exposed to antimony trioxide via inhalation showed a high frequency of lung tumours. An excess of deaths due to cancer of the lung among workers engaged in antimony smelting for more than 4 years, at an average concentration in air of 8 mg/m^3, has been reported from Newcastle. In addition to antimony dust and fumes, the workers were exposed to zircon plant effluents and caustic soda. No other experiences were informative on the carcinogenic potential of antimony trioxide. This has been classified by the American Conference of Governmental Industrial Hygienists (ACGIH) as a chemical substance associated with industrial processes which are suspected of inducing cancer.

Antimony pentoxide (Sb_2O_5) is produced by the oxidation of the trioxide or the pure metal, in nitric acid under heat. It is used in the manufacture of paints and lacquers, glass, pottery and pharmaceuticals. Antimony pentoxide is noted for its low degree of toxic hazard.

Antimony trisulphide (Sb_2S_3) is found as a natural mineral, antimonite, but can also be synthesized. It is used in the pyrotechnics, match and explosives industries, in ruby glass manufacture, and as a pigment and plasticizer in the rubber industry. An apparent increase in heart abnormalities has been found in persons exposed to the trisulphide. Antimony pentasulphide (Sb_2S_5) has much the same uses as the trisulphide and has a low level of toxicity.

Antimony trichloride ($SbCl_3$), or antimonous chloride (butter of antimony), is produced by the interaction of chlorine and antimony or by dissolving antimony trisulphide in hydrochloric acid. Antimony pentachloride ($SbCl_5$) is produced by the action of chlorine on molten antimony trichloride. The antimony chlorides are used for blueing steel and colouring aluminium, pewter and zinc, and as catalysts in organic synthesis, especially in the rubber and pharmaceutical industries. In addition, antimony trichloride is used in the match and petroleum industries. They are highly toxic substances, act as irritants and are corrosive to the skin. The trichloride has an LD_{50} of 2.5 mg/100 g.

Antimony trifluoride (SbF_3) is prepared by dissolving antimony trioxide in hydrofluoric acid, and is used in organic synthesis. It is also employed in dyeing and pottery manufacture. Antimony trifluoride is highly toxic and an irritant to the skin. It has an LD_{50} of 2.3 mg/100 g.

Safety and Health Measures

The essence of any safety programme for the prevention of antimony poisoning should be the control of dust and fume formation at all stages of processing.

In mining, dust prevention measures are similar to those for metal mining in general. During crushing, the ore should be sprayed or the process completely enclosed and fitted with local exhaust ventilation combined with adequate general ventilation. In antimony smelting the hazards of charge preparation, furnace operation, fettling and electrolytic cell operation should be eliminated, where possible, by isolation and process automation. Furnace workers should be provided with water sprays and effective ventilation.

Where complete elimination of exposure is not possible, the hands, arms and faces of workers should be protected by gloves, dustproof clothing and goggles, and, where atmospheric exposure is high, respirators should be provided. Barrier creams should also be applied, especially when handling soluble antimony compounds, in which case they should be combined with the use of waterproof clothing and rubber gloves. Personal hygiene measures should be strictly observed; no food or beverages should be consumed in the workshops, and suitable sanitary facilities should be provided so that workers can wash before meals and before leaving work.

ARSENIC

There are three major groups of arsenic (As) compounds:

1. inorganic arsenic compounds

2. organic arsenic compounds
3. arsine gas and substituted arsines.

Occurrence and Uses

Arsenic is found widely in nature and most abundantly in sulphide ores. Arsenopyrite (FeAsS) is the most abundant one.

Elemental arsenic

Elemental arsenic is utilized in alloys in order to increase their hardness and heat resistance (e.g., alloys with lead in shot-making and battery grids). It is also used in the manufacture of certain types of glass, as a component of electrical devices and as a doping agent in germanium and silicon solid-state products.

Trivalent inorganic compounds

Arsenic trichloride ($AsCl_3$) is used in the ceramics industry and in the manufacturing of chlorine-containing arsenicals. Arsenic trioxide (As_2O_3), or white arsenic, is useful in the purification of synthesis gas and as a primary material for all arsenic compounds. It is also a preservative for hides and wood, a textile mordant, a reagent in mineral flotation, and a decolourizing and refining agent in glass manufacture. Calcium arsenite ($Ca(As_2H_2O_4)$) and cupric acetoarsenite (usually considered $Cu(COOCH_3)_2$ $3Cu(AsO_2)_2$) are insecticides. Cupric acetoarsenite is also used for painting ships and submarines. Sodium arsenite ($NaAsO_2$) is employed as a herbicide, a corrosion inhibitor, and as a drying agent in the textile industry. Arsenic trisulphide is a component of infrared-transmitting glass and a dehairing agent in the tanning industry. It is also used in the manufacturing of pyrotechnics and semiconductors.

Pentavalent inorganic compounds

Arsenic acid ($H_3AsO_4 \cdot ½H_2O$) is found in the manufacture of arsenates, glass making and wood-treating processes. Arsenic pentoxide (As_2O_5), an herbicide and a wood preservative, is also used in the manufacture of coloured glass.

Calcium arsenate ($Ca_3(AsO_4)_2$) is used as an insecticide.

Organic arsenic compounds

Cacodylic acid ($(CH_3)_2AsOOH$) is used as a herbicide and a defoliant. Arsanilic acid ($NH_2C_6H_4AsO(OH)_2$) finds use as a grasshopper bait and as an additive in animal feeds. Organic arsenic compounds in marine organisms occur in concentrations corresponding to a concentration of arsenic in the range 1 to 100 mg/kg in marine organisms such as shrimp and fish. Such arsenic is mainly made up of arsenobetaine and arsenocholine, organic arsenic compounds of low toxicity.

Arsine gas and the substituted arsines. Arsine gas is used in organic syntheses and in the processing of solid-state electronic components. Arsine gas may also be generated inadvertently in industrial processes when nascent hydrogen is formed and arsenic is present.

The substituted arsines are trivalent organic arsenical compounds which, depending on the number of alkyl or phenyl groups that they have attached to the arsenic nucleus, are known as mono-, di- or tri-substituted arsines. Dichloroethylarsine ($C_2H_5AsCl_2$), or ethyldichloroarsine, is a colourless liquid with an irritant odour. This compound, like the following one, was developed as a potential chemical warfare agent.

Dichloro(2-chlorovinyl-)arsine ($ClCH:CHAsCl_2$), or chlorovinyldichloroarsine (lewisite), is an olive-green liquid with a germanium-like odour. It was developed as a potential warfare agent but never used. The agent dimercaprol or British anti-lewisite (BAL) was developed as an antidote.

Dimethyl-arsine $(CH_3)_2AsH$, or cacodyl hydride and trimethylarsine $(CH_3)_3As$), or trimethylarsenic, are both colourless liquids. These two compounds can be produced after metabolic transformation of arsenic compounds by bacteria and fungi.

Hazards

Inorganic arsenic compounds

General aspects of toxicity. Although it is possible that very small amounts of certain arsenic compounds may have beneficial effects, as indicated by some animal studies, arsenic compounds, particularly the inorganic ones, are otherwise regarded as very potent poisons. Acute toxicity varies widely among compounds, depending on their valency state and solubility in biological media. The soluble trivalent compounds are the most toxic. Uptake of inorganic arsenic compounds from the gastrointestinal tract is almost complete, but uptake may be delayed for less soluble forms such as arsenic trioxide in particle form. Uptake after inhalation is also almost complete, since even less soluble material deposited on the respiratory mucosa, will be transferred to the gastrointestinal tract and subsequently taken up.

Occupational exposure to inorganic arsenic compounds through inhalation, ingestion or skin contact with subsequent absorption may occur in industry. Acute effects at the point of entry may occur if exposure is excessive. Dermatitis may occur as an acute symptom but is more often the result of toxicity from long-term exposure, sometimes subsequent to sensitization (see the section “Long-term exposure (chronic poisoning)”).

Acute poisoning

Exposure to high doses of inorganic arsenic compounds by a combination of inhalation and ingestion may occur as a result of accidents in industries where large amounts of arsenic (e.g., arsenic trioxide), are handled. Depending on dose, various symptoms may develop, and when doses are excessive, fatal cases may occur. Symptoms of conjunctivitis, bronchitis and dyspnoea, followed by gastrointestinal

discomfort with vomiting, and subsequently cardiac involvement with irreversible shock, may occur in a time course of hours. Arsenic in blood was reported to be above 3 mg/l in a case with fatal outcome.

With exposure to sub-lethal doses of irritant arsenic compounds in air (e.g., arsenic trioxide), there may be symptoms related to acute damage to the mucous membranes of the respiratory system and acute symptoms from exposed skin. Severe irritation of the nasal mucosae, larynx and bronchi, as well as conjunctivitis and dermatitis, occur in such cases. Perforation of the nasal septum can be observed in some individuals only after a few weeks following exposure. A certain tolerance against acute poisoning is believed to develop upon repeated exposure. This phenomenon, however, is not well documented in the scientific literature.

Effects due to accidental ingestion of inorganic arsenicals, mainly arsenic trioxide, have been described in the literature. However, such incidents are rare in industry today. Cases of poisoning are characterized by profound gastrointestinal damage, resulting in severe vomiting and diarrhoea, which may result in shock and subsequent oliguria and albuminuria. Other acute symptoms are facial oedema, muscular cramps and cardiac abnormalities. Symptoms may occur within a few minutes following exposure to the poison in solution, but may be delayed for several hours if the arsenic compound is in solid form or if it is taken with a meal. When ingested as a particulate, toxicity is also dependent on solubility and particle size of the ingested compound. The fatal dose of ingested arsenic trioxide has been reported to range from 70 to 180 mg. Death may occur within 24 hours, but the usual course runs from 3 to 7 days. Acute intoxication with arsenic compounds is usually accompanied by anaemia and leucopenia, especially granulocytopenia. In survivors these effects are usually reversible within 2 to 3 weeks. Reversible enlargement of the liver is also seen in acute poisoning, but liver function tests and liver enzymes are usually normal.

In individuals surviving acute poisoning, peripheral nervous disturbances frequently develop a few weeks after ingestion.

Long-term exposure (chronic poisoning)

General aspects. Chronic arsenic poisoning may occur in workers exposed for a long time to excessive concentrations of airborne arsenic compounds. Local effects in the mucous membranes of the respiratory tract and the skin are prominent features. Involvement of the nervous and circulatory system and the liver may also occur, as well as cancer of the respiratory tract.

With long-term exposure to arsenic via ingestion in food, drinking water or medication, symptoms are partly different from those after inhalation exposure. Vague abdominal symptoms—diarrhoea or constipation, flushing of the skin, pigmentation and hyperkeratosis—dominate the clinical picture. In addition, there may be vascular involvement, reported in one area to have given rise to peripheral gangrene.

Anaemia and leucocytopenia often occur in chronic arsenic poisoning. Liver involvement has been more commonly seen in persons exposed for a long time via oral ingestion than in those exposed via inhalation, particularly in vineyard workers considered to have been exposed mainly through drinking contaminated wine. Skin cancer occurs with excess frequency in this type of poisoning.

Vascular disorders. Long-term oral exposure to inorganic arsenic via drinking water may give rise to peripheral vascular disorders with Raynaud's phenomenon. In one area of Taiwan, China, peripheral gangrene (so-called Blackfoot disease) has occurred. Such severe manifestations of peripheral vascular involvement have not been observed in occupationally exposed persons, but slight changes with Raynaud's phenomenon and an increased prevalence of low peripheral blood presssure on cooling have been found in workers exposed for a long time to airborne inorganic arsenic (doses of absorbed arsenic are given below.

Dermatological disorders. Arsenical skin lesions differ somewhat, depending on the type of exposure. Eczematoid symptoms of varying degrees of severity do occur. In occupational exposure to mainly airborne arsenic, skin lesions may result from local irritation. Two types of dermatological disorders may occur:

1. an eczematous type with erythema (redness), swelling and papules or vesicles
2. a follicular type with erythema and follicular swelling or follicular pustules.

Dermatitis is primarily localized on the most heavily exposed areas, such as the face, back of the neck, forearms, wrists and hands. However, it may also occur on the scrotum, the inner surfaces of the thighs, the upper chest and back, the lower legs and around the ankles. Hyperpigmentation and keratoses are not prominent features of this type of arsenical lesions. Patch tests have demonstrated that the dermatitis is due to arsenic, not to impurities present in the crude arsenic trioxide. Chronic dermal lesions may follow this type of initial reaction, depending on the concentration and duration of exposure. These chronic lesions may occur after many years of occupational or environmental exposure. Hyperkeratosis, warts and melanosis of the skin are the conspicuous signs. Melanosis is most commonly seen on the upper and lower eyelids, around the temples, on the neck, on the areolae of the nipples and in the folds of the axillae. In severe cases arsenomelanosis is observed on the abdomen, chest, back and scrotum, along with hyperkeratosis and warts. In chronic arsenic poisoning, depigmentation (i.e., leukoderma), especially on the pigmented areas, commonly called "raindrop" pigmentation, also occurs. These chronic skin lesions, particularly the hyperkeratoses, may develop into pre-cancerous and cancerous lesions. A transverse striation of the nails (so-called Mees lines) also occurs in chronic arsenical poisoning. It should be noted that the chronic skin lesions may develop long after cessation of exposure, when arsenic concentrations in skin have returned to normal.

Mucous membrane lesions in chronic arsenic exposure is most classically reported as perforation of the nasal septum after inhalation exposure. This lesion is a result of irritation of the mucous membranes of the nose. Such irritation also extends to the larynx, trachea and bronchi. Both in inhalation exposure and in poisoning caused by repeated ingestion, dermatitis of the face and eyelids sometimes extends to keratoconjunctivitis.

Peripheral neuropathy. Peripheral nervous disturbances are frequently encountered in survivors of acute poisoning. They usually start within a few weeks after the acute poisoning, and recovery is slow. The neuropathy is characterized by both motor dysfunction and paresthaesia, but in less severe cases only sensory unilateral neuropathy may occur. Often the lower extremities are more affected than the upper

ones. In subjects recovering from arsenical poisoning, Mees lines of the fingernails may develop. Histological examination has revealed Wallerian degeneration, especially in the longer axons. Peripheral neuropathy also may occur in industrial arsenic exposure, in most cases in a subclinical form that can be detected only by neurophysiological methods. In a group of smelter workers with long-term exposure corresponding to a mean cumulative total absorption of approximately 5 g (maximal absorption of 20 g), there was a negative correlation between cumulative absorption of arsenic and nerve conduction velocity. There were also some light clinical manifestations of peripheral vascular involvement in these workers (see above). In children exposed to arsenic, hearing loss has been reported.

Carcinogenic effects. Inorganic arsenic compounds are classified by the International Agency for Research on Cancer (IARC) as lung and skin carcinogens. There is also some evidence to suggest that persons exposed to inorganic arsenic compounds suffer a higher incidence of angiosarcoma of the liver and possibly of stomach cancer. Cancer of the respiratory tract has been reported in excess frequency among workers engaged in the production of insecticides containing lead arsenate and calcium arsenate, in vine-growers spraying insecticides containing inorganic copper and arsenic compounds, and in smelter workers exposed to inorganic compounds of arsenic and a number of other metals. The latency time between onset of exposure and the appearance of cancer is long, usually between 15 and 30 years. A synergistic action of tobacco smoking has been demonstrated for lung cancer.

Long-term exposure to inorganic arsenic via drinking water has been associated with an increased incidence of skin cancer in Taiwan and in Chile. This increase has been shown to be related to concentration in drinking water.

Teratogenic effects. High doses of trivalent inorganic arsenic compounds may cause malformations in hamsters when injected intravenously. With regard to human beings there is no firm evidence that arsenic compounds cause malformations under industrial conditions. Some evidence, however, suggests such an effect in workers in a smelting environment who were exposed simultaneously also to a number of other metals as well as other compounds.

Organic arsenic compounds

Organic arsenicals used as pesticides or as drugs may also give rise to toxicity, although such adverse effects are incompletely documented in humans.

Toxic effects on the nervous system have been reported in experimental animals following feeding with high doses of arsanilic acid, which is commonly used as a feed additive in poultry and swine.

The organic arsenic compounds that occur in foodstuffs of marine origin, such as shrimp, crab and fish, are made up of arsinocholine and arsinobetaine. It is well known that the amounts of organic arsenic that are present in fish and shellfish can be consumed without ill effects. These compounds are quickly excreted, mainly via urine.

Arsine gas and the substituted arsines. Many cases of acute arsine poisoning have been recorded, and there is a high fatality rate. Arsine is one of the most powerful haemolytic agents found in industry. Its haemolytic activity is due to its ability to cause a fall in erythrocyte-reduced glutathion content.

Signs and symptoms of arsine poisoning include haemolysis, which develops after a latent period that is dependent on the intensity of exposure. Inhalation of 250 ppm of arsine gas is instantly lethal. Exposure to 25 to 50 ppm for 30 minutes is lethal, and 10 ppm may be lethal after longer exposures. The signs and symptoms of poisoning are those characteristic of an acute and massive haemolysis. Initially there is a painless haemoglobinuria, gastrointestinal disturbance such as nausea and possibly vomiting. There may also be abdominal cramps and tenderness. Jaundice accompanied by anuria and oliguria subsequently occurs. Evidence of bone marrow depression may be present. After acute and severe exposure, a peripheral neuropathy may develop and can still be present several months after poisoning. Little is known about repeated or chronic exposure to arsine, but since the arsine gas is metabolized to inorganic arsenic in the body, it can be assumed that there is a risk for symptoms similar to those in long-term exposure to inorganic arsenic compounds.

The differential diagnosis should take account of acute haemolytic anaemias that could be caused by other chemical agents such as stibine or drugs, and secondary immunohaemolytic anaemias.

The substituted arsines do not give rise to haemolysis as their main effect, but they act as powerful local and pulmonary irritants and systemic poisons. The local effect on the skin gives rise to sharply circumscribed blisters in the case of dichloro(2-chlorovinyl-)arsine (lewisite). The vapour induces marked spasmodic coughing with frowzy or blood-stained sputum, progressing to acute pulmonary oedema. Dimercaprol (BAL) is an effective antidote if given in the early stages of poisoning.

Safety and Health Measures

The most common type of occupational arsenic exposure is to inorganic arsenic compounds, and these safety and health measures are mainly related to such exposures. When there is a risk of exposure to arsine gas, particular attention needs to be paid to accidental leaks, since peak exposures for short intervals may be of special concern.

The best means of prevention is to keep exposure well below accepted exposure limits. A programme of measurement of air-concentrations of arsenic is thus of importance. In addition to inhalation exposure, oral exposure via contaminated clothes, hands, tobacco and so on should be watched, and biological monitoring of inorganic arsenic in urine may be useful for evaluation of absorbed doses. Workers should be supplied with suitable protective clothing, protective boots and, when there is a risk that the exposure limit for airborne arsenic will be exceeded, respiratory protective equipment. Lockers should be provided with separate compartments for work and personal clothes, and adjacent sanitary facilities of a high standard should be made available. Smoking, eating and drinking at the workplace should not be allowed. Pre-employment medical examinations should be carried out. It is not recommended to employ persons with pre-existing diabetes, cardiovascular diseases, anaemia, allergic or other skin diseases, neurologic, hepatic or renal lesions, in arsenic work. Periodic medical examinations of all arsenic-exposed employees should be performed with special attention to possible arsenic-related symptoms.

Determination of the level of inorganic arsenic and its metabolites in urine allows estimation of the total dose of inorganic arsenic taken up by various exposure routes. Only when inorganic arsenic and

its metabolites can be specifically measured is this method useful. Total arsenic in urine may often give erroneous information about industrial exposure, since even a single meal of fish or other marine organisms (containing considerable amounts of non-toxic organic arsenic compound) may cause greatly elevated urinary arsenic concentrations for several days.

Treatment

Arsine gas poisoning. When there is reason to believe that there has been considerable exposure to arsine gas, or upon observation of the first symptoms (e.g., haemoglobinuria and abdominal pain), immediate removal of the individual from the contaminated environment and prompt medical attention are required. The recommended treatment, if there is any evidence of impaired renal function, consists of total-replacement blood transfusion associated with prolonged artificial dialysis. Forced diuresis has proved useful in some cases, whereas, in the opinion of most authors, treatment with BAL or other chelating agents seems to have only limited effect.

Exposure to the substituted arsines should be treated in the same way as inorganic arsenic poisoning (see below).

Poisoning by inorganic arsenic. If there has been exposure to doses that can be estimated to give rise to acute poisoning, or if severe symptoms from the respiratory system, the skin or the gastrointestinal tract occur in the course of long-term exposures, the worker should immediately be removed from exposure and treated with a complexing agent.

The classical agent which has been used most widely in such situations is 2,3-dimercapto-1-propanol or British anti-lewisite (BAL, dimercaprol). Prompt administration in such cases is vital: to obtain maximal benefit such treatment should be given within 4 hours of poisoning. Other pharmaceuticals which may be used are sodium 2,3-dimercaptopropanesulphonate (DMPS or unithiol) or meso-2,3-dimercaptosuccinic acid (DMSA). These drugs are less likely to give side effects and are believed to be more effective than BAL. Intravenous administration of N-acetylcysteine has been reported in one case to be of value; in addition, general treatment, such as prevention of further absorption by removal from exposure and minimizing absorption from the gastrointestinal tract by gastric lavage and administration by gastric tube of chelating agents or charcoal, is mandatory. General supportive therapy, such as maintenance of respiration and circulation, maintenance of water and electrolyte balance, and control of nervous system effects, as well as elimination of absorbed poison through haemodialysis and exchange transfusion, may be used if feasible.

Acute skin lesions such as contact dermatitis and mild manifestations of peripheral vascular involvement, such as Raynaud’s syndrome, usually do not require treatment other than removal from exposure.

BARIUM

Occurrence and Uses

Barium (Ba) is abundant in nature and accounts for approximately 0.04% of the earth's crust. The chief sources are the minerals barite (barium sulphate, $BaSO_4$) and witherite (barium carbonate, $BaCO_3$).

Barium metal is produced in only limited quantities, by aluminium reduction of barium oxide in a retort.

Barium is used extensively in the manufacture of alloys for nickel barium parts found in ignition equipment for automobiles and in the manufacture of glass, ceramics and television picture tubes. Barite ($BaSO_4$), or barium sulphate, is primarily used in the manufacture of lithopone, a white powder containing 20% barium sulphate, 30% zinc sulphide and less than 8% zinc oxide. Lithopone is widely employed as a pigment in white paints. Chemically precipitated barium sulphate—blanc fixe—is used in high-quality paints, in x-ray diagnostic work and in the glass and paper industries. It is also used in the manufacture of photographic papers, artificial ivory and cellophane. Crude barite is used as a thixotropic mud in oil-well drilling.

Barium hydroxide ($Ba(OH)_2$) is found in lubricants, pesticides, the sugar industry, corrosion inhibitors, drilling fluids and water softeners. It is also used in glass manufacture, synthetic rubber vulcanization, animal and vegetable oil refining, and fresco painting. Barium carbonate ($BaCO_3$) is obtained as a precipitate of barite and is used in the brick, ceramics, paint, rubber, oil-well drilling and paper industries. It also finds use in enamels, marble substitutes, optical glass and electrodes.

Barium oxide (BaO) is a white alkaline powder which is used to dry gases and solvents. At 450°C it combines with oxygen to produce barium peroxide (BaO_2), an oxidizing agent in organic synthesis and a bleaching material for animal substances and vegetable fibres. Barium peroxide is used in the textile industry for dyeing and printing, in powder aluminium for welding and in pyrotechnics.

Barium chloride ($BaCl_2$) is obtained by roasting barite with coal and calcium chloride, and is used in the manufacture of pigments, colour lakes and glass, and as a mordant for acid dyes. It is also useful for weighting and dyeing textile fabrics and in aluminium refining. Barium chloride is a pesticide, a compound added to boilers for softening water, and a tanning and finishing agent for leather. Barium nitrate ($Ba(NO_3)_2$) is used in pyrotechnics and the electronics industries.

Hazards

Barium metal has only limited use and presents an explosion hazard. The soluble compounds of barium (chloride, nitrate, hydroxide) are highly toxic; the inhalation of the insoluble compounds (sulphate) may give rise to pneumoconiosis. Many of the compounds, including the sulphide, oxide and carbonate, may cause local irritation to the eyes, nose, throat and skin. Certain compounds, particularly the peroxide, nitrate and chlorate, present fire hazards in use and storage.

Toxicity

When the soluble compounds enter by the oral route they are highly toxic, with a fatal dose of the chloride thought to be 0.8 to 0.9 g. However, although poisoning due to the ingestion of these compounds does occasionally occur, very few cases of industrial poisoning have been reported. Poisoning may result when workers are exposed to atmospheric concentrations of the dust of soluble compounds such as may occur during grinding. These compounds exert a strong and prolonged stimulant action on all forms of muscle, markedly increasing contractility. In the heart, irregular contractions may be followed by fibrillation, and there is evidence of a coronary constrictor action. Other effects include intestinal peristalsis, vascular constriction, bladder contraction and an increase in voluntary muscle tension. Barium compounds also have irritant effects on mucous membranes and the eye.

Barium carbonate, an insoluble compound, does not appear to have pathological effects from inhalation; however, it can cause severe poisoning from oral intake, and in rats it impairs the function of the male and female gonads; the foetus is sensitive to barium carbonate during the first half of pregnancy.

Pneumoconiosis

Barium sulphate is characterized by its extreme insolubility, a property which makes it non-toxic to humans. For this reason and due to its high radio-opacity, barium sulphate is used as an opaque medium in x-ray examination of the gastrointestinal, respiratory and urinary systems. It is also inert in the human lung, as has been demonstrated by its lack of adverse effects following deliberate introduction into the bronchial tract as a contrast medium in bronchography and by industrial exposure to high concentrations of fine dust.

Inhalation, however, may lead to deposition in the lungs in sufficient quantities to produce baritosis (a benign pneumoconiosis, which principally occurs in the mining, grinding and bagging of barite, but has been reported in the manufacture of lithopone). The first reported case of baritosis was accompanied by symptoms and disability, but these were associated later with other lung disease. Subsequent studies have contrasted the unimpressive nature of the clinical picture and the total absence of symptoms and abnormal physical signs with the well marked x-ray changes, which show disseminated nodular opacities throughout both lungs. The opacities are discrete but sometimes so numerous as to overlap and appear confluent. No massive shadows have been reported. The outstanding feature of the radiographs is the marked radio-opacity of the nodules, which is understandable in view of the substance’s use as a radio-opaque medium. The size of the individual elements may vary between 1 and 5 mm in diameter, although the average is about 3 mm or less, and the shape has been described variously as “rounded” and “dendritic”. In some cases, a number of very dense points have been found to lie in a matrix of lower density.

In one series of cases, dust concentrations of up to 11,000 particles/cm^3 were measured at the workplace, and chemical analysis showed that the total silica content lay between 0.07 and 1.96%, quartz not being detectable by x-ray diffraction. Men exposed for up to 20 years and exhibiting x-ray

changes were symptomless, had excellent lung function and were capable of carrying out strenuous work. Years after the exposure has ceased, follow-up examinations show a marked clearing of x-ray abnormalities.

Reports of post-mortem findings in pure baritosis are practically non-existent. However, baritosis may be associated with silicosis in mining due to contamination of barite ore by siliceous rock, and, in grinding, if siliceous millstones are used.

Safety and Health Measures

Adequate washing and other sanitary facilities should be provided for workers exposed to toxic soluble barium compounds, and rigorous personal hygiene measures should be encouraged. Smoking and consumption of food and beverages in workshops should be prohibited. Floors in workshops should be made of impermeable materials and frequently washed down. Employees working on such processes as barite leaching with sulphuric acid should be supplied with acid-resistant clothing and suitable hand and face protection. Although baritosis is benign, efforts should still be made to reduce atmospheric concentrations of barite dust to a minimum. In addition, particular attention should be paid to the presence of free silica in the airborne dust.

BISMUTH

Occurrence and Uses

In nature, bismuth (Bi) occurs both as the free metal and in ores such as bismutite (carbonate) and bismuthinite (double bismuth and tellurium sulphide), where it is accompanied by other elements, mainly lead and antimony.

Bismuth is used in metallurgy for the manufacture of numerous alloys, especially alloys with a low melting point. Some of these alloys are used for welding. Bismuth also finds use in safety devices in fire detection and extinguishing systems, and in the production of malleable irons. It acts as a catalyst for making acrylic fibres.

Bismuth telluride is used as a semiconductor. Bismuth oxide, hydroxide, oxychloride, trichloride and nitrate are employed in the cosmetics industry. Other salts (e.g., succinate, orthoxyquinoleate, subnitrate, carbonate, phosphate and so on) are used in medicine.

Hazards

There have been no reports of occupational exposure during the production of metallic bismuth and the manufacture of pharmaceuticals, cosmetics and industrial chemicals. Because bismuth and its compounds do not appear to have been responsible for poisoning associated with work, they are regarded as the least toxic of the heavy metals currently used in industry.

Bismuth compounds are absorbed through the respiratory and gastrointestinal tracts. The main systemic effects in humans and animals are exerted in the kidney and liver. The organic derivatives cause alterations of the convoluted tubules and may result in serious, and sometimes fatal, nephrosis.

Gum discolouration has been reported with exposure to bismuth dusts. The insoluble mineral salts, taken orally over prolonged periods in doses generally exceeding 1 per day, may provoke brain disease characterized by mental disorders (confused state), muscular disorders (myoclonia), motor coordination disorders (loss of balance, unsteadiness) and dysarthria. These disorders stem from an accumulation of bismuth in the nerve centres which manifests itself when bismuthaemia exceeds a certain level, estimated at around 50 mg/l. In most cases, bismuth-linked encephalopathy gradually disappears without medication within a period of from 10 days to 2 months, during which time the bismuth is eliminated in the urine. Fatal cases of encephalopathy have, however, been recorded.

Such effects have been observed in France and Australia since 1973. They are caused by a factor not yet fully investigated which encourages the absorption of bismuth through the intestinal mucous membrane and leads to an increase in bismuthaemia to a level as high as several hundred mg/l. The danger of encephalopathy caused by inhaling metallic dust or oxide smoke in the workplace is very remote. The poor solubility of bismuth and bismuth oxide in blood plasma and its fairly rapid elimination in the urine (its half-life is about 6 days) argue against the likelihood of a sufficiently acute impregnation of the nerve centres to reach pathological levels.

In animals, inhalation of insoluble compounds such as bismuth telluride provokes the usual lung response of an inert dust. However, long-term exposure to bismuth telluride “doped” with selenium sulphide can produce in various species a mild reversible granulomatous reaction of the lung.

Some bismuth compounds decompose into dangerous chemicals. Bismuth pentafluoride decomposes on heating and emits highly toxic fumes.

CADMIUM

Occurrence and Uses

Cadmium (Cd) has many chemical and physical similarities to zinc and occurs together with zinc in nature. In minerals and ores, cadmium and zinc generally have a ratio of 1:100 to 1:1,000.

Cadmium is highly resistant to corrosion and has been widely used for electroplating of other metals, mainly steel and iron. Screws, screw nuts, locks and various parts for aircraft and motor vehicles are frequently treated with cadmium in order to withstand corrosion. Nowadays, however, only 8% of all refined cadmium is used for platings and coatings. Cadmium compounds (30% of the use in developed countries) are used as pigments and stabilizers in plastics, and cadmium is also used in certain alloys (3%). Rechargeable, small portable cadmium-containing batteries, used, for example, in mobile telephones, comprise a rapidly increasing usage of cadmium (55% of all cadmium in industrialized countries in 1994 was used in batteries).

Cadmium occurs in various inorganic salts. The most important is cadmium stearate, which is used as a heat stabilizer in polyvinyl chloride (PVC) plastics. Cadmium sulphide and cadmium sulphoselenide are used as yellow and red pigments in plastics and colours. Cadmium sulphide is also used in photo- and solar cells. Cadmium chloride acts as a fungicide, an ingredient in elecroplating baths, a colourant for pyrotechnics, an additive to tinning solution and a mordant in dyeing and printing textiles. It is also

used in the production of certain photographic films and in the manufacture of special mirrors and coatings for electronic vacuum tubes. Cadmium oxide is an elecroplating agent, a starting material for PVC heat stabilizers and a component of silver alloys, phosphors, semiconductors and glass and ceramic glazes.

Cadmium can represent an environmental hazard, and many countries have introduced legislative actions aimed towards decreasing the use and subsequent environmental spread of cadmium.

Metabolism and accumulation

Gastrointestinal absorption of ingested cadmium is about 2 to 6% under normal conditions. Individuals with low body iron stores, reflected by low concentrations of serum ferritin, may have considerably higher absorption of cadmium, up to 20% of a given dose of cadmium. Significant amounts of cadmium may also be absorbed via the lung from the inhalation of tobacco smoke or from occupational exposure to atmospheric cadmium dust. Pulmonary absorption of inhaled respirable cadmium dust is estimated at 20 to 50%. After absorption via the gastrointestinal tract or the lung, cadmium is transported to the liver, where production of a cadmium-binding low-molecular-weight protein, metallothionein, is initiated.

About 80 to 90% of the total amount of cadmium in the body is considered to be bound to metallothionein. This prevents the free cadmium ions from exerting their toxic effects. It is likely that small amounts of metallothionein-bound cadmium are constantly leaving the liver and being transported to the kidney via the blood. The metallothionein with the cadmium bound to it is filtered through the glomeruli into the primary urine. Like other low-molecular-weight proteins and amino acids, the metallothionein-cadmium complex is subsequently reabsorbed from the primary urine into the proximal tubular cells, where digestive enzymes degrade the engulfed proteins into smaller peptides and amino acids. Free cadmium ions in the cells result from degradation of metallothionein and initiate a new synthesis of metallothionein, binding the cadmium, and thus protecting the cell from the highly toxic free cadmium ions. Kidney dysfunction is considered to occur when the metallothionein-producing capacity of the tubular cells is exceeded. The kidney and liver have the highest concentrations of cadmium, together containing about 50% of the body burden of cadmium. The cadmium concentration in the kidney cortex, before cadmium-induced kidney damage occurs, is generally about 15 times the concentration in liver. Elimination of cadmium is very slow. As a result of this, cadmium accumulates in the body, the concentrations increasing with age and length of exposure. Based on organ concentration at different ages the biological half-life of cadmium in humans has been estimated in the range of 7 to 30 years.

Acute toxicity

Inhalation of cadmium compounds at concentrations above 1 mg Cd/m^3 in air for 8 hours, or at higher concentrations for shorter periods, may lead to chemical pneumonitis, and in severe cases pulmonary oedema. Symptoms generally occur within 1 to 8 hours after exposure. They are influenza-like and similar to those in metal fume fever. The more severe symptoms of chemical pneumonitis and pulmonary oedema may have a latency period up to 24 hours. Death may occur after 4 to 7 days.

Exposure to cadmium in the air at concentrations exceeding 5 mg Cd/m^3 is most likely to occur where cadmium alloys are smelted, welded or soldered. Ingestion of drinks contaminated with cadmium at concentrations exceeding 15 mg Cd/l gives rise to symptoms of food poisoning. Symptoms are nausea, vomiting, abdominal pains and sometimes diarrhoea. Sources of food contamination may be pots and pans with cadmium-containing glazing and cadmium solderings used in vending machines for hot and cold drinks. In animals parenteral administration of cadmium at doses exceeding 2 mg Cd/kg body weight causes necrosis of the testis. No such effect has been reported in humans.

Chronic toxicity

Chronic cadmium poisoning has been reported after prolonged occupational exposure to cadmium oxide fumes, cadmium oxide dust and cadmium stearates. Changes associated with chronic cadmium poisoning may be local, in which case they involve the respiratory tract, or they may be systemic, resulting from absorption of cadmium. Systemic changes include kidney damage with proteinuria and anaemia. Lung disease in the form of emphysema is the main symptom at heavy exposure to cadmium in air, whereas kidney dysfunction and damage are the most prominent findings after long-term exposure to lower levels of cadmium in workroom air or via cadmium-contaminated food. Mild hypochromic anaemia is frequently found among workers exposed to high levels of cadmium. This may be due to both increased destruction of red blood cells and to iron deficiency. Yellow discolouration of the necks of teeth and loss of sense of smell (anosmia) may also be seen in cases of exposure to very high cadmium concentrations.

Pulmonary emphysema is considered a possible effect of prolonged exposure to cadmium in air at concentrations exceeding 0.1 mg Cd/m^3. It has been reported that exposure to concentrations of about 0.02 mg Cd/m^3 for more than 20 years can cause certain pulmonary effects. Cadmium-induced pulmonary emphysema can reduce working capacity and may be the cause of invalidity and life shortening. With long-term low-level cadmium exposure the kidney is the critical organ (i.e., the organ first affected). Cadmium accumulates in renal cortex. Concentrations exceeding 200 µg Cd/g wet weight have previously been estimated to cause tubular dysfunction with decreased reabsorption of proteins from the urine. This causes tubular proteinuria with increased excretion of low-molecular-weight proteins such as α,α-1-microglobulin (protein HC), β-2-microglobulin and retinol binding protein (RTB). Recent research suggests, however, that tubular damage may occur at lower levels of cadmium in kidney cortex. As the kidney dysfunction progresses, amino acids, glucose and minerals, such as calcium and phosphorus, are also lost into the urine. Increased excretion of calcium and phosphorous may disturb bone metabolism, and kidney stones are frequently reported by cadmium workers. After long-term medium-to-high levels of exposure to cadmium, the kidney's glomeruli may also be affected, leading to a decreased glomerular filtration rate. In severe cases uraemia may develop. Recent studies have shown the glomerular dysfunction to be irreversible and dose dependent. Osteomalacia has been reported in cases of severe chronic cadmium poisoning.

In order to prevent kidney dysfunction, as manifested by β-2-microglobulinuria, particularly if the occupational exposure to cadmium fumes and dust is likely to last for 25 years (at 8 hours workday and

225 workdays/year), it is recommended that the average workroom concentration of respirable cadmium should be kept below 0.01 mg/m^3.

Excessive cadmium exposure has occurred in the general population through ingestion of contaminated rice and other foodstuffs, and possibly drinking water. The itai-itai disease, a painful type of osteomalacia, with multiple fractures appearing together with kidney dysfunction, has occurred in Japan in areas with high cadmium exposure. Though the pathogenesis of itai-itai disease is still under dispute, it is generally accepted that cadmium is a necessary aetiological factor. It should be stressed that cadmium-induced kidney damage is irreversible and may grow worse even after exposure has ceased.

Cadmium and cancer

There is strong evidence of dose-response relationships and an increased mortality from lung cancer in several epidemiological studies on cadmium-exposed workers. The interpretation is complicated by concurrent exposures to other metals which are known or suspected carcinogens. Continuing observations of cadmium-exposed workers have, however, failed to yield evidence of increased mortality from prostatic cancer, as initially suspected. The IARC in 1993 assessed the risk of cancer from exposure to cadmium and concluded that it should be regarded as a human carcinogen. Since then additional epidemiological evidence has come forth with somewhat contradictory results, and the possible carcinogenicity of cadmium thus remains unclear. It is nevertheless clear that cadmium possesses strong carcinogenic properties in animal experiments.

Safety and Health Measures

The kidney cortex is the critical organ with long-term cadmium exposure via air or food. The critical concentration is estimated at about 200 µg Cd/g wet weight, but may be lower, as stated above. In order to keep the kidney cortex concentration below this level even after lifelong exposure, the average cadmium concentration in workroom air (8 hours per day) should not exceed 0.01 mg Cd/m^3.

Work processes and operations which may release cadmium fumes or dust into the atmosphere should be designed to keep concentration levels to a minimum and, if practicable, be enclosed and fitted with exhaust ventilation. When adequate ventilation is impossible to maintain (e.g., during welding and cutting), respirators should be carried and air should be sampled to determine the cadmium concentration. In areas with hazards of flying particles, chemical splashes, radiant heat and so on (e.g., near electroplating tanks and furnaces), workers should wear appropriate safety equipment, such as eye, face, hand and arm protection and impermeable clothing. Adequate sanitary facilities should be supplied, and workers should be encouraged to wash before meals and to wash thoroughly and change clothes before leaving work. Smoking, eating and drinking in work areas should be prohibited. Tobacco contaminated with cadmium dust from workrooms can be an important exposure route. Cigarettes and pipe tobacco should not be carried in the workroom. Contaminated exhaust air should be filtered, and persons in charge of dust collectors and filters should wear respirators while working on the equipment.

To ensure that excessive accumulation of cadmium in the kidney does not occur, cadmium levels in blood and in urine should be checked regularly. Cadmium levels in blood are mainly an indication of the last few months exposure, but can be used to assess body burden a few years after exposure has ceased. A value of 100 nmol Cd/l whole blood is an approximate critical level if exposure is regular for long periods. Cadmium values in urine can be used to estimate the cadmium body burden, providing kidney damage has not occurred. It has been estimated by the WHO that 10 nmol/mmol creatinine is the concentration below which kidney dysfunction should not occur. Recent research has, however, shown that kidney dysfunction may occur already at around 5 nmol/mmol creatinine. Since the mentioned blood and urinary levels are levels at which action of cadmium on kidney has been observed, it is recommended that control measures be applied whenever the individual concentrations of cadmium in urine and/or in blood exceed 50 nmol/l whole blood or 3 nmol/mmol creatinine respectively. Pre-employment medical examinations should be given to workers who will be exposed to cadmium dust or fumes. Persons with respiratory or kidney disorders should avoid such work. Medical examination of cadmium-exposed workers should be carried out at least once every year. In workers exposed to cadmium for longer periods, quantitative measurements of ß-2-microglobulin or other relevant low-molecular-weight proteins in urine should be made regularly. Concentrations of ß-2-microglobulin in urine should normally not exceed 34 µg/mmol creatinine.

Treatment of cadmium poisoning

Persons who have ingested cadmium salts should be made to vomit or given gastric lavage; persons exposed to acute inhalation should be removed from exposure and given oxygen therapy if necessary. No specific treatment for chronic cadmium poisoning is available, and symptomatic treatment has to be relied upon. As a rule the administration of chelating agents such as BAL and EDTA is contraindicated since they are nephrotoxic in combination with cadmium.

CHROMIUM

Occurrence and Uses

Elemental chromium (Cr) is not found free in nature, and the only ore of any importance is the spinel ore, chromite or chrome iron stone, which is ferrous chromite ($FeOCr_2O_3$), widely distributed over the earth's surface. In addition to chromic acid, this ore contains variable quantities of other substances. Only ores or concentrates containing more than 40% chromic oxide (Cr_2O_3) are used commercially, and countries having the most suitable deposits are the Russian Federation, South Africa, Zimbabwe, Turkey, the Philippines and India. The prime consumers of chromites are the United States, the Russian Federation, Germany, Japan, France and the United Kingdom.

Chromite may be obtained from both underground and open cast mines. The ore is crusted and, if necessary, concentrated.

The most significant usage of pure chromium is for electroplating of a wide range of equipment, such as automobile parts and electric equipment. Chromium is used extensively for alloying with iron and

nickel to form stainless steel, and with nickel, titanium, niobium, cobalt, copper and other metals to form special-purpose alloys.

Chromium Compounds

Chromium forms a number of compounds in various oxidation states. Those of II (chromous), III (chromic) and VI (chromate) states are most important; the II state is basic, the III state is amphoteric and the VI state is acidic. Commercial applications mainly concern compounds in the VI state, with some interest in III state chromium compounds.

The chromous state (Cr^{II}) is unstable and is readily oxidized to the chromic state (Cr^{III}). This instability limits the use of chromous compounds. The chromic compounds are very stable and form many compounds which have commercial use, the principal of which are chromic oxide and basic chromium sulphate.

Chromium in the +6 oxidation state (Cr^{VI}) has its greatest industrial application as a consequence of its acidic and oxidant properties, as well as its ability to form strongly coloured and insoluble salts. The most important compounds containing chromium in the Cr^{VI} state are sodium dichromate, potassium dichromate and chromium trioxide. Most other chromate compounds are produced industrially using dichromate as the source of Cr^{VI}.

Production

Sodium mono- and dichromate are the starting materials from which most of the chromium compounds are manufactured. Sodium chromate and dichromate are prepared directly from chrome ore. Chrome ore is crushed, dried and ground; soda ash is added and lime or leached calcine may also be added. After thorough mixing the mixture is roasted in a rotary furnace at an optimum temperature of about 1,100°C; an oxidizing atmosphere is essential to convert the chromium to the Cr^{VI} state. The melt from the furnace is cooled and leached and the sodium chromate or dichromate is isolated by conventional processes from the solution.

Chromium^{III} compounds

Technically, chromium oxide (Cr_2O_3, or chromic oxide), is made by reducing sodium dichromate either with charcoal or with sulphur. Reduction with sulphur is usually employed when the chromic oxide is to be used as a pigment. For metallurgical purposes carbon reduction is normally employed.

The commercial material is normally basic chromic sulphate $[Cr(OH)(H_2O)_5]SO_4$, which is prepared from sodium dichromate by reduction with carbohydrate in the presence of sulphuric acid; the reaction is vigorously exothermic. Alternatively, sulphur dioxide reduction of a solution of sodium dichromate will yield basic chromic sulphurate. It is used in the tanning of leather, and the material is sold on the basis of Cr_2O_3 content, which ranges from 20.5 to 25%.

Chromium^{VI} compounds

Sodium dichromate can be converted into the anhydrous salt. It is the starting point for preparation of chromium compounds.

Chromium trioxide or chromium anhydride (sometimes referred to as “chromic acid”, although true chromic acid cannot be isolated from solution) is formed by treating a concentrated solution of a dichromate with strong sulphuric acid excess. It is a violent oxidizing agent, and the solution is the principal constituent of chromium plating.

Insoluble chromates

Chromates of weak bases are of limited solubility and more deeply coloured than the oxides; hence their use as pigments. These are not always distinct compounds and may contain mixtures of other materials to provide the right pigment colour. They are prepared by the addition of sodium or potassium dichromate to a solution of the appropriate salt.

Lead chromate is trimorphic; the stable monoclinic form is orange-yellow, “chrome yellow”, and the unstable orthombic form is yellow, isomorphous with lead sulphate and stabilized by it. An orange-red tetragonal form is similar and isomorphous with lead molybdate (VI) $PbMoO_4$ and stabilized by it. On these properties depends the versatility of lead chromate as a pigment in producing a variety of yellow-orange pigments.

Uses

Compounds containing Cr^{VI} are used in many industrial operations. The manufacture of important inorganic pigments such as lead chromes (which are themselves used to prepare chrome greens), molybdate-oranges, zinc chromate and chromium-oxide green; wood preservation; corrosion inhibition; and coloured glasses and glazes. Basic chromic sulphates are widely used for tanning.

The dyeing of textiles, the preparation of many important catalysts containing chromic oxide and the production of light-sensitive dichromated colloids for use in lithography are also well-known industrial uses of chromium-containing chemicals.

Chromic acid is used not only for “decorative” chromium plating but also for “hard” chromium plating, where it is deposited in much thicker layers to give an extremely hard surface with a low coefficient of friction.

Because of the strong oxidizing action of chromates in acid solution, there are many industrial applications particularly involving organic materials, such as the oxidation of trinitrotoluene (TNT) to give phloroglucinol and the oxidation of picoline to give nicotine acid.

Chromium oxide is also used for the production of pure chromium metal that is suitable for incorporation in creep-resistant, high-temperature alloys, and as a refractory oxide. It may be included in a number of refractory compositions with advantage—for example, in magnetite and magnetite-chromate mixtures.

Hazards

Compounds with Cr^{III} oxidation states are considerably less hazardous than are Cr^{VI} compounds. Compounds of Cr^{III} are poorly absorbed from the digestive system. These Cr^{III} compounds may also combine with proteins in the superficial layers of the skin to form stable complexes. Compounds of Cr^{III} do not cause chrome ulcerations and do not generally initiate allergic dermatitis without prior sensitization by Cr^{VI} compounds.

In the Cr^{VI} oxidation state, chromium compounds are readily absorbed after ingestion as well as during inhalation. The uptake through intact skin is less well elucidated. The irritant and corrosive effects caused by Cr^{VI} occur readily after uptake through mucous membranes, where they are readily absorbed. Work-related exposure to Cr^{VI} compounds may induce skin and mucous membrane irritation or corrosion, allergic skin reactions or skin ulcerations.

The untoward effects of chromium compounds generally occur among workers in workplaces where Cr^{VI} is encountered, in particular during manufacture or use. The effects frequently involve the skin or respiratory system. Typical industrial hazards are inhalation of the dust or fumes arising during the manufacture of dichromate from chromite ore and the manufacture of lead and zinc chromates, inhalation of chromic acid mists during electroplating or surface treatment of metals, and skin contact with Cr^{VI} compounds in manufacture or use. Exposure to Cr^{VI}-containing fumes may also occur during welding of stainless steels.

Chrome ulcerations. Such lesions used to be common after work-related exposure to Cr^{VI} compounds. The ulcers result from the corrosive action of Cr^{VI}, which penetrates the skin through cuts or abrasions. The lesion usually begins as a painless papule, commonly on the hands, forearms or feet, resulting in ulcerations. The ulcer may penetrate deeply into soft tissue and may reach underlying bone. Healing is slow unless the ulcer is treated at an early stage, and atrophic scars remain. There are no reports about skin cancer following such ulcers.

Dermatitis. The Cr^{VI} compounds may cause both primary skin irritation and sensitization. In chromate-producing industries, some workers may develop skin irritation, particularly at the neck or wrist, soon after starting work with chromates. In the majority of cases, this clears rapidly and does not recur. However, sometimes it may be necessary to recommend a change of work.

Numerous sources of exposure to Cr^{VI} have been listed (e.g., contact with cement, plaster, leather, graphic work, work in match factories, work in tanneries and various sources of metal work). Workers employed in wet sandpapering of car bodies have also been reported with allergy. Affected subjects react positively to patch testing with 0.5% dichromate. Some affected subjects had only erythema or

scattered papules, and in others the lesions resembled dyshidriotic pompholyx; nummular eczema may lead to misdiagnosis of genuine cases of occupational dermatitis.

It has been shown that Cr^{VI} penetrates the skin through the sweat glands and is reduced to Cr^{III} in the corium. It is shown that the Cr^{III} then reacts with protein to form the antigen-antibody complex. This explains the localization of lesions around sweat glands and why very small amounts of dichromate can cause sensitization. The chronic character of the dermatitis may be due to the fact that the antigen-antibody complex is removed more slowly than would be the case if the reaction occurred in the epidermis.

Acute respiratory effects. Inhalation of dust or mist containing Cr^{VI} is irritating to mucous membranes. At high concentrations of such dust, sneezing, rhinorrhoea, lesions of the nasal septum and redness of the throat are documented effects. Sensitization has also been reported, resulting in typical asthmatic attacks, which may recur on subsequent exposure. At exposure for several days to chromic acid mist at concentrations of about 20 to 30 mg/m^3, cough, headache, dyspnoea and substernal pain have also been reported after exposure. The occurrence of bronchospasm in a person working with chromates should suggest chemical irritation of the lungs. Treatment is only symptomatic.

Ulcerations of the nasal septum. In previous years, when the exposure levels to Cr^{VI} compounds could be high, ulcerations of the nasal septum were frequently seen among exposed workers. This untoward effect results from deposition of Cr^{VI}-containing particulates or mist droplets on the nasal septum, resulting in ulceration of the cartilaginous portion followed, in many cases, by perforation at the site of ulceration. Frequent nose-picking may enhance the formation of perforation. The mucosa covering the lower anterior part of the septum, known as the Kiesselbach’s and Little’s area, is relatively avascular and closely adherent to the underlying cartilage. Crusts containing necrotic debris from the cartilage of the septum continue to form, and within a week or two the septum becomes perforated. The periphery of the ulceration remains active for up to several months, during which time the perforation may increase in size. It heals by the formation of vascular scar tissue. Sense of smell is almost never impaired. During the active phase, rhinorrhoea and nose-bleeding may be troublesome symptoms. When soundly healed, symptoms are rare and many persons are unaware that the septum is perforated.

Effects in other organs. Necrosis of the kidneys has been reported, starting with tubular necrosis, leaving the glomeruli undamaged. Diffuse necrosis of the liver and subsequent loss of architecture has also been reported. Soon after the turn of the century there were a number of reports on human ingestion of Cr^{VI} compounds resulting in major gastro-intestinal bleeding from ulcerations of the intestinal mucosa. Sometimes such bleedings resulted in cardiovascular shock as a possible complication. If the patient survived, tubular necrosis of the kidneys or liver necrosis could occur.

Carcinogenic effects. Increased incidence of lung cancer among workers in manufacture and use of Cr^{VI} compounds has been reported in a great number of studies from France, Germany, Italy, Japan, Norway, the United States and the United Kingdom. Chromates of zinc and calcium appear to be

among the most potent carcinogenic chromates, as well as among the most potent human carcinogens. Elevated incidence of lung cancer has also been reported among subjects exposed to lead chromates, and to fumes of chromium trioxides. Heavy exposures to Cr^{VI} compounds have resulted in very high incidence of lung cancer in exposed workers 15 or more years after first exposure, as reported in both cohort studies and case reports.

Thus, it is well established that an increase in the incidence of lung cancer of workers employed in the manufacture of zinc chromate and the manufacture of mono- and dichromates from chromite ore is a long-term effect of work-related heavy exposure to Cr^{VI} compounds. Some of the cohort studies have reported measurements of exposure levels among the exposed cohorts. Also, a small number of studies have indicated that exposure to fumes generated from welding on Cr-alloyed steel may result in elevated incidence of lung cancer among these welders.

There is no firmly established “safe” level of exposure. However, most of the reports on association between Cr^{VI} exposure and cancer of the respiratory organs and exposure levels report on air levels exceeding 50 mg Cr^{VI}/m^3 air.

The symptoms, signs, course, x-ray appearance, method of diagnosis and prognosis of lung cancers resulting from exposure to chromates differ in no way from those of cancer of the lung due to other causes. It has been found that the tumours often originate in the periphery of the bronchial tree. The tumours may be of all histological types, but a majority of the tumours seem to be anaplastic oat-celled tumours. Water-soluble, acid soluble and water insoluble chromium is found in the lung tissues of chromate workers in varying amounts.

Although it has not been firmly established, some studies have indicated that exposure to chromates may result in increased risk of cancer in the nasal sinuses and the alimentary tract. The studies that indicate excess cancer of the alimentary tract are case reports from the 1930s or cohort studies that reflect exposure at high levels than generally encountered today.

Safety and Health Measures

On the technical side, avoidance of exposure to chromium depends on appropriate design of processes, including adequate exhaust ventilation and the suppression of dust or mist containing chromium in the hexavalent state. Built-in control measures are also necessary, requiring the least possible action by either process operators or maintenance staff.

Wet methods of cleaning should be used where possible; at other sites, the only acceptable alternative is vacuum cleaning. Spill of liquids or solids must be removed to prevent dispersion as airborne dust. The concentration in the work environment of chromium-containing dust and fumes should preferably be measured at regular intervals by individual and area sampling. Where unacceptable concentration levels are found by either method, the sources of dust or fumes should be identified and controlled. Dust masks, preferably with an efficiency of more than 99% in retaining particles of 0.5 μm size, should be worn in situations above non-hazardous levels, and it may be necessary to provide air-supplied

respiratory protective equipment for jobs considered to be hazardous. Management should ensure that dust deposits and other surface contaminants should be removed by washing down or suction before work of this type begins. Providing laundering overalls daily may help in avoiding skin contamination. Hand and eye protection is generally recommended, as is repair and replacement of all personal protective equipment (PPE).

The medical surveillance of workers on processes in which Cr^{VI} compounds may be encountered should include education in toxic and the carcinogenic properties of both Cr^{VI} and Cr^{III} compounds, as well as on the differences between the two groups of compounds. The nature of the exposure hazards and subsequent risks of various diseases (e.g., lung cancer) should be given at job entry as well as at regular intervals during employment. The need to observe a high standard of personal hygiene should be emphasized.

All untoward effects of exposure to chromium can be avoided. Chrome ulcers of the skin can be prevented by eliminating sources of contact and by preventing injury to the skin. Skin cuts and abrasions, however slight, should be cleaned immediately and treated with 10% sodium EDTA ointment. Together with the use of a frequently renewed impervious dressing, this will enhance rapid healing for any ulcer that may develop. Although EDTA does not chelate Cr^{VI} compounds at room temperature, it reduces the Cr^{VI} to Cr^{III} rapidly, and the excess EDTA chelates Cr^{III}. Both the direct irritant and corrosive action of Cr^{VI} compounds and the formation of protein/Cr^{III} complexes are thus prevented. After accidental ingestion of Cr^{VI} compounds, immediate swallowing of ascorbic acid may also quickly reduce the Cr^{VI}.

Careful washing of the skin after contact and care to avoid friction and sweating are important in the prevention and the control of primary irritation due to chromates. In previous years an ointment containing 10% sodium EDTA was applied regularly to the nasal septum before exposure. This preventive treatment could assist in keeping the septum intact. Soreness of the nose and early ulceration were also treated by regular application of this ointment, and healing could be achieved without perforation.

Results from research indicate that workers exposed to high air concentrations of Cr^{VI} could be monitored successfully by monitoring the excretion of chromium in the urine. Such results, however, bear no relation to the hazard of skin allergy. As of today, with the very long latent period of Cr^{VI}-related lung cancer, hardly anything can be said regarding the cancer hazard on the basis of urinary levels of Cr.

COPPER

Copper (Cu) is malleable and ductile, conducts heat and electricity exceedingly well and is very little altered in its functional capacity by exposure to dry air. In a moist atmosphere containing carbon

dioxide it becomes coated with a green carbonate. Copper is an essential element in human metabolism.

Occurrence and Uses

Copper occurs principally as mineral compounds in which ^{63}Cu constitutes 69.1% and ^{65}Cu, 30.9% of the element. Copper is widely distributed in all continents and is present in most living organisms. Although some natural deposits of metallic copper have been found, it is generally mined either as sulphide ores, including covellite (CuS), chalcocite (Cu_2S), chalcopyrite ($CuFeS_2$) and bornite (Cu_3FeS_3); or as oxides, including malachite ($Cu_2CO_3(OH)_2$); chrysocolla ($CuSiO_3 \cdot 2H_2O$) and chalcanthite ($CuSO_4 \cdot 5H_2O$).

Because of its electrical properties, more than 75% of copper output is used in the electrical industries. Other applications for copper include water piping, roofing material, kitchenware, chemical and pharmaceutical equipment, and the production of copper alloys. Copper metal is also used as a pigment, and as a precipitant of selenium.

Alloys and Compounds

The most widely used non-ferrous copper alloys are those of copper and zinc (brass), tin (bronze), nickel (monel metal), aluminium, gold, lead, cadmium, chromium, beryllium, silicon or phosphorus.

Copper sulphate is used as an algicide and molluscicide in water; with lime, as a plant fungicide; as a mordant; in electroplating; as a froth flotation agent for the separation of zinc sulphide ore; and as an agent for leather tanning and hide preservation. Copper sulphate neutralized with hydrated lime, known as Bordeaux mixture, is used for the prevention of mildew in vineyards.

Cupric oxide has been used as a component of paint for ship bottoms and as a pigment in glass, ceramics, enamels, porcelain glazes and artificial gems. It is also used in the manufacture of rayon and other copper compounds, and as an optical glass polishing agent and a solvent for chromic iron ores. Cupric oxide is a component of flux in copper metallurgy, pyrotechnic compositions, welding fluxes for bronze and agricultural products such as insecticides and fungicides. Black cupric oxide is used for correcting copper-deficient soils and as a feed supplement.

Copper chromates are pigments, catalysts for liquid-phase hydrogenation and potato fungicides. A solution of cupric hydroxide in excess ammonia is a solvent for cellulose used in the manufacture of rayon (viscose). Cupric hydroxide is used in the manufacture of battery electrodes and for treating and staining paper. It is also a pigment, a feed additive, a mordant in dyeing and an ingredient in fungicides and insecticides.

Hazards

Amine complexes of cupric chlorate, cupric dithionate, cupric azide and cuprous acetylide are explosive but are of no industrial or public health importance. Copper acetylide was found to be the cause of explosions in acetylene plants and has caused the abandonment of the use of copper in the

construction of such plants. Fragments of metallic copper or copper alloys that lodge in the eye, a condition known as chalcosis, may lead to uveitis, abscess and loss of the eye. Workers who spray vineyards with Bordeaux mixture may suffer from pulmonary lesions (sometimes called “vineyard sprayer’s lung”) and copper-laden hepatic granulomas.

Accidental ingestion of soluble copper salts is generally innocuous since the vomiting induced rids the patient of much of the copper. The possibility of copper-induced toxicity may occur in the following situations:

· The oral administration of copper salts is occasionally employed for therapeutic purposes, particularly in India.

· Copper dissolved from the wire used in certain intra-uterine contraceptive devices has been shown to be absorbed systemically.

· An appreciable fraction of the copper dissolved from the tubing commonly used in haemodialysis equipment may be retained by the patient and can produce significant increases in hepatic copper.

· Copper, not uncommonly added to feed for livestock and poultry, concentrates in the liver of these animals and can greatly increase the intake of the element when these livers are eaten. Copper is also added, in large amounts relative to the normal human dietary intake, to a number of pet animal foods that are occasionally consumed by people. Manure from animals with copper-supplemented diets can result in an excessive amount of copper in vegetables and feed grains grown on soil dressed with this manure.

Acute toxicity

Although some chemical reference works contain statements to the effect that soluble salts of copper are poisonous, in practical terms this is true only if such solutions are used with misguided or suicidal intent, or as topical treatment of extensively burned areas. When copper sulphate, known as bluestone or blue vitriol, is ingested in gram quantities, it induces nausea, vomiting, diarrhoea, sweating, intravascular haemolysis and possible kidney failure; rarely, convulsions, coma and death may result. Drinking of carbonated water or citrus fruit juices which have been in contact with copper vessels, pipes, tubing or valves can cause gastrointestinal irritation, which is seldom serious. Such beverages are acidic enough to dissolve irritating levels of copper. There is a report of corneal ulcers and skin irritation, but little other toxicity, in a copper-mine worker who fell into an electrolytic bath, but the acidity, rather than the copper, may have been the cause. In some instances where copper salts have been used in the treatment of burns, high concentrations of serum copper and toxic manifestations have ensued.

The inhalation of dusts, fumes and mists of copper salts can cause congestion of the nasal and mucous membranes and ulceration with perforation of the nasal septum. Fumes from the heating of metallic copper can cause metal fume fever, nausea, gastric pain and diarrhoea.

Chronic toxicity

Chronic toxic effects in human beings attributable to copper appears only to be found in individuals who have inherited a particular pair of abnormal autosomal recessive genes and in whom, as a consequence, hepatolenticular degeneration (Wilson's disease) develops. This is a rare occurrence. Most daily human diets contain 2 to 5 mg of copper, almost none of which is retained. The adult human body copper content is quite constant at about 100 to 150 mg. In normal individuals (without Wilson's disease), almost all of the copper is present as an integral and functional moiety of one of perhaps a dozen proteins and enzyme systems including, for example, cytochrome oxidase, dopa-oxidase and serum ceruloplasmin. Tenfold, or more, increases in the daily intake of copper can occur in individuals who eat large quantities of oysters (and other shellfish), liver, mushrooms, nuts and chocolate—all rich in copper; or in miners who may work and eat meals, for 20 years or more, in an atmosphere laden with 1 to 2% copper ores dusts. Yet evidence of primary chronic copper toxicity (well defined from observations of patients with inherited chronic copper toxicosis—Wilson's disease—as dysfunction of and structural damage to the liver, central nervous system, kidney, bones and eyes) has never been found in any individuals except those with Wilson's disease. However, the excessive copper deposits that are found in the livers of patients with primary biliary cirrhosis, cholestasis and Indian childhood cirrhosis may be one contributing factor to the severity of the hepatic disease that is characteristic of these conditions.

Safety and Health Measures

Workers exposed to copper dusts or mists should be provided with adequate protective clothing to prevent repeated or prolonged skin contact. Where dust conditions cannot be sufficiently controlled, appropriate respirators and eye protection are necessary. Housekeeping and the provision of adequate sanitary facilities is essential since eating, drinking and smoking should be prohibited at the worksite. In mines where there are water-soluble ores such as chalcanthite, workers should be particularly careful to wash their hands with water before eating.

The prevention of metal fume fever is a matter of keeping exposure below the level of concentration currently accepted as satisfactory for working with copper in industry. The employment of local exhaust ventilation (LEV) is a necessary measure to collect copper fumes at the source.

People with Wilson's disease should avoid employment in copper industries. The serum concentration of ceruloplasmin is a screen for this condition, since unaffected individuals have levels which range from 20 to 50 mg/100 cm^3 of this copper protein whereas 97% of patients with Wilson's disease have less than 20 mg/100 cm^3. This is a relatively expensive procedure for broad-based screening programmes.

IRON

Occurrence and Uses

Iron is second in abundance amongst the metals and is fourth amongst the elements, surpassed only by oxygen, silicon and aluminium. The most common iron ores are: haematite, or red iron ore (Fe_2O_3), which is 70% iron; limonite, or brown iron ore ($FeO(OH)\cdot nH_2O$), containing 42% iron; magnetite, or magnetic iron ore (Fe_3O_4), which has a high iron content; siderite, or spathic iron ore ($FeCO_3$); pyrite (FeS_2), the most common sulphide mineral; and pyrrhotite, or magnetic pyrite (FeS). Iron is used in the manufacture of iron and steel castings, and it is alloyed with other metals to form steels. Iron is also used to increase the density of oil-well drilling fluids.

Alloys and Compounds

Iron itself is not particularly strong, but its strength is greatly increased when it is alloyed with carbon and rapidly cooled to produce steel. Its presence in steel accounts for its importance as an industrial metal. Certain characteristics of steel—that is, whether it is soft, mild, medium or hard—are largely determined by the carbon content, which may vary from 0.10 to 1.15%. About 20 other elements are used in varied combinations and proportions in the production of steel alloys with many different qualities—hardness, ductility, corrosion resistance and so on. The most important of these are manganese (ferromanganese and spiegeleisen), silicon (ferrosilicon) and chromium, which is discussed below.

The most important industrial iron compounds are the oxides and the carbonate, which constitute the principal ores from which the metal is obtained. Of lesser industrial importance are cyanides, nitrides, nitrates, phosphides, phosphates and iron carbonyl.

Hazards

Industrial dangers are present during the mining, transportation and preparation of the ores, during the production and use of the metal and alloys in iron and steel works and in foundries, and during the manufacture and use of certain compounds. Inhalation of iron dust or fumes occurs in iron-ore mining; arc welding; metal grinding, polishing and working; and in boiler scaling. If inhaled, iron is a local irritant to the lung and gastrointestinal tract. Reports indicate that long-term exposure to a mixture of iron and other metallic dusts may impair pulmonary function.

Accidents are liable to occur during the mining, transportation and preparation of the ores because of the heavy cutting, conveying, crushing and sieving machinery that is used for this purpose. Injuries may also arise from the handling of explosives used in the mining operations.

Inhaling dust containing silica or iron oxide can lead to pneumoconiosis, but there are no definite conclusions as to the role of iron oxide particles in the development of lung cancer in humans. Based on animal experiments, it is suspected that iron oxide dust may serve as a “co-carcinogenic” substance,

thus enhancing the development of cancer when combined simultaneously with exposure to carcinogenic substances.

Mortality studies of haematite miners have shown an increased risk of lung cancer, generally among smokers, in several mining areas such as Cumberland, Lorraine, Kiruna and Krivoi Rog. Epidemiological studies of iron and steel foundry workers have typically noted risks of lung cancer elevated by 1.5- to 2.5-fold. The International Agency for Research on Cancer (IARC) classifies iron and steel founding as a carcinogenic process for humans. The specific chemical agents involved (e.g., polynuclear aromatic hydrocarbons, silica, metal fumes) have not been identified. An increased incidence of lung cancer has also been reported, but less significantly, among metal grinders. The conclusions for lung cancer among welders are controversial.

In experimental studies, ferric oxide has not been found to be carcinogenic; however, the experiments were not carried out with haematite. The presence of radon in the atmosphere of haematite mines has been suggested to be an important carcinogenic factor.

Serious accidents can occur in iron processing. Burns can occur in the course of work with molten metal, as described elsewhere in this Encyclopaedia. Finely divided freshly reduced iron powder is pyrophoric and ignites on exposure to air at normal temperatures. Fires and dust explosions have occurred in ducts and separators of dust-extraction plants, associated with grinding and polishing wheels and finishing belts, when sparks from the grinding operation have ignited the fine steel dust in the extraction plant.

The dangerous properties of the remaining iron compounds are usually due to the radical with which the iron is associated. Thus ferric arsenate ($FeAsO_4$) and ferric arsenite ($FeAsO_3 \cdot Fe_2O_3$) possess the poisonous properties of arsenical compounds. Iron carbonyl ($FeCO_5$) is one of the more dangerous of the metal carbonyls, having both toxic and flammable properties. Carbonyls are discussed in more detail elsewhere in this chapter.

Ferrous sulphide (FeS), in addition to its natural occurrence as pyrite, is occasionally formed unintentionally when materials containing sulphur are treated in iron and steel vessels, such as in petroleum refineries. If the plant is opened and the deposit of ferrous sulphide is exposed to the air, its exothermic oxidation may raise the temperature of the deposit to the ignition temperature of gases and vapours in the vicinity. A fine water spray should be directed on such deposits until flammable vapours have been removed by purging. Similar problems may occur in pyrite mines, where the air temperature is increased by a continuous slow oxidation of the ore.

Safety and health measures

The precautions for the prevention of mechanical accidents include the fencing and remote control of machinery, the design of plant (which, in modern steel-making, includes computerized control) and the safety training of workers.

The danger arising from toxic and flammable gases, vapours and dusts is countered by local exhaust and general ventilation coupled with the various forms of remote control. Protective clothing and eye

protection should be provided to safeguard the worker from the effects of hot and corrosive substances, and heat.

It is especially important that the ducting at grinding and polishing machines and at finishing belts be maintained at regular intervals to keep up the efficiency of the exhaust ventilation as well as to reduce the risk of explosion.

Ferroalloys

A ferroalloy is an alloy of iron with an element other than carbon. These metallic mixtures are used as a vehicle for introducing specific elements into the manufacture of steel in order to produce steels with specific properties. The element may alloy with the steel by solution or it may neutralize harmful impurities.

Alloys have unique properties dependent on the concentration of their elements. These properties vary directly in relation to the concentration of the individual components and depend, in part, on the presence of trace quantities of other elements. Although the biological effect of each element in the alloy may be used as a guide, there is sufficient evidence for the modification of action by the mixture of elements to warrant extreme caution in making critical decisions based on extrapolation of effect from the single element.

The ferroalloys constitute a wide and diverse list of alloys with many different mixtures within each class of alloy. The trade generally limits the number of types of ferroalloy available in any one class but metallurgical developments can result in frequent additions or changes. Some of the more common ferroalloys are as follows:

- ferroboron—16.2% boron
- ferrochromium—60 to 70% chromium, that may also contain silicon and manganese
- ferromanganese—78 to 90% manganese; 1.25 to 7% silicon
- ferromolybdenum—55 to 75% molybdenum; 1.5% silicon
- ferrophosphorus—18 to 25% phosphorus
- ferrosilicon—5 to 90% silica
- ferrotitanium—14 to 45% titanium; 4 to 13% silicon
- ferrotungsten—70 to 80% tungsten
- ferrovanadium—30 to 40% vanadium; 13% silicon; 1.5% aluminium.

Hazards

Although certain ferroalloys do have non-metallurgical uses, the main sources of hazardous exposure are encountered in the manufacture of these alloys and in their use during steel production. Some ferroalloys are produced and used in fine particulate form; airborne dust constitutes a potential toxicity

hazard as well as a fire and explosion hazard. In addition, occupational exposure to the fumes of certain alloys has been associated with serious health problems.

Ferroboron. Airborne dust produced during the cleaning of this alloy may cause irritation of the nose and throat, which is due, possibly, to the presence of a boron oxide film on the alloy surface. Some animal studies (dogs exposed to atmospheric ferroboron concentrations of 57 mg/m^3 for 23 weeks) found no adverse effects.

Ferrochromium. One study in Norway on the overall mortality and the incidence of cancer in workers producing ferrochromium has shown an increased incidence of lung cancer in causal relationship with the exposure to hexavalent chromium around the furnaces. Perforation of the nasal septum was also found in a few workers. Another study concludes that excess mortality due to lung cancer in steel-manufacturing workers is associated with exposure to polycyclic aromatic hydrocarbons (PAHs) during ferrochromium production. Yet another study investigating the association between occupational exposure to fumes and lung cancer found that ferrochromium workers demonstrated excess cases of both lung and prostate cancer.

Ferromanganese may be produced by reducing manganese ores in an electric furnace with coke and adding dolomite and limestone as flux. Transportation, storage, sorting and crushing of the ores produce managanese dust in concentrations which can be hazardous. The pathological effects resulting from exposure to dust, from both the ore and the alloy, are virtually indistinguishable from those described in the article “Manganese” in this chapter. Both acute and chronic intoxications have been observed. Ferromanganese alloys containing very high proportions of manganese will react with moisture to produce manganese carbide, which, when combined with moisture, releases hydrogen, creating a fire and explosion hazard.

Ferrosilicon production can result in both aerosols and dusts of ferrosilicon. Animal studies indicate that ferrosilicon dust can cause thickening of the alveolar walls with the occasional disappearance of the alveolar structure. The raw materials used in alloy production may also contain free silica, although in relatively low concentrations. There is some disagreement as to whether classical silicosis may be a potential hazard in ferrosilicon production. There is no doubt, however, that chronic pulmonary disease, whatever its classification, can result from excessive exposure to the dust or aerosols encountered in ferrosilicon plants.

Ferrovanadium. Atmospheric contamination with dust and fumes is also a hazard in ferrovanadium production. Under normal conditions, the aerosols will not produce acute intoxication but may cause bronchitis and a pulmonary interstitial proliferative process. The vanadium in the ferrovanadium alloy has been reported to be appreciably more toxic than free vanadium as a result of its greater solubility in biological fluids.

Leaded steel is used for automobile sheet steel in order to increase malleability. It contains approximately 0.35% lead. Whenever the leaded steel is subject to high temperature, as in welding, there is always the danger of generating lead fumes.

Safety and health measures

Control of fumes, dust and aerosols during the manufacture and use of ferroalloys is essential. Good dust control is required in the transport and handling of the ores and alloys. Ore piles should be wetted down to reduce dust formation. In addition to these basic dust-control measures, special precautions are needed in the handling of specific ferroalloys.

Ferrosilicon reacts with moisture to produce phosphine and arsine; consequently this material should not be loaded in damp weather, and special precautions should be taken to ensure that it remains dry during storage and transport. Whenever ferrosilicon is being shipped or handled in quantities of any importance, notices should be posted warning workers of the hazard, and detection and analysis procedures should be implemented at frequent intervals to check for the presence of phosphine and arsine in the air. Good dust and aerosol control is required for respiratory protection. Suitable respiratory protective equipment should be available for emergencies.

Workers engaged in the production and use of ferroalloys should receive careful medical supervision. Their working environment should be monitored continuously or periodically, depending on the degree of risk. The toxic effects of the various ferroalloys are sufficiently divergent from those of the pure metals to warrant a more intense level of medical supervision until more data have been obtained. Where ferroalloys give rise to dust, fumes and aerosols, workers should receive periodic chest x-ray examinations for early detection of respiratory changes. Lung function testing and monitoring of metal concentrations in the blood and/or urine of exposed workers may also be required.

GALLIUM

Chemically, gallium (Ga) is similar to aluminium. It is not attacked by air and does not react with water. When cold, gallium reacts with chlorine and bromine, and when heated, with iodine, oxygen and sulphur. There are 12 known artificial radioactive isotopes, with atomic weights between 64 and 74 and half-lives between 2.6 minutes and 77.9 hours. When gallium is dissolved in inorganic acids, salts are formed, which change into insoluble hydroxide $Ga(OH)_3$ with amphoteric properties (i.e., both acidic and basic) when the pH is higher than 3. The three oxides of gallium are GaO, Ga_2O and Ga_2O_3.

Occurrence and Uses

The richest source of gallium is the mineral germanite, a copper sulphide ore which may contain 0.5 to 0.7% gallium and is found in southwest Africa. It is also widely distributed in small amounts together with zinc blendes, in aluminium clays, feldspars, coal and in the ores of iron, manganese and chromium. On a relatively small scale, the metal, alloys, oxides and salts are used in industries such as machine construction (coatings, lubricants), instrument making (solders, washers, fillers), electronics and electrical equipment production (diodes, transistors, lasers, conductor coverings), and in vacuum technology.

In the chemical industries gallium and its compounds are used as catalysts. Gallium arsenide has been widely used for semiconductor applications including transistors, solar cells, lasers and microwave

generation. Gallium arsenide is used in the production of optoelectronic devices and integrated circuits. Other applications include the use of ^{72}Ga for the study of gallium interactions in the organism and ^{67}Ga as a tumour-scanning agent. Because of the high affinity of macrophages of the lymphoreticular tissues for ^{67}Ga, it can be used in the diagnosis of Hodgkin's disease, Boeck's sarcoid and lymphatic tuberculosis. Gallium scintography is a pulmonary imaging technique which can be used in conjunction with an initial chest radiograph to evaluate workers at risk of developing occupational lung disease.

Hazards

Workers in the electronics industry using gallium arsenide may be exposed to hazardous substances such as arsenic and arsine. Inhalation exposures of dusts are possible during the production of the oxides and powdered salts ($Ga_2(SO_4)_3$, Ga_3Cl) and in the production and processing of monocrystals of semiconductor compounds. The splashing or spilling of the solutions of the metal and its salts may act on the skin or mucous membranes of workers. Grinding of gallium phosphide in water gives rise to considerable quantities of phosphine, requiring preventive measures. Gallium compounds may be ingested via soiled hands and by eating, drinking and smoking in workplaces.

Occupational diseases from gallium have not been described, except for a case report of a petechial rash followed by a radial neuritis after a short exposure to a small amount of fumes containing gallium fluoride. The biological action of the metal and its compounds has been studied experimentally. The toxicity of gallium and compounds depends upon the mode of entry into the body. When administered orally in rabbits over a long period of time (4 to 5 months), its action was insignificant and included disturbances in protein reactions and reduced enzyme activity. The low toxicity in this case is explained by the relatively inactive absorption of gallium in the digestive tract. In the stomach and intestines, compounds are formed which are either insoluble or difficult to absorb, such as metal gallates and hydroxides. The dust of the oxide, nitride and arsenide of gallium was generally toxic when introduced into the respiratory system (intratracheal injections in white rats), causing dystrophy of the liver and kidneys. In the lungs it caused inflammatory and sclerotic changes. One study concludes that exposing rats to gallium oxide particles at concentrations near the threshold limit value induces progressive lung damage that is similar to that induced by quartz. Gallium nitrate has a powerful caustic effect on the conjunctivae, cornea and skin. The high toxicity of the acetate, citrate and chloride of gallium was demonstrated by intraperitoneal injection, leading to death of animals from paralysis of the respiratory centre.

Safety and Health Measures

In order to avoid contamination of the atmosphere of workplaces by the dusts of gallium dioxide, nitride and semiconductor compounds, precautionary measures should include enclosure of dust-producing equipment and effective local exhaust ventilation (LEV). Personal protective measures during the production of gallium should prevent ingestion and contact of gallium compounds with the skin. Consequently, good personal hygiene and the use of personal protective equipment (PPE) are important. The US National Institute for Occupational Safety and Health (NIOSH) recommends control

of worker exposure to gallium-arsenide by observing the recommended exposure limit for inorganic arsenic, and advises that concentration of gallium arsenide in air should be estimated by determining arsenic. Workers should be educated in possible hazards, and proper engineering controls should be installed during production of microelectronic devices where exposure to gallium arsenide is likely. In view of the toxicity of gallium and its compounds, as shown by experiments, all persons involved in work with these substances should undergo periodic medical examinations, during which special attention should be paid to the condition of the liver, kidneys, respiratory organs and skin.

GERMANIUM

Occurrence and Uses

Germanium (Ge) is always found in combination with other elements and never in the free state. Among the most common germanium-bearing minerals are argyrodite (Ag_8GeS_6), containing 5.7% germanium, and germanite ($CuS{\cdot}FeS{\cdot}GeS_2$), containing up to 10% Ge. Extensive deposits of germanium minerals are rare, but the element is widely distributed within the structure of other minerals, especially in sulphides (most commonly in zinc sulphide and in silicates). Small quantities are also found in different types of coal.

The largest end use of germanium is the production of infrared sensing and identification systems. Its use in fibre-optical systems has increased, while consumption for semiconductors has continued to decline due to advances in silicon semiconductor technology. Germanium is also used in electroplating and in the production of alloys, one of which, germanium-bronze, is characterized by high corrosion resistance. Germanium tetrachloride ($GeCl_4$) is an intermediate in the preparation of germanium dioxide and organogermanium compounds. Germanium dioxide (GeO_2) is used in the manufacture of optical glass and in cathodes.

Hazards

Occupational health problems may arise from the dispersion of dust during the loading of germanium concentrate, breaking up and loading of the dioxide for reduction to metallic germanium, and loading of powdered germanium for melting into ingots. In the process of producing metal, during chlorination of the concentrate, distillation, rectification and hydrolysis of germanium tetrachloride, the fumes of germanium tetrachloride, chlorine and germanium chloride pyrolysis products may also present a health hazard. Other sources of health hazards are the production of radiant heat from tube furnaces for GeO_2 reduction and during melting of germanium powder into ingots, and the formation of carbon monoxide during GeO_2 reduction with carbon.

The production of single crystals of germanium for the manufacture of semiconductors brings about high air temperatures (up to 45 ºC), electromagnetic radiation with field strengths of more than 100 V/m and magnetic radiation of more than 25 A/m, and pollution of the workplace air with metal hydrides. When alloying germanium with arsenic, arsine may form in the air (1 to 3 mg/m^3), and when

alloying it with antimony, stibine or antimonous hydride may be present (1.5 to 3.5 mg/m^3). Germanium hydride, which is used for the production of high-purity germanium, may also be a pollutant of the workplace air. The frequently required cleaning of the vertical furnaces causes the formation of dust, which contains, apart from germanium, silicon dioxide, antimony and other substances.

Machining and grinding of germanium crystals also give rise to dust. Concentrations of up to 5 mg/m^3 have been measured during dry machining.

Absorbed germanium is rapidly excreted, mainly in urine. There is little information on the toxicity of inorganic germanium compounds to humans. Germanium tetrachloride may produce skin irritation. In clinical trials and other long-term oral exposures to cumulative doses exceeding 16 g of spirogermanium, an organogermanium antitumour agent or other germanium compounds have been shown to be neurotoxic and nephrotoxic. Such doses are not usually absorbed in the occupational setting. Animal experiments on the effects of germanium and its compounds have shown that dust of metallic germanium and germanium dioxide causes general health impairment (inhibition of body weight increase) when inhaled in high concentrations. The lungs of the animals presented morphological changes of the type of proliferative reactions, such as thickening of the alveolar partitions and hyperplasia of the lymphatic vessels around the bronchi and blood vessels. Germanium dioxide does not irritate the skin, but if it comes into contact with the moist conjunctiva it forms germanic acid, which acts as an eye irritant. Prolonged intra-abdominal administration in doses of 10 mg/kg leads to peripheral blood changes.

The effects of germanium concentrate dust are not due to germanium, but to a number of other dust constituents, in particular silica (SiO_2). The concentrate dust exerts a pronounced fibrogenic effect resulting in the development of connective tissue and formation of nodules in the lungs similar to those observed in silicosis.

The most harmful germanium compounds are germanium hydride (GeH_4) and germanium chloride.

The hydride may provoke acute poisoning. Morphological examinations of organs of animals which died during the acute phase revealed circulatory disorders and degenerative cell changes in the parenchymatous organs. Thus the hydride appears to be a multi-system poison that may affect the nervous functions and peripheral blood.

Germanium tetrachloride is a strong irritant of the respiratory system, skin and eyes. Its threshold of irritation is 13 mg/m^3. In this concentration it depresses the pulmonary cell reaction in experimental animals. In stronger concentrations it leads to irritation of the upper airways and conjunctivitis, and to changes in respiratory rate and rhythm. Animals which survive acute poisoning develop catarrhal-desquamative bronchitis and interstitial pneumonia a few days later. Germanium chloride also exerts general toxic effects. Morphological changes have been observed in the liver, kidneys and other organs of the animals.

Safety and Health Measures

Basic measures during the manufacture and use of germanium should be aimed at preventing the contamination of the air by dust or fumes. In the production of metal, continuity of the process and enclosure of the apparatus is advisable. Adequate exhaust ventilation should be provided in areas where the dust of metallic germanium, the dioxide or the concentrate is dispersed. Local exhaust ventilation should be provided near the melting furnaces during the manufacture of semiconductors, for example on zone-refining furnaces, and during the cleaning of the furnaces. The process of manufacturing and alloying monocrystals of germanium should be carried out in a vacuum, followed by the evacuation of the formed compounds under reduced pressure. Local exhaust ventilation is essential in operations such as dry cutting and grinding of germanium crystals. Exhaust ventilation is also important in premises for the chlorination, rectification and hydrolysis of germanium tetrachloride. Appliances, connections and fittings in these premises should be made of corrosion-proof material. The workers should wear acid-proof clothing and footwear. Respirators should be worn during the cleaning of appliances.

Workers exposed to dust, concentrated hydrochloric acid, germanium hydride and germanium chloride and its hydrolysis products should undergo regular medical examinations.

INDIUM

Occurrence and Uses

In nature, Indium (In) is widely distributed and occurs most frequently together with zinc minerals (sphalerite, marmatite, christophite), its chief commercial source. It is also found in the ores of tin, manganese, tungsten, copper, iron, lead, cobalt and bismuth, but generally in amounts of less than 0.1%.

Indium is generally used in industry for surface protection or in alloys. A thin coat of indium increases the resistance of metals to corrosion and wear. It prolongs the life of moving parts in bearings and finds wide use in the aircraft and automobile industries. It is used in dental alloys, and its “wettability” makes it ideal for plating glass. Because of its resistance to corrosion, indium is utilized extensively in making motion picture screens, cathode ray oscilloscopes and mirrors. When joined with antimony and germanium in an extremely pure combination, it is widely used in the manufacture of transistors and other sensitive electronic components. Radioisotopes of indium in compounds such as indium trichloride and colloidal indium hydroxide are used in organic scanning and in the treatment of tumours.

In addition to the metal, the most common industrial compounds of indium are the trichloride, used in electroplating; the sesquioxide, used in glass manufacture; the sulphate; and the antimonide and the arsenide used as semiconductor material.

Hazards

No cases have been reported of systemic effects in humans exposed to indium. Probably the greatest current potential hazard comes from the use of indium together with arsenic, antimony and germanium in the electronics industry. This is due primarily to the fumes given off during welding and soldering

processes in the manufacture of electronic components. Any hazard arising from the purification of indium is probably attributable to the presence of other metals, such as lead, or chemicals, such as cyanide, used in the electroplating process. Exposure of the skin to indium does not seem to present a serious hazard. The tissue distribution of indium in various chemical forms has been studied by administration to laboratory animals.

The sites of highest concentration were kidney, spleen, liver and salivary glands. After inhalation, widespread lung changes were observed, such as interstitial and desquamative pneumonitis with consequent respiratory insufficiency.

The results of animal studies showed that the more soluble salts of indium were very toxic, with lethality occurring after administration of less than 5 mg/kg by way of parenteral routes of injection. However, after gavage, indium was poorly absorbed and essentially non-toxic. Histophathological studies indicated that death was due primarily to degenerative lesions in the liver and kidney. Minor changes in the blood have also been noted. In chronic poisoning by indium chloride the main change is a chronic interstitial nephritis with proteinuria. The toxicity from the more insoluble form, indium sesquioxide, was only moderate to slight, requiring up to several hundred mg/kg for lethal effect. After administration of indium arsenide to hamsters, the uptake in various organs differed from the distribution of ionic indium or arsenic compounds.

Safety and Health Measures

Preventing the inhalation of indium fumes by the use of correct ventilation appears to be the most practical safety measure. When handling indium arsenide, safety precautions such as those applied for arsenic should be observed. In the field of nuclear medicine, correct radiation safety measures must be followed when handling radioactive indium isotopes. Intoxication in rats from indium-induced hepatic necrosis has been reduced considerably by administration of ferric dextran, the action of which is apparently very specific. The use of ferric dextran as a prophylactic treatment in humans has not been possible owing to a lack of serious cases of industrial exposures to indium.

IRIDIUM

Iridium (Ir) belongs to the platinum family. Its name derives from the colours of its salt, which are reminiscent of a rainbow (iris). Although it is very hard and the most corrosion-resistant metal known, it is attacked by some salts.

Occurrence and Uses

Iridium occurs in nature in the metallic state, usually alloyed with osmium (osmiridium), platinum or gold, and it is produced from these minerals. The metal is used to manufacture crucibles for chemical laboratories and to harden platinum. Recent in vitro studies indicate the possible effects of iridium on Leishmania donovani and the trypanocidal activity of iridium against Trypanosoma brucei. Ir is used in industrial radiology and is a gamma emitter (0.31 MeV at 82.7%) and beta emitter (0.67 MeV at 47.2%). ^{192}Ir is a radioisotope which has also been used for clinical treatment, particularly cancer therapy. It is one of the most frequently used isotopes in interstitial brain irradiation.

Hazards

Very little is known about the toxicity of iridium and its compounds. There has been little opportunity to note any adverse human effects since it is used only in small amounts. All radioisotopes are potentially harmful and must be treated with appropriate safeguards required for handling radioactive sources. Soluble iridium compounds such as iridium tribromide and tetrabromide and iridium trichloride could present both toxic effects of the iridium or the halogen, but data as to its chronic toxicity are unavailable. Iridium trichloride has been reported to be a mild irritant to the skin and is positive in eye irritation test. Inhaled aerosol of metallic iridium is deposited in the upper respiratory ways of rats; the metal is then quickly removed via the gastrointestinal tract, and approximately 95% can be found in the faeces. In humans the only reports are those concerning radiation injuries due to accidental exposure to ^{192}Ir.

Safety and Health Measures

A radiation safety and medical surveillance programme should be in place for persons responsible for nursing care during interstitial brachytherapy. Radiation safety principles include exposure reduction by time, distance and shielding. Nurses who care for brachytherapy patients must wear radiation monitoring devices to record the amount of exposure. To avoid industrial radiography accidents, only trained industrial radiographers should be allowed to handle radionuclides.

LEAD*

*Adapted from ATSDR 1995.

Occurrence and Uses

Lead ores are found in many parts of the world. The richest ore is galena (lead sulphide) and this is the main commercial source of lead. Other lead ores include cerussite (carbonate), anglesite (sulphate), corcoite (chromate), wulfenite (molybdate), pyromorphite (phosphate), mutlockite (chloride) and vanadinite (vanadate). In many cases the lead ores may also contain other toxic metals.

Lead minerals are separated from gangue and other materials in the ore by dry crushing, wet grinding (to produce a slurry), gravity classification and flotation. The liberated lead minerals are smelted by a three-stage process of charge preparation (blending, conditioning, etc.), blast sintering and blast furnace reduction. The blast-furnace bullion is then refined by the removal of copper, tin, arsenic, antimony, zinc, silver and bismuth.

Metallic lead is used in the form of sheeting or pipes where pliability and resistance to corrosion are required, such as in chemical plants and the building industry; it is used also for cable sheathing, as an ingredient in solder and as a filler in the automobile industry. It is a valuable shielding material for ionizing radiations. It is used for metallizing to provide protective coatings, in the manufacture of storage batteries and as a heat treatment bath in wire drawing. Lead is present in a variety of alloys and its compounds are prepared and used in large quantities in many industries.

About 40% of lead is used as a metal, 25% in alloys and 35% in chemical compounds. Lead oxides are used in the plates of electric batteries and accumulators (PbO and Pb_3O_4), as compounding agents in rubber manufacture (PbO), as paint ingredients (Pb_3O_4) and as constituents of glazes, enamels and glass.

Lead salts form the basis of many paints and pigments; lead carbonate and lead sulphate are used as white pigments and the lead chromates provide chrome yellow, chrome orange, chrome red and chrome green. Lead arsenate is an insecticide, lead sulphate is used in rubber compounding, lead acetate has important uses in the chemical industry, lead naphthenate is an extensively used dryer and tetraethyllead is an antiknock additive for gasoline, where still permitted by law.

Lead alloys. Other metals such as antimony, arsenic, tin and bismuth may be added to lead to improve its mechanical or chemical properties, and lead itself may be added to alloys such as brass, bronze and steel to obtain certain desirable characteristics.

Inorganic lead compounds. Space is not available to describe the very large number of organic and inorganic lead compounds encountered in industry. However, the common inorganic compounds include lead monoxide (PbO), lead dioxide (PbO_2), lead tetroxide (Pb_3O_4), lead sesquioxide (Pb_2O_3), lead carbonate, lead sulphate, lead chromates, lead arsenate, lead chloride, lead silicate and lead azide.

The maximum concentration of the organic (alkyl) lead compounds in gasolines is subject to legal prescriptions in many countries, and to limitation by the manufacturers with governmental concurrence in others. Many jurisdictions have simply banned its use.

Hazards

The prime hazard of lead is its toxicity. Clinical lead poisoning has always been one of the most important occupational diseases. Medico-technical prevention has resulted in a considerable decrease in reported cases and also in less serious clinical manifestations. However, it is now evident that adverse effects occur at exposure levels hitherto regarded as acceptable.

Industrial consumption of lead is increasing and traditional consumers are being supplemented by new users such as the plastics industry. Hazardous exposure to lead, therefore, occurs in many occupations.

In lead mining, a considerable proportion of lead absorption occurs through the alimentary tract and consequently the extent of the hazard in this industry depends, to some extent, on the solubility of ores being worked. The lead sulphide (PbS) in galena is insoluble and absorption from the lung is limited; however, in the stomach, some lead sulphide may be converted to slightly soluble lead chloride which may then be absorbed in moderate quantities.

In lead smelting, the main hazards are the lead dust produced during crushing and dry grinding operations, and lead fumes and lead oxide encountered in sintering, blast-furnace reduction and refining.

Lead sheet and pipe are used principally for the constructon of equipment for storing and handling sulphuric acid. The use of lead for water and town gas pipes is limited nowadays. The hazards of working with lead increase with temperature. If lead is worked at temperatures below 500 °C, as in soldering, the risk of fume exposure is far less than in lead welding, where higher flame temperatures are used and the danger is higher. The spray coating of metals with molten lead is dangerous since it gives rise to dust and fumes at high temperatures.

The demolition of steel structures such as bridges and ships that have been painted with lead-based paints frequently gives rise to cases of lead poisoning. When metallic lead is heated to 550 °C, lead vapour will be evolved and will become oxidized. This is a condition that is liable to be present in metal refining, the melting of bronze and brass, the spraying of metallic lead, lead burning, chemical plant plumbing, ship breaking and the burning, cutting and welding of steel structures coated with paints containing lead tetroxide.

Routes of entry

The main route of entry in industry is the respiratory tract. A certain amount may be absorbed in the air passages, but the main portion is taken up by the pulmonary bloodstream. The degree of absorption depends on the proportion of the dust accounted for by particles less than 5 microns in size and the exposed worker’s respiratory minute volume. Increased workload therefore results in higher lead absorption. Although the respiratory tract is the main route of entry, poor work hygiene, smoking during work (pollution of tobacco, polluted fingers while smoking) and poor personal hygiene may considerably increase total exposure mainly by the oral route. This is one of the reasons why the correlation between the concentration of lead in workroom air and lead in blood levels often is very poor, certainly on an individual basis.

Another important factor is the level of energy expenditure: the product of concentration in air and of respiratory minute volume determines lead uptake. The effect of working overtime is to increase exposure time and reduce recovery time. Total exposure time is also much more complicated than official personnel records indicate. Only time analysis in the workplace can yield relevant data. The worker may move around the department or the factory; a job with frequent changes in posture (e.g., turning and bending) results in exposure to a great range of concentrations. A representative measure of lead intake is almost impossible to obtain without the use of a personal sampler applied for many hours and for many days.

Particle size. Since the most important route of lead absorption is by the lungs, the particle size of industrial lead dust is of considerable significance and this depends on the nature of the operation giving rise to the dust. Fine dust of respirable particle size is produced by processes such as the pulverizing and blending of lead colours, the abrasive working of lead-based fillers in automobile bodies and the dry rubbing-down of lead paint. The exhaust gases of gasoline engines yield lead chloride and lead bromide particles of 1 micron diameter. The larger particles, however, may be ingested and be absorbed via the stomach. A more informative picture of the hazard associated with a sample of lead dust might be given by including a size distribution as well as a total lead determination.

But this information is probably more important for the research investigator than for the field hygienist.

Biological fate

In the human body, inorganic lead is not metabolized but is directly absorbed, distributed and excreted. The rate at which lead is absorbed depends on its chemical and physical form and on the physiological characteristics of the exposed person (e.g., nutritional status and age). Inhaled lead deposited in the lower respiratory tract is completely absorbed. The amount of lead absorbed from the gastrointestinal tract of adults is typically 10 to 15% of the ingested quantity; for pregnant women and children, the amount absorbed can increase to as much as 50%. The quantity absorbed increases significantly under fasting conditions and with iron or calcium deficiency.

Once in the blood, lead is distributed primarily among three compartments—blood, soft tissue (kidney, bone marrow, liver, and brain), and mineralizing tissue (bones and teeth). Mineralizing tissue contains about 95% of the total body burden of lead in adults.

The lead in mineralizing tissues accumulates in subcompartments that differ in the rate at which lead is resorbed. In bone, there is both a labile component, which readily exchanges lead with the blood, and an inert pool. The lead in the inert pool poses a special risk because it is a potential endogenous source of lead. When the body is under physiological stress such as pregnancy, lactation or chronic disease, this normally inert lead can be mobilized, increasing the lead level in blood. Because of these mobile lead stores, significant drops in a person’s blood lead level can take several months or sometimes years, even after complete removal from the source of lead exposure.

Of the lead in the blood, 99% is associated with erythrocytes; the remaining 1% is in the plasma, where it is available for transport to the tissues. The blood lead not retained is either excreted by the kidneys or through biliary clearance into the gastrointestinal tract. In single-exposure studies with adults, lead has a half-life, in blood, of approximately 25 days; in soft tissue, about 40 days; and in the non-labile portion of bone, more than 25 years. Consequently, after a single exposure a person’s blood lead level may begin to return to normal; the total body burden, however, may still be elevated.

For lead poisoning to develop, major acute exposures to lead need not occur. The body accumulates this metal over a lifetime and releases it slowly, so even small doses, over time, can cause lead poisoning. It is the total body burden of lead that is related to the risk of adverse effects.

Physiological effects

Whether lead enters the body through inhalation or ingestion, the biologic effects are the same; there is interference with normal cell function and with a number of physiological processes.

Neurological effects. The most sensitive target of lead poisoning is the nervous system. In children, neurological deficits have been documented at exposure levels once thought to cause no harmful effects. In addition to the lack of a precise threshold, childhood lead toxicity may have permanent effects. One study showed that damage to the central nervous system (CNS) that occurred as a result of lead exposure at age 2 resulted in continued deficits in neurological development, such as lower IQ

scores and cognitive deficits, at age 5. In another study that measured total body burden, primary school children with high tooth lead levels but with no known history of lead poisoning had larger deficits in psychometric intelligence scores, speech and language processing, attention and classroom performance than children with lower levels of lead. A 1990 follow-up report of children with elevated lead levels in their teeth noted a sevenfold increase in the odds of failure to graduate from high school, lower class standing, greater absenteeism, more reading disabilities and deficits in vocabulary, fine motor skills, reaction time and hand-eye coordination 11 years later. The reported effects are more likely caused by the enduring toxicity of lead than by recent excessive exposures because the blood lead levels found in the young adults were low (less than 10 micrograms per deciliter (µg/dL)).

Hearing acuity, particularly at higher frequencies, has been found to decrease with increasing blood lead levels. Hearing loss may contribute to the apparent learning disabilities or poor classroom behavior exhibited by children with lead intoxication.

Adults also experience CNS effects at relatively low blood lead levels, manifested by subtle behavioural changes, fatigue and impaired concentration. Peripheral nervous system damage, primarily motor, is seen mainly in adults. Peripheral neuropathy with mild slowing of nerve conduction velocity has been reported in asymptomatic lead workers. Lead neuropathy is believed to be a motor neuron, anterior horn cell disease with peripheral dying-back of the axons. Frank wrist drop occurs only as a late sign of lead intoxication.

Haematological effects. Lead inhibits the body’s ability to make hemoglobin by interfering with several enzymatic steps in the heme pathway. Ferrochelatase, which catalyzes the insertion of iron into protoporphyrin IX, is quite sensitive to lead. A decrease in the activity of this enzyme results in an increase of the substrate, erythrocyte protoporphyrin (EP), in the red blood cells. Recent data indicate that the EP level, which has been used to screen for lead toxicity in the past, is not sufficiently sensitive at lower levels of blood lead and is therefore not as useful a screening test for lead poisoning as previously thought.

Lead can induce two types of anaemia. Acute high-level lead poisoning has been associated with hemolytic anaemia. In chronic lead poisoning, lead induces anemia by both interfering with erythropoiesis and by diminishing red blood cell survival. It should be emphasized, however, that anemia is not an early manifestation of lead poisoning and is evident only when the blood lead level is significantly elevated for prolonged periods.

Endocrine effects. A strong inverse correlation exists between blood lead levels and levels of vitamin D. Because the vitamin D-endocrine system is responsible in large part for the maintenance of extra- and intra-cellular calcium homeostasis, it is likely that lead impairs cell growth and maturation and tooth and bone development.

Renal effects. A direct effect on the kidney of long-term lead exposure is nephropathy. Impairment of proximal tubular function manifests in aminoaciduria, glycosuria and hyperphosphaturia (a Fanconi-like syndrome). There is also evidence of an association between lead exposure and hypertension, an effect that may be mediated through renal mechanisms. Gout may develop as a result of lead-induced

hyperuricemia, with selective decreases in the fractional excretion of uric acid before a decline in creatinine clearance. Renal failure accounts for 10% of deaths in patients with gout.

Reproductive and developmental effects. Maternal lead stores readily cross the placenta, placing the foetus at risk. An increased frequency of miscarriages and stillbirths among women working in the lead trades was reported as early as the end of the 19th century. Although the data concerning exposure levels are incomplete, these effects were probably a result of far greater exposures than are currently found in lead industries. Reliable dose-effect data for reproductive effects in women are still lacking today.

Increasing evidence indicates that lead not only affects the viability of the foetus, but development as well. Developmental consequences of prenatal exposure to low levels of lead include reduced birth weight and premature birth. Lead is an animal teratogen; however, most studies in humans have failed to show a relationship between lead levels and congenital malformations.

The effects of lead on the male reproductive system in humans have not been well characterized. The available data support a tentative conclusion that testicular effects, including reduced sperm counts and motility, may result from chronic exposure to lead.

Carcinogenic effects. Inorganic lead and inorganic lead compounds have been classified as Group 2B, possible human carcinogens, by the International Agency for Research on Cancer (IARC). Case reports have implicated lead as a potential renal carcinogen in humans, but the association remains uncertain. Soluble salts, such as lead acetate and lead phosphate, have been reported to cause kidney tumors in rats.

Continuum of signs and symptoms associated with lead toxicity

Mild toxicity associated with lead exposure includes the following:

- myalgia or paresthesia
- mild fatigue
- irritability
- lethargy
- occasional abdominal discomfort.

The signs and symptoms associated with moderate toxicity include:

- arthralgia
- general fatigue
- difficulty concentrating
- muscular exhaustibility
- tremor

- headache
- diffuse abdominal pain
- vomiting
- weight loss
- constipation.

The signs and symptoms of severe toxicity include:

- paresis or paralysis
- encephalopathy, which may abruptly lead to seizures, changes in consciousness, coma and death
- lead line (blue-black) on gingival tissue
- colic (intermittent, severe abdominal cramps).

Some of the haematological signs of lead poisoning mimic other diseases or conditions. In the differential diagnosis of microcytic anaemia, lead poisoning can usually be ruled out by obtaining a venous blood lead concentration; if the blood lead level is less than 25 µg/dL, the anaemia usually reflects iron deficiency or haemoglobinopathy. Two rare diseases, acute intermittent porphyria and coproporphyria, also result in haeme abnormalities similar to those of lead poisoning.

Other effects of lead poisoning can be misleading. Patients exhibiting neurological signs due to lead poisoning have been treated only for peripheral neuropathy or carpal tunnel syndrome, delaying treatment for lead intoxication. Failure to correctly diagnose lead induced gastrointestinal distress has led to inappropriate abdominal surgery.

Laboratory evaluation

If pica or accidental ingestion of lead-containing objects (such as curtain weights or fishing sinkers) is suspected, an abdominal radiograph should be taken. Hair analysis is not usually an appropriate assay for lead toxicity because no correlation has been found between the amount of lead in the hair and the exposure level.

The probability of environmental lead contamination of a laboratory specimen and inconsistent sample preparation make the results of hair analysis difficult to interpret. Suggested laboratory tests to evaluate lead intoxication include the following:

- CBC with peripheral smear
- blood lead level
- erythrocyte protoporphyrin level
- BUN and creatinine level
- urinalysis.

CBC with peripheral smear. In a lead-poisoned patient, the haematocrit and haemoglobin values may be slightly to moderately low. The differential and total white count may appear normal. The peripheral smear may be either normochromic and normocytic or hypochromic and microcytic. Basophilic stippling is usually seen only in patients who have been significantly poisoned for a prolonged period. Eosinophilia may appear in patients with lead toxicity but does not show a clear dose-response effect.

It is important to note that basophilic stippling is not always seen in lead poisoned patients.

Blood lead level. A blood lead level is the most useful screening and diagnostic test for lead exposure. A blood lead level reflects lead's dynamic equilibrium between absorption, excretion and deposition in soft- and hard-tissue compartments. For chronic exposures, blood lead levels often underrepresent the total body burden; nevertheless, it is the most widely accepted and commonly used measure of lead exposure. Blood lead levels srespond relatively rapidly to abrupt or intermittent changes in lead intake (e.g., ingestion of lead paint chips by children) and, within a limited range, bear a linear relationship to those intake levels.

Today, the average blood lead level in the US population, for example, is below 10 µg/dL, down from an average of 16 µg/dL (in the 1970s), the level before the legislated removal of lead from gasoline. A blood lead level of 10 µmg/dL is about three times higher than the average level found in some remote populations.

The levels defining lead poisoning have been progressively declining. Taken together, effects occur over a wide range of blood lead concentrations, with no indication of a threshold. No safe level has yet been found for children. Even in adults, effects are being discovered at lower and lower levels as more sensitive analyses and measures are developed.

Erythrocyte protoporhyrin level. Until recently, the test of choice for screening asymptomatic populations at risk was erythrocyte protoporphyrin (EP), commonly assayed as zinc protoporphyrin (ZPP). An elevated level of protoporphyrin in the blood is a result of accumulation secondary to enzyme dysfunction in the erythrocytes. It reaches a steady state in the blood only after the entire population of circulating erythrocyles has turned over, about 120 days. Consequently, it lags behind blood lead levels and is an indirect measure of long-term lead exposure.

The major disadvantage of using EP (ZPP) testing as a method for lead screening is that it is not sensitive at the lower levels of lead poisoning. Data from the second US National Health and Nutrition Examination Survey (NHANES II) indicate that 58% of 118 children with blood lead levels above 30 µg/dL had EP levels within normal limits. This finding shows that a significant number of children with lead toxicity would be missed by reliance on EP (ZPP) testing alone as the screening tool. An EP (ZPP) level is still useful in screening patients for iron deficiency anaemia.

Normal values of ZPP are usually below 35 µg/dL. Hyperbilirubinaemia (jaundice) will cause falsely elevated readings when the haematofluorometer is used. EP is elevated in iron deficiency anaemia and in sickle cell and other haemolytic anaemias. In erythropoietic protoporphyria, an extremely rare disease, EP is markedly elevated (usually above 300 µg/dL).

BUN, creatinine and urinalysis. These parameters may reveal only late, significant effects of lead on renal function. Renal function in adults can also be assessed by measuring the fractional excretion of uric acid (normal range 5 to 10%; less than 5% in saturnine gout; greater than 10% in Fanconi syndrome).

Organic lead intoxication

The absorption of a sufficient quantity of tetraethyllead, whether briefly at a high rate or for prolonged periods at a lower rate, induces acute intoxication of the CNS. The milder manifestations are those of insomnia, lassitude and nervous excitation which reveals itself in lurid dreams and dream-like waking states of anxiety, in association with tremor, hyper-reflexia, spasmodic muscular contractions, bradycardia, vascular hypotension and hypothermia. The more severe responses include recurrent (sometimes nearly continuous) episodes of complete disorientation with hallucinations, facial contortions and intense general somatic muscular activity with resistance to physical restraint. Such episodes may be converted abruptly into maniacal or violent convulsive seizures which may terminate in coma and death.

Illness may persist for days or weeks, with intervals of quietude readily triggered into over-activity by any type of disturbance. In these less acute cases, fall in blood pressure and loss of body weight are common. When the onset of such symptomatology follows promptly (within a few hours) after brief, severe exposure to tetraethyllead, and when the symptomatology develops rapidly, an early fatal outcome is to be feared. When, however, the interval between the termination of brief or prolonged exposure and the onset of symptoms is delayed (by up to 8 days), the prognosis is guardedly hopeful, although partial or recurrent disorientation and depressed circulatory function may persist for weeks.

The initial diagnosis is suggested by a valid history of significant exposure to tetraethyllead, or by the clinical pattern of the presenting illness. It may be supported by the further development of the illness, and confirmed by evidence of a significant degree of absorption of tetraethyllead, provided by analyses of urine and blood which reveal typical findings (i.e., a striking elevation of the rate of excretion of lead in the urine) and a concurrently negligible or slight elevation of the concentration of lead in the blood.

Lead Control in the Working Environment

Clinical lead poisoning has historically been one of the most important occupational diseases, and it remains a major risk today. The considerable body of scientific knowledge concerning the toxic effects of lead has been enriched since the 1980s by significant new knowledge regarding the more subtle subclinical effects. Similarly, in a number of countries it was felt necessary to redraft or modernize work protective measures enacted over the last half-century and more.

Thus, in November 1979, in the US, the Final Standard on Occupational Exposure to Lead was issued by the Occupational Safety and Health Administration (OSHA) and in November 1980 a comprehensive Approved Code of Practice was issued in the United Kingdom regarding the control of lead at work.

The main features of the legislation, regulations and codes of practice emerging in the 1970s concerning the protection of the health of workers at work involved establishing comprehensive systems covering all work circumstances where lead is present and giving equal importance to hygiene measures, ambient monitoring and health surveillance (including biological monitoring).

Most codes of practice include the following aspects:

- assessment of work which exposes persons to lead
- information, instruction and training
- control measures for materials, plant and processes
- use and maintenance of control measures
- respiratory protective equipment and protective clothing
- washing and changing facilities and cleaning
- separate eating, drinking and smoking areas
- duty to avoid spread of contamination by lead
- air monitoring
- medical surveillance and biological tests
- keeping of records.

Some regulation, such as the OSHA lead standard, specifies the permissible exposure limit (PEL) of lead in the workplace, the frequency and extent of medical monitoring, and other responsibilities of the employer. As of this writing, if blood monitoring reveals a blood lead level greater than 40 µg/dL, the worker must be notified in writing and provided with medical examination. If a worker's blood lead level reaches 60 µg/dL (or averages 50 µg/dL or more), the employer is obligated to remove the employee from excessive exposure, with maintenance of seniority and pay, until the employee's blood lead level falls below 40 µg/dL (29 CFR 91 O.1025) (medical removal protection benefits).

Safety and Health Measures

The object of precautions is first to prevent the inhalation of lead and secondly to prevent its ingestion. These objects are most effectively achieved by the substitution of a less toxic substance for the lead compound. The use of lead polysilicates in the potteries is one example. The avoidance of lead carbonate paints for the painting of the interiors of buildings has proved very effective in reducing painters' colic; effective substitutes for lead for this purpose have become so readily available that it has been considered reasonable in some countries to prohibit the use of lead paint for the interiors of buildings.

Even if it is not possible to avoid the use of lead itself, it is still possible to avoid dust. Water sprays may be used in large quantities to prevent the formation of dust and to prevent it from becoming airborne. In lead smelting, the ore and the scrap may be treated in this way and the floors on which it

has been lying may be kept wet. Unfortunately, there is always a potential source of dust in these circumstances if the treated material or floors are ever allowed to become dry. In some instances, arrangements are made to ensure that the dust will be coarse rather than fine. Other specific engineering precautions are discussed elsewhere in this Encyclopaedia.

Workers who are exposed to lead in any of its forms should be provided with personal protective equipment (PPE), which should be washed or renewed regularly. Protective clothing made of certain man-made fibres retains much less dust than cotton overalls and should be used where the conditions of work render it possible; turn-ups, pleats and pockets in which lead dust may collect should be avoided.

Cloakroom accommodation should be provided for this PPE, with separate accommodation for clothing taken off during working hours. Washing accommodation, including bathing accommodation with warm water, should be provided and used. Time should be allowed for washing before eating. Arrangements should be made to prohibit eating and smoking in the vicinity of lead processes and suitable eating facilities should be provided.

It is essential that the rooms and the plant associated with lead processes should be kept clean by continuous cleaning either by a wet process or by vacuum cleaners. Where, in spite of these precautions, workers may still be exposed to lead, respiratory protective equipment should be provided and properly maintained. Supervision should ensure that this equipment is maintained in a clean and efficient condition and that it is used when necessary.

Organic lead

Both the toxic properties of organic lead compounds, and their ease of absorption, require that contact of the skin of workers with these compounds, alone or in concentrated mixtures in commercial formulations or in gasoline or other organic solvents, must be scrupulously avoided. Both technological and management control are essential, and appropriate training of workers in safe work practices and the use of PPE is required. It is essential that atmospheric concentrations of alkyl lead compounds in the workplace air should be maintained at extremely low levels. Personnel should not be allowed to eat, smoke or keep unsealed food or beverages at the workplace. Good sanitary facilities, including showers, should be provided and workers should be encouraged to practise good personal hygiene, especially by showering or washing after the work shift. Separate lockers should be supplied for working and private clothes.

MAGNESIUM

Magnesium (Mg) is the lightest structural metal known. It is 40% lighter than aluminium. Metallic magnesium can be rolled and drawn when heated between 300 and 475 ºC, but is brittle below this temperature and is apt to burn if heated much above it. It is soluble in, and forms compounds with, a number of acids, but is not affected by hydrofluoric or chromic acids. Unlike aluminium, it is resistant to alkali corrosion.

Occurrence and Uses

Magnesium does not exist in a pure state in nature, but is generally found in one of the following forms: dolomite ($CaCO_3 \cdot MgCO_3$), magnesite ($MgCO_3$), brucite ($Mg(OH)_2$), periclase (MgO), carnallite ($KClMgCl_2 \cdot 6H_2O$) or kieserite ($MgSO_4 \cdot H_2O$). In addition, it is found as a silicate in asbestos and talc. Magnesium is so widely distributed over the earth that facilities for processing and transporting the ore are often the determining factors in selecting a site for mining.

Magnesium is used, mainly in alloy form, for components of aircraft, ships, automobiles, machinery and hand tools for which both lightness and strength are required. It is used in the manufacture of precision instruments and optical mirrors, and in the recovery of titanium. Magnesium is also extensively used in military equipment. Because it burns with such intense light, magnesium is widely used in pyrotechnics, signal flares, incendiary and tracer bullets, and in flash bulbs.

Magnesium oxide has a high melting point (2,500 ºC) and is often incorporated into the linings of refractories. It is also a component of animal feeds, fertilizers, insulation, wallboard, petroleum additives and electrical heating rods. Magnesium oxide is useful in the pulp and paper industry. In addition, it serves as an accelerator in the rubber industry and as a reflector in optical instruments.

Other important compounds include magnesium chloride, magnesium hydroxide, magnesium nitrate and magnesium sulphate. Magnesium chloride is a component of fire extinguishers and ceramics. It is also an agent in fireproofing wood and textile and paper manufacture. Magnesium chloride is a chemical intermediate for magnesium oxychloride, which is used for cement. A mixture of magnesium oxide and magnesium chloride forms a paste which is useful for floors. Magnesium hydroxide is useful for the neutralization of acids in the chemical industry. It is also used in uranium processing and in sugar refining. Magnesium hydroxide serves as a residual fuel-oil additive and an ingredient in toothpaste and antacid stomach powder. Magnesium nitrate is used in pyrotechnics and as a catalyst in the manufacture of petrochemicals. Magnesium sulphate has numerous functions in the textile industry, including weighting cotton and silk, fireproofing fabrics, and dyeing and printing calicos. It also finds use in fertilizers, explosives, matches, mineral water, ceramics and cosmetic lotions, and in the manufacture of mother-of-pearl and frosted papers. Magnesium sulphate increases the bleaching action of chlorinated lime and acts as a water-correcting agent in the brewing industry and a cathartic and analgesic in medicine.

Alloys. When magnesium is alloyed with other metals, such as manganese, aluminium and zinc, it improves their toughness and resistance to strain. In combination with lithium, cerium, thorium and zirconium, alloys are produced which have an enhanced strength-to-weight ratio, along with considerable heat-resisting properties. This renders them invaluable in the aircraft and aerospace industries for the construction of jet engines, rocket launchers and space vehicles. A large number of alloys, all containing over 85% magnesium, are known under the general name of Dow metal.

Hazards

Biological roles. As an essential ingredient of chlorophyll, the magnesium requirements of the human body are largely supplied by the consumption of green vegetables. The average human body contains about 25 g of magnesium. It is the fourth most abundant cation in the body, after calcium, sodium and potassium. The oxidation of foods releases energy, which is stored in the high-energy phosphate bonds. It is believed that this process of oxidative phosphorylation is carried out in the mitochondria of the cells and that magnesium is necessary for this reaction.

Experimentally produced magnesium deficiency in rats leads to a dilation of the peripheral blood vessels and later to hyperexcitability and convulsions. Tetany similar to that associated with hypocalcaemia occurred in calves fed only milk. Older animals with magnesium deficiency developed “grass staggers”, a condition which appears to be associated with malabsorption rather than with a lack of magnesium in the fodder.

Cases of magnesium tetany resembling those caused by calcium deficiency have been described in humans. In the reported cases, however, a “conditioning factor”, such as an excessive vomiting or fluid loss, has been present, in addition to inadequate dietary intake. Since this tetany clinically resembles that caused by calcium deficiency, a diagnosis can be made only by determining the blood levels of calcium and magnesium. Normal blood levels range from 1.8 to 3 mg per 100 cm^3, and it has been found that persons tend to become comatose when the blood concentration approaches 17 mg per cent. “Aeroform tumours” due to the evolution of hydrogen have been produced in animals by introducing finely divided magnesium into the tissues.

Toxicity. Magnesium and alloys containing 85% of the metal may be considered together in their toxicological properties. In industry, their toxicity is regarded as low. The most frequently used compounds, magnesite and dolomite, may irritate the respiratory tract. However, the fumes of magnesium oxide, as those of certain other metals, can cause metal fume fever. Some investigators have reported a higher incidence of digestive disorders in magnesium plant workers and suggest that a relationship may exist between magnesium absorption and gastroduodenal ulcers. In foundry-casting magnesium or high-magnesium alloys, fluoride fluxes and sulphur-containing inhibitors are used in order to separate the molten metal from the air with a layer of sulphur dioxide. This prevents burning during the casting operations, but the fumes of fluorides or of sulphur dioxide could present a greater hazard.

The greatest danger in handling magnesium is that of fire. Small fragments of the metal, such as would result from grinding, polishing or machining, can readily be ignited by a chance spark or flame, and as they burn at a temperature of 1,250ºC, these fragments can cause deep destructive lesions of the skin. Accidents of this type have occurred when a tool was sharpened on a wheel which was previously used to grind magnesium alloy castings. In addition, magnesium reacts with water and acids, forming combustible hydrogen gas.

Slivers of magnesium penetrating the skin or entering deep wounds could cause “aeroform tumours” of the type already mentioned. This would be rather exceptional; however, wounds contaminated with

magnesium are very slow to heal. Fine dust from the buffing of magnesium could be irritating to the eyes and respiratory passages, but it is not specifically toxic.

Safety and Health Measures

As with any potentially hazardous industrial process, constant care is needed in handling and working magnesium. Those engaged in casting the metal should wear aprons and hand protection made of leather or some other suitable material to protect them against the “spatter” of small particles. Transparent face shields should also be worn as face protection, especially for the eyes. Where workers are exposed to magnesium dust, contact lenses should not be worn and eyewash facilities should be immediately available. Workers machining or buffing the metal should wear overalls to which small fragments of the metal will not adhere. Sufficient local exhaust ventilation is also essential in areas where magnesium oxide fumes may develop, in addition to good general ventilation. Cutting tools should be sharp, as blunt ones may heat the metal to the point of ignition.

Buildings in which magnesium is cast or machined should be constructed, if possible, of non-flammable materials and without ledges or protuberances on which magnesium dust might accumulate. The accumulation of shavings and “swarf” should be prevented, preferably by wet sweeping. Until final disposal, the scrapings should be collected in small containers and placed apart at safe intervals. The safest method for disposal of magnesium waste is probably wetting and burying.

Since the accidental ignition of magnesium presents a serious fire hazard, fire training and adequate firefighting facilities are essential. Workers should be trained never to use water in fighting such a blaze, because this merely scatters the burning fragments, and may spread the fire. Among the materials which have been suggested for the control of such fires are carbon and sand. Commercially prepared firefighting dusts are also available, one of which consists of powdered polyethylene and sodium borate.

MANGANESE

Occurrence and Uses

Manganese (Mn) is one of the most abundant elements in the earth’s crust. It is found in soils, sediments, rocks, water and biological materials. At least a hundred minerals contain manganese. Oxides, carbonates and silicates are the most important among manganese-containing minerals. Manganese can exist in eight oxidation states, the most important being +2, +3, and +7. Manganese dioxide (MnO_2) is the most stable oxide. Manganese forms various organometallic compounds. Of major practical interest is methylcyclopentadienyl manganese tricarbonyl $CH_3C_5H_4Mn(CO)_3$, often referred to as MMT.

The most important commercial source of manganese is manganese dioxide (MnO_2), which is found naturally in sedimentary deposits as pyrolusite. Two other types of deposit can be distinguished: carbonate accumulations, which are usually composed mainly of rhodocrosite ($MnCO_3$), and stratiform deposits. However, only the sedimentary deposits are significant, and those are usually worked by

opencast techniques. Sometimes underground mining is necessary, and room and pillar extraction is carried out; seldom is there any call for the techniques used in deep metal mining.

Manganese is used in the production of steel as a reagent to reduce oxygen and sulphur and as an alloying agent for special steels, aluminium and copper. It is used in the chemical industry as an oxidizing agent and for the production of potassium permanganate and other manganese chemicals. Manganese is used for electrode coating in welding rods and for rock crushers, railway points and crossings. It also finds use in the ceramics, match, glass and dyestuff industries.

Several manganese salts are used in fertilizers and as driers for linseed oil. They are also utilized for glass and textile bleaching and for leather tanning. MMT has been used as a fuel-oil additive, a smoke inhibitor, and as an antiknock gasoline additive.

Hazards

Absorption, distribution and excretion

In occupational situations manganese is primarily absorbed by inhalation. Manganese dioxide and other manganese compounds which occur as volatile by-products of metal refining are practically insoluble in water. Thus, only particles small enough to reach the alveoli are eventually absorbed into the blood. Large inhaled particles may be cleared from the respiratory tract and swallowed. Manganese may also enter the gastrointestinal tract with contaminated food and water. The rate of absorption can be influenced by a dietary level of manganese and iron, the type of manganese compound, iron deficiency and age. However, the risk of intoxication by this route is not great. Absorption of manganese through the skin is negligible.

After inhalation, or after parenteral and oral exposure, the absorbed manganese is rapidly eliminated from the blood and distributed mainly to the liver. The kinetic patterns for blood clearance and liver uptake of manganese are similar, indicating that these two manganese pools rapidly enter equilibrium. Excess metal may be distributed to other tissues such as kidneys, small intestine, endocrine glands and bones. Manganese preferentially accumulates in tissues rich in mitochondria. It also penetrates the blood-brain barrier and the placenta. Higher concentrations of manganese are also associated with pigmented portions of the body, including the retina, pigmented conjunctiva and dark skin. Dark hair also accumulates manganese. It is estimated that the total body burden for manganese is between 10 and 20 mg for a 70 kg male. The biological half-life for manganese is between 36 and 41 days, but for manganese sequestered in the brain, the half-life is considerably longer. In the blood, manganese is bound to proteins.

The organic compound MMT is rapidly metabolized in the body. The distribution seems to be similar to that seen after exposure to inorganic manganese.

Bile flow is the main route of excretion of manganese. Consequently, it is eliminated almost entirely with faeces, and only 0.1 to 1.3% of daily intake with urine. It seems that biliary excretion is the main regulatory mechanism in the homeostatic control of manganese in the body, accounting for a relative stability of manganese content in tissues. After exposure to the organic compound MMT, excretion of manganese goes to a large extent with urine. This has been explained as a result of biotransformation of

the organic compound in the kidney. As a metalloprotein compound of some enzymes, manganese is an essential element for humans.

Exposure

Intoxication by manganese is reported in mining and processing of manganese ores, in the production of manganese alloys, dry-cell batteries, welding electrodes, varnishes and ceramic tiles. Mining of ore can still present important occupational hazards, and the ferromanganese industry is the next most important source of risk. The operations that produce the highest concentrations of manganese dioxide dust are those of drilling and shotfiring. Consequently, the most dangerous job is high-speed drilling.

Considering the dependence of deposition sites and solubility rate of particle size, the dangerous effect of exposure is closely related to the particle size composition of manganese aerosol. There is also evidence that aerosols formed by condensation may be more harmful than those formed by disintegration, which can be connected again with the difference in particle size distribution. The toxicity of different manganese compounds appears to depend on the type of manganese ion present and on the oxidation state of manganese. The less oxidized the compound, the higher the toxicity.

Chronic manganese poisoning (manganism)

Chronic manganese poisoning can take either a nervous or pulmonary form. If the nervous system is attacked, three phases can be distinguished. During the initial period, diagnosis may be difficult. Early diagnosis, however, is critical because cessation of exposure appears to be effective in arresting the course of the disease. Symptoms include indifference and apathy, sleepiness, loss of appetite, headache, dizziness and asthenia. There may be bouts of excitablity, difficulty in walking and coordination, and cramps and pains in the back. These symptoms can be present in varying degrees and appear either together or in isolation. They mark the onset of the disease.

The intermediate stage is marked by the appearance of objective symptoms. First the voice become monotonous and sinks to a whisper, and speech is slow and irregular, perhaps with a stammer. There is fixed and hilarious or dazed and vacant facies, which may be attributable to an increase in the tonus of the facial muscles. The patient may abruptly burst into laughter or (more rarely) into tears. Although the faculties are much decayed, the victim appears to be in a perpetual state of euphoria. Gestures are slow and awkward, gait is normal but there may be a waving movement of the arms. The patient is unable to run and can walk backwards only with difficulty, sometimes with retropulsion. Inability to perform rapid alternating movements (adiadochokinesia) may develop, but neurological examination displays no changes except, in certain cases, exaggeration of the patellar reflexes.

Within a few months, the patient’s condition deteriorates noticeably and the various disorders, especially those affecting the gait, grow steadily more pronounced. The earliest and most obvious symptom during this phase is muscular rigidity, constant but varying in degree, which results in a very characteristic gait (slow, spasmodic and unsteady), the patient putting his or her weight on the metatarsus and producing a movement variously described as “cock-walk” or “hen’s gait”. The victim is totally incapable of walking backwards and, should he or she try to do so, falls; balance can hardly

be preserved, even when trying to stand with both feet together. A sufferer can turn round only slowly. There may be tremor, frequently in the lower limbs, even generalized.

The tendinous reflexes, rarely normal, become exaggerated. Sometimes there are vasomotor disorders with sudden sweating, pallor or blushing; on occasion there is cyanosis of the extremities. The sensory functions remain intact. The patient's mind may work only slowly; writing becomes irregular, some words being illegible. There may be changes in the pulse rate. This is the stage at which the disease becomes progressive and irreversible.

Pulmonary form. Reports of "manganese pneumoconiosis" have been contested in view of the high silica content of the rock at the site of exposure; manganese pneumonia has also been described. There is also controversy over the correlation between pneumonia and manganese exposure unless manganese acts as an aggravating factor. In view of its epidemic character and severity, the disease may be a non-typical viral pneumopathy. These manganic pneumonias respond well to antibiotics.

Pathology. Some authors maintain that there are widespread lesions to the corpus striatum, then to the cerebral cortex, the hippocampus and corpora quadrigemina (in the posterior corpora). However, others are of the opinion that the lesions to the frontal lobes provide a better explanation for all the symptoms observed than do those observed in the basal ganglia; this would be confirmed by electroencephalography. The lesions are always bilateral and more or less symmetrical.

Course. Manganese poisoning ultimately becomes chronic. However, if the disease is diagnosed while still at the early stages and the patient is removed from exposure, the course may be reversed. Once well established, it becomes progressive and irreversible, even when exposure is terminated. The nervous disorders show no tendency to regress and may be followed by deformation of the joints. Although the severity of certain symptoms may be reduced, gait remains permanently affected. The patient's general condition remains good, and he or she may live a long time, eventually dying from an intercurrent ailment.

Diagnosis. This is based primarily on the patient's personal and occupational history (job, length of exposure and so on). However, the subjective nature of the initial symptoms makes early diagnosis difficult; consequently, at this stage, questioning must be supplemented by information supplied by friends, colleagues and relatives. During the intermediate and full-blown stages of the intoxication, occupational history and objective symptoms facilitate diagnosis; laboratory examinations can provide information for supplementing the diagnosis.

Haematological changes are variable; on the one hand, there may be no changes at all, whereas, on the other, there may be leucopenia, lymphocytosis and inversion of leucocyte formula in 50% of cases, or increase in haemoglobin count (considered as the first sign of poisoning) and slight polycythaemia.

There is diminished urinary excretion of 17-ketosteroids, and it may be assumed that the adrenal function is affected. Albumin level in the cerebrospinal fluid is increased, often to a marked degree (40 to 55 and even 75 mg per cent). Digestive and hepatic symptoms are non-indicative; there is no sign of hepatomegalia or splenomegalia; however, accumulation of manganese in the liver may result in

metabolic lesions which seem to be related to the patient's endocrinological condition and may be influenced by the existence of neurological lesions.

Differential diagnosis. There may be difficulty in distinguishing between manganese poisoning and the following diseases: nerve syphilis, Parkinson's disease, disseminated sclerosis, Wilson's disease, hepatic cirrhosis and Westphal-Strümpell's disease (pseudo-sclerosis).

Safety and Health Measures

The prevention of manganese poisoning is primarily a question of suppression of manganese dusts and fumes. In mines, dry drilling should always be replaced by wet drilling. Shotfiring should be carried out after the shift so that the heading can be well ventilated before the next shift starts up. Good general ventilation at source is also essential. Airline respiratory protection equipment as well as independent respirators have to be used in specific situations to avoid excessive short-term exposures.

A high standard of personal hygiene is essential, and personal cleanliness and adequate sanitary facilities, clothing and time must be provided so that compulsory showering after work, a change of clothes and a ban on eating at the workplace can be effected. Smoking at work should be prohibited as well.

Periodic measurements of exposure levels should be performed, and attention should be given to the size distribution of airborne manganese. Contamination of drinking water and food as well as workers' dietary habits ought to be considered as a potential additional source of exposure.

It is inadvisable for workers with psychological or neurological disorders to be employed in work associated with exposure to manganese. Nutritional deficiency states may predispose to anaemia and thus increase susceptibility to manganese. Therefore workers suffering from such deficiencies have to be kept under strict surveillance. During the anaemic state, subjects should avoid exposure to manganese. The same relates to those suffering from lesions of the excretory organs, or from chronic obstructive lung disease. A study has suggested that long-term manganese exposure may contribute to the development of chronic obstructive lung disease, particularly if the exposure is combined with smoking. On the other hand impaired lungs may be more susceptible to the potential acute effect of manganese aerosols.

During the periodic medical examinations the worker should be screened for symptoms which might be connected with the subclinical stage of manganese poisoning. In addition, the worker should be examined clinically, particularly with a view to detecting early psychomotor changes and neurological signs. Subjective symptoms and abnormal behaviour may often constitute the only early indications of health impairment. Manganese can be measured in blood, urine, stools and hair. Estimation of the extent of manganese exposure by means of manganese concentration in urine and blood did not prove to be of great value.

The average manganese blood level in exposed workers seems to be of the same order as that in non-exposed persons. Contamination during sampling and analytical procedures may at least partly explain a rather wide range found in literature particularly for blood. The use of heparin as an anticoagulant is

still quite common although the manganese content in heparin may exceed that in blood. The mean concentration of manganese in urine of non-exposed people is usually estimated to be between 1 and 8 mg/l, but values up to 21 mg/l have been reported. Daily manganese intake from human diets varies greatly with the amount of unrefined cereals, nuts, leafy vegetables and tea consumed, owing to their relatively high content of manganese, and thus affects the results of normal manganese content in biological media.

A manganese concentration of 60 mg/kg of faeces and higher has been suggested as indicative of occupational exposure to manganese. Manganese content in hair is normally below 4 mg/kg. As the determination of manganese in urine, which is often used in practice, has not yet been validated enough for assessment of individual exposure, it can be used only as a group indicator of the mean level of exposure. Collection of the stool and the analysis of manganese content is not easy to perform. Our present knowledge does not include any other reliable biological parameter which might be used as an indicator of individual exposure to manganese. Thus the assessment of workers' exposure to manganese still has to rely on manganese air levels. There is also very little reliable information about the correlation between the manganese content in the blood and urine and the findings of neurological symptoms and signs.

Persons with the signs of manganese intoxication should be removed from exposure. If the worker is removed from exposure shortly after the onset of symptoms and signs (before the fully developed stage of manganism) many of the symptoms and signs will disappear. There may be some residual disturbances, however, particularly in speech and gait.

METAL CARBONYLS (especially Nickel Carbonyl)

Occurrence and Uses

Metal carbonyls have the general formula $Me_x(CO)_y$, and are formed by combination of the metal (Me) with carbon monoxide (CO). Physical properties of some metal carbonyls are listed in table 63.1 . Most are solids at ordinary temperatures, but nickel carbonyl, iron pentacarbonyl and ruthenium pentacarbonyl are liquids, and cobalt hydrocarbonyl is a gas. This article focuses on nickel carbonyl, which, because of its volatility, exceptional toxicity and industrial importance merits special attention in regard to occupational toxicology. Since iron pentacarbonyl and cobalt hydrocarbonyl also have high vapour pressures and potential for inadvertant formation, they warrant serious consideration as possible occupational toxicants. Most metal carbonyls react vigorously with oxygen and oxidizing substances, and some ignite spontaneously. Upon exposure to air and light, nickel carbonyl decomposes to carbon monoxide and particulate nickel metal, cobalt hydrocarbonyl decomposes to cobalt octacarbonyl and hydrogen, and iron pentacarbonyl decomposes to iron nonacarbonyl and carbon monoxide.

Table 63.1 Physical properties of some metal carbonyls

Metal carbonyl	Mol. Wt.	Sp. Gr. (20°C)	M.P. (°C)	B.P. (°C)	V.P. (25°C) mm Hg

$Ni(CO)_4$	170.75	1.31	–19	43	390
$CoH(CO)_4$	171.99	–	–26	–	high
$Co_2(CO)_8$	341.95	1.87	51	52*	1.5
$Co_4(CO)_{12}$	571.86	–	60*	–	very low
$Cr(CO)_6$	220.06	1.77	110*	151	0.4
$Fe_2(CO)_9$	363.79	2.08	80*	–	–
$Fe(CO)_5$	195.90	1.46	–25	103	30.5
$Fe(CO)_4$	167.89	2.00	approx. 140*	–	–
$Mo(CO)_6$	264.00	1.96	150*	156	0.2
$Ru(CO)_5$	241.12	–	–22	–	–
$W(CO)_6$	351.91	2.65	approx. 150*	175	0.1

*Decomposition starts at temperature shown.

Source: Adapted from Brief et al. 1971.

Metal carbonyls are used in isolating certain metals (e.g., nickel) from complex ores, for producing carbon steel, and for metallizing by vapour deposition. They are also used as catalysts in organic reactions (e.g., cobalt hydrocarbonyl or nickel carbonyl in olefin oxidation; cobalt octacarbonyl for the synthesis of aldehydes; nickel carbonyl for the synthesis of acrylic esters). Iron pentacarbonyl is used as a catalyst for various organic reactions, and is decomposed to make finely powdered, ultra pure iron (so-called carbonyl iron), which is used in the computer and electronics industries. Methycyclopentadienyl manganese tricarbonyl (MMT) ($CH_3C_5H_4Mn(CO)_3$) is an antiknock additive to gasoline and is discussed in the article “Manganese”.

Health Hazards

The toxicity of a given metal carbonyl depends on the toxicity of carbon monoxide and of the metal from which it is derived, as well as the volatility and instability of the carbonyl itself. The principal route of exposure is inhalation, but skin absorption can occur with the liquid carbonyls. The relative acute toxicity (LD_{50} for the rat) of nickel carbonyl, cobalt hydrocarbonyl and iron pentacarbonyl may be expressed by the ratio 1:0.52:0.33. Inhalation exposures of experimental animals to these substances induce acute interstitial pneumonitis, with pulmonary oedema and capillary damage, as well as injury to the brain, liver and kidneys.

Judging from the sparse literature on their toxicity, cobalt hydrocarbonyl and iron pentacarbonyl rarely pose health hazards in industry. None the less, iron pentacarbonyl can be formed inadvertently when carbon monoxide, or a gas mixture containing carbon monoxide, is stored under pressure in steel cylinders or fed through steel pipes, when illuminating gas is produced by petroleum reforming, or when gas welding is carried out. Presence of carbon monoxide in emission discharges from blast furnaces, electric arc furnaces and cupola furnaces during steel-making can also lead to the formation of iron pentacarbonyl.

Safety and Health Measures

Special precautions are mandatory in the storage of metal carbonyls; their handling must be mechanized to the maximum degree, and decanting should be avoided wherever possible. Vessels and piping should be purged with an inert gas (e.g., nitrogen, carbon dioxide) before being opened, and carbonyl residues should be burnt or neutralized with bromine water. Where there is an inhalation hazard, workers should be provided with airline respirators or self-contained breathing apparatus. Workshops should be fitted with down-draught ventilation.

Nickel Carbonyl

Nickel carbonyl ($Ni(CO)_4$) is mainly used as an intermediate in the Mond process for nickel refining, but it is also used for vapour-plating in the metallurgical and electronics industries and as a catalyst for synthesis of acrylic monomers in the plastics industry. Inadvertent formation of nickel carbonyl can occur in industrial processes that use nickel catalysts, such as coal gasification, petroleum refining and hydrogenation reactions, or during incineration of nickel-coated papers that are used for pressure-sensitive business forms.

Hazards

Acute, accidental exposure of workers to inhalation of nickel carbonyl usually produces mild, non-specific, immediate symptoms, including nausea, vertigo, headache, dyspnoea and chest pain. These initial symptoms usually disappear within a few hours. After 12 to 36 hours, and occasionally as long as 5 days after exposure, severe pulmonary symptoms develop, with cough, dyspnoea, tachycardia, cyanosis, profound weakness and often gastrointestinal symptoms. Human fatalities have occurred 4 to 13 days after exposure to nickel carbonyl; deaths have resulted from diffuse interstitial pneumonitis, cerebral hemorrhage or cerebral oedema. In addition to pathologic lesions in the lungs and brain,

lesions have been found in liver, kidneys, adrenals and spleen. In patients who survive acute nickel carbonyl poisoning, pulmonary insufficiency often causes protracted convalescence. Nickel carbonyl is carcinogenic and teratogenic in rats; the European Union has classified nickel carbonyl as an animal teratogen. Processes that use nickel carbonyl constitute disaster hazards, since fire and explosion can occur when nickel carbonyl is exposed to air, heat, flames or oxidizers. Decomposition of nickel carbonyl is attended by additional toxic hazards from inhalation of its decomposition products, carbon monoxide and finely particulate nickel metal.

Chronic exposure of workers to inhalation of low atmospheric concentrations of nickel carbonyl (0.007 to 0.52 mg/m^3) can cause neurological symptoms (e.g., insomnia, headache, dizziness, memory loss) and other manifestations (e.g., chest tightness, excessive sweating, alopecia). Electroencephalographic abnormalities and elevated serum monoamine oxidase activity have been observed in workers with chronic exposures to nickel carbonyl. A synergistic effect of cigarette smoking and nickel carbonyl exposure on the frequency of sister-chromatid exchanges was noted in a cytogenetic evaluation of workers with chronic exposure to nickel carbonyl.

Safety and Health Measures

Fire and explosion prevention. Because of its flammability and tendency to explode, nickel carbonyl should be stored in tightly closed containers in a cool, well-ventilated area, away from heat and oxidizers such as nitric acid and chlorine. Flames and sources of ignition should be prohibited wherever nickel carbonyl is handled, used or stored. Nickel carbonyl should be transported in steel cylinders. Foam, dry chemical, or CO_2 fire extinguishers should be used to extinguish burning nickel carbonyl, rather than a stream of water, which might scatter and spread the fire.

Health protection. In addition to the medical surveillance measures recommended for all nickel-exposed workers, persons with occupational exposures to nickel carbonyl should have biological monitoring of nickel concentration in urine specimens on a regular basis, typically monthly. Persons who enter confined spaces where they might possibly be exposed to nickel carbonyl should have self-contained breathing apparatus and a suitable harness with lifeline tended by another employee outside the space. Analytical instruments for continuous atmospheric monitoring of nickel carbonyl include (a) Fourier-transform infrared absorption spectroscopes, (b) plasma chromatographs and (c) chemiluminescent detectors. Atmospheric samples can also be analysed for nickel carbonyl by (d) gas chromatography, (e) atomic absorption spectrophotometry and (f) colourimetric procedures.

Treatment. Workers suspected to have been acutely exposed to nickel carbonyl should be immediately removed from the exposure site. Contaminated clothing should be removed. Oxygen should be administered and the patient kept at rest until seen by a physician. Each voiding of urine is saved for nickel analysis. The severity of acute nickel carbonyl poisoning correlates with the urine nickel concentrations during the first 3 days after exposure. Exposures are classified as “mild” if the initial 8-h specimen of urine has a nickel concentration less than 100 µg/l, “moderate” if the nickel concentration is 100 to 500 µg/l, and “severe” if the nickel concentration exceeds 500 µg/l. Sodium diethyldithiocarbamate is the drug of choice for chelation therapy of acute nickel carbonyl poisoning.

Ancillary therapeutic measures include bed rest, oxygen therapy, corticosteroids and prophylactic antibiotics. Carbon monoxide poisoning may occur simultaneously and requires treatment.

MERCURY

Inorganic Mercury

Mercury combines readily with sulphur and halogens at ordinary temperatures and forms amalgams with all metals except iron, nickel, cadmium, aluminium, cobalt and platinum. It reacts exothermically (generates heat) with alkaline metals, is attacked by nitric acid but not by hydrochloric acid and, when hot, will combine with sulphuric acid.

Inorganic mercury is found in nature in the form of the sulphide (HgS) as cinnabar ore, which has an average mercury content of 0.1 to 4%. It is also encountered in the earth's crust in the form of geodes of liquid mercury (in Almadén) and as impregnated schist or slate (e.g., in India and Yugoslavia).

Extraction. Mercury ore is extracted by underground mining, and mercury metal is separated from the ore by roasting in a rotary kiln or shaft furnace, or by reduction with iron or calcium oxide. The vapour is carried off in the combustion gases and is condensed in vertical tubes.

The most important uses of metallic mercury and its inorganic compounds have included the treatment of gold and silver ores; the manufacture of amalgams; the manufacture and repair of measurement or laboratory apparatus; the manufacture of incandescent electric bulbs, mercury vapour tubes, radio valves, x-ray tubes, switches, batteries, rectifiers, etc.; as a catalyst for the production of chlorine and alkali and the production of acetic acid and acetaldehyde from acetylene; chemical, physical and biological laboratory research; gold, silver, bronze and tin plating; tanning and currying; feltmaking; taxidermy; textile manufacture; photography and photogravure; mercury-based paints and pigments; and the manufacture of artificial silk. Some of these uses have been discontinued because of the toxic effects that the mercury exposure exerted upon workers.

Organic Mercury Compounds

Organic compounds of mercury may be considered as the organic compounds in which the mercury is chemically linked directly to a carbon atom. Carbon-mercury bonds have a wide range of stability; in general, the carbon-to-mercury bond in aliphatic compounds is more stable than that in aromatic compounds. According to one reliable estimate, more than 400 phenyl mercurials and at least that number of alkyl mercury compounds have been synthesized. The three most important groups in common usage are the alkyls, the aromatic hydrocarbons or aryls and the alkoxyalkyls. Examples of aryl mercury compounds are phenylmercuric acetate (PMA), nitrate, oleate, propionate and benzoate. Most available information is about PMA.

Uses. All the important uses of the organic mercury compounds depend on the biological activity of these substances. In medical practice organic mercury compounds are used as antiseptics, germicides, diuretics and contraceptives. In the field of pesticides they serve as algicides, fungicides, herbicides, slimacides and as preservatives in paints, waxes and pastes; they are used for mildew suppression, in antifouling paints, in latex paints and in the fungus-proofing of fabrics, paper, cork, rubber and wood

for use in humid climates. In the chemical industry they act as catalysts in a number of reactions and the mercury alkyls are used as alkylating agents in organic syntheses.

Hazards

Absorption and effects: Inorganic and metallic mercury

Vapour inhalation is the main route for the entry of metallic mercury into the body. Around 80% of inhaled mercury vapour is absorbed in the lung (alveoli). Digestive absorption of metallic mercury is negligible (lower than 0.01% of the administered dose). Subcutaneous penetration of metallic mercury as the result of an accident (e.g. the breakage of a thermometer) is also possible.

The main routes of entry of inorganic mercury compounds (mercury salts) are the lungs (atomization of mercury salts) and the gastrointestinal tract. In the latter case, absorption is often the result of accidental or voluntary ingestion. It is estimated that 2 to 10% of ingested mercury salts are absorbed through the intestinal tract.

Skin absorption of metallic mercury and certain of its compounds is possible, although the rate of absorption is low. After entry into the body, metallic mercury continues to exist for a short time in metallic form, which explains its penetration of the blood-brain barrier. In blood and tissues metallic mercury is rapidly oxidized to Hg^{2+} mercury ion, which fixes to proteins. In the blood, inorganic mercury is also distributed between plasma and red blood cells.

The kidney and brain are the sites of deposition following exposure to metallic mercury vapours, and the kidney following exposure to inorganic mercury salts.

Acute poisoning

The symptoms of acute poisoning include pulmonary irritation (chemical pneumonia), perhaps leading to acute pulmonary oedema. Renal involvement is also possible. Acute poisoning is more often the result of accidental or voluntary ingestion of a mercury salt. This leads to severe inflammation of the gastrointestinal tract followed rapidly by renal insufficiency due to necrosis of the proximal convoluted tubules.

The severe chronic form of mercury poisoning encountered in places like Almadén up until the early 20th century, and which presented spectacular renal, digestive, mental and nervous disorders and terminated in cachexia, was eliminated by means of preventive measures. However, a chronic, “intermittent” poisoning in which periods of active intoxication are interspersed between periods of latent intoxication can still be detected among mercury miners. In the latent periods, symptoms remit to such a degree that they are visible only on close search; only the neurological manifestations persist in the form of profuse sweating, dermographia and, to some extent, emotional instability.

A condition of “micromercurialism” characterized by functional neurosis (frequent hysteria, neurasthenia, and mixed forms), cardiovascular lability and secretory neurosis of the stomach has also been described.

Digestive system. Gingivitis is the most common gastrointestinal disorder encountered in mercury poisoning. It is favoured by poor oral hygiene and is accompanied by an unpleasant, metallic or bitter taste in the mouth. Ulceromembranous stomatitis is much less common and is normally found in persons already suffering from gingivitis who have accidentally inhaled mercury vapours. This stomatitis commences with the subjective symptoms of gingivitis with increased salivation (mercurial ptyalism) and coating of the tongue. Eating and drinking produce a burning sensation and discomfort in the mouth, the gums become increasingly inflamed and swollen, ulcers appear and there is spontaneous bleeding. In acute cases, there is high fever, inflammation of the submaxillary ganglions and extremely fetid breath. Alveolodental periostitis has also been observed.

There may be a bluish line on the tooth edge of the gums, in particular in the vicinity of infected areas; this line is, however, never encountered in persons without teeth. Slate-grey punctiform pigmentation of the oral mucosae—the vestibular side of the gums (usually those of the lower jaw), the palate, and even the inside of the cheeks—has also been observed.

Recurrent gingivitis affects the supporting tissues of the teeth, and in many cases the teeth have to be extracted or merely fall out. Other gastrointestinal disorders encountered in mercury poisoning include gastritis and gastroduodenitis.

Non-specific pharyngitis is relatively common. A rarer manifestation is that of Kussmaul’s pharyngitis which presents as a bright-red coloration of the pharynx, tonsils and soft palate with fine arborisation.

Nervous system involvement may occur with or without gastrointestinal symptoms and may evolve in line with two main clinical pictures: (a) fine-intention tremor reminiscent of that encountered in persons suffering from multiple sclerosis; and (b) Parkinsonism with tremor at rest and reduced motor function. Usually one of these two conditions is dominant in the over-all clinical picture which may be further complicated by morbid irritability and pronounced mental hyperactivity (mercurial erethism).

Mercurial Parkinsonism presents a picture of unsteady and staggering gait, absence of balance-recovery reflexes and hypotonia; vegetative symptoms are slight with mask-like facies, sialorrhea, etc. However, Parkinsonism is usually encountered in milder forms, in particular as micro-Parkinsonism.

The most frequently encountered symptoms resemble those presented by persons with multiple sclerosis, except that there is no nystagmus and the two conditions have a different serology and different clinical courses. The most striking feature is tremor which is usually a late symptom but may develop prior to stomatitis.

Tremor usually disappears during sleep, although sudden generalized cramps or contractions may occur; however, it always increases under emotional stress and this is such a characteristic feature that it provides firm grounds for a diagnosis of mercury poisoning. Tremor is particularly pronounced in situations where the patient feels embarrassed or ashamed; often he or she will have to eat in solitude since otherwise he would be incapable of raising food to his lips. In its most acute form, the tremor may invade all the voluntary muscles and be continuous. Cases still occur in which the patient has to be strapped down to prevent him falling out of bed; such cases also present massive, choreiform movements sufficient to wake the patient from his sleep.

The patient tends to utter his words in staccato fashion, so that his sentences are difficult to follow (psellismus mercurialis); when a spasm ceases, the words come out too fast. In cases more reminiscent of parkinsonism, speech is slow and monotonous and the voice may be low or completely absent; spasmodic utterence is, however, more common.

A highly characteristic symptom is a desire for sleep, and the patient often sleeps for long periods although lightly and is frequently disturbed by cramps and spasms. However, insomnia may occur in some cases.

Loss of memory is an early and dementia a terminal symptom. Dermographia and profuse sweating (for no obvious reason) are frequently encountered. In chronic mercury poisoning, the eyes may show the picture of “mercurialentis” characterized by a light-grey to dark, reddish-grey discoloration of the anterior capsule of the crystalline lens due to the deposition of finely divided particles of mercury. Mercurialentis can be detected by examination with a slit-lamp microscope and is bilateral and symmetrical; it usually appears some considerable time before the onset of general signs of mercury poisoning.

Chronic exposure

Chronic mercury poisoning usually starts insidiously, which makes the early detection of incipient poisoning difficult. The main target organ is the nervous system. Initially, suitable tests can be used to detect psychomotor and neuro-muscular changes and slight tremor. Slight renal involvement (proteinuria, albuminuria, enzymuria) may be detectable earlier than neurological involvement.

If excessive exposure is not corrected, neurological and other manifestations (e.g., tremor, sweating, dermatography) become more pronounced, associated with changes in behaviour and personality disorders and, perhaps, digestive disorders (stomatitis, diarrhoea) and a deterioration in general status (anorexia, weight loss). Once this stage has been reached, termination of exposure may not lead to total recovery.

In chronic mercury poisoning, digestive and nervous symptoms predominate and, although the former are of earlier onset, the latter are more obvious; other significant but less intense symptoms may be present. The duration of the period of mercury absorption preceding the appearance of clinical symptoms depends on the level of absorption and individual factors. The main early signs include slight digestive disorders, in particular, loss of appetite; intermittent tremor, sometimes in specific muscle groups; and neurotic disorders varying in intensity. The course of intoxication may vary considerably from case to case. If exposure is terminated immediately upon the appearance of the first symptoms, full recovery usually occurs; however, if exposure is not terminated and the intoxication becomes firmly established, no more than an alleviation of symptoms can be expected in the majority of cases.

Kidney. There have been studies over the years on the relationships between renal function and urinary mercury levels. The effects of low-level exposures are still not well documented or understood. At higher levels (above 50 µg/g (micrograms per gram) abnormal renal function (as evidenced by N-acetyl-B-D-glucosaminidase (NAG), which is a sensitive indicator of damage to the kidneys) have been

observed. The NAG levels were correlated with both the urinary mercury levels and the results of neurological and behavioural testing.

Nervous system. Recent years have seen the development of more data on low levels of mercury, which are discussed in more detail in the chapter Nervous system in this Encyclopaedia.

Blood. Chronic poisoning is accompanied by mild anaemia sometimes preceded by polycythaemia resulting from bone marrow irritation. Lymphocytosis and eosinophilia have also been observed.

Organic Mercury Compounds

Phenylmercuric acetate (PMA). Absorption may occur through inhalation of aerosols containing PMA, through skin absorption or by ingestion. The solubility of the mercurial and the particle size of the aerosols are determining factors for the extent of absorption. PMA is more efficiently absorbed by ingestion than are inorganic mercuric salts. Phenylmercury is transported mainly in blood and distributed in the blood cells (90%), accumulates in the liver and is there decomposed into inorganic mercury. Some phenylmercury is excreted in the bile. The main portion absorbed in the body is distributed in the tissues as inorganic mercury and accumulated in the kidney. On chronic exposure, mercury distribution and excretion follow the pattern seen on exposure to inorganic mercury.

Occupational exposure to phenylmercury compounds occurs in the manufacture and handling of products treated with fungicides containing phenylmercury compounds. Acute inhalation of large amounts may cause lung damage. Exposure of the skin to a concentrated solution of phenylmercury compounds may cause chemical burns with blistering. Sensitization to phenylmercury compounds may occur. Ingestion of large amounts of phenylmercury may cause renal and liver damage. Chronic poisoning gives rise to renal damage due to accumulation of inorganic mercury in the renal tubules.

Available clinical data do not permit extensive conclusions about dose-response relationships. They suggest, however, that phenylmercury compounds are less toxic than inorganic mercury compounds or long-term exposure. There is some evidence of mild adverse effects on the blood.

Alkyl mercury compounds. From a practical point of view, the short-chained alkyl mercury compounds, like methylmercury and ethylmercury, are the most important, although some exotic mercury compounds, generally used in laboratory research, have led to spectacular rapid deaths from acute poisoning. These compounds have been extensively used in seed treatment where they have been responsible for a number of fatalities. Methylmercuric chloride forms white crystals with a characteristic odour, while ethylmercury chloride; (chloroethylmercury) forms white flakes. Volatile methylmercury compounds, like methylmercury chloride, are absorbed to about 80% upon inhalation of vapour. More than 95% of short-chained alkyl mercury compounds is absorbed by ingestion, although the absorption of methylmercury compounds by the skin can be efficient, depending on their solubility and concentration and the condition of the skin.

Transport, distribution and excretion. Methylmercury is transported in the red blood cells (95%), and a small fraction is bound to plasma proteins. The distribution to the different tissues of the body is rather slow and it takes about four days before equilibrium is obtained. Methylmercury is concentrated in the

central nervous system and especially in grey matter. About 10% of the body burden of mercury is found in the brain. The highest concentration is found in the occipital cortex and the cerebellum. In pregnant women methylmercury is transferred in the placenta to the foetus and especially accumulated in the foetal brain.

Hazards of organic mercury

Poisoning by alkyl mercury may occur on inhalation of vapour and dust containing alkyl mercury and in the manufacture of the mercurial or in handling the final material. Skin contact with concentrated solutions results in chemical burns and blistering. In small agricultural operations there is a risk of exchange between treated seed and products intended for food, followed by involuntary intake of large amounts of alkyl mercury. On acute exposure the signs and symptoms of poisoning have an insidious onset and appear with a latency period which may vary from one to several weeks. The latency period is dependent on the size of the dose—the larger the dose, the shorter the period.

On chronic exposure the onset is more insidious, but the symptoms and signs are essentially the same, due to the accumulation of mercury in the central nervous system, causing neuron damage in the sensory cortex, such as visual cortex, auditory cortex and the pre- and post-central areas. The signs are characterized by sensory disturbances with paresthaesia in the distal extremities, in the tongue and around the lips. With more severe intoxications ataxia, concentric constrictions of the visual fields, impairment of hearing and extrapyramidal symptoms may appear. In severe cases chronic seizures occur.

The period in life most sensitive to methylmercury poisoning is the time in utero; the foetus seems to be between 2 and 5 times more sensitive than the adult. Exposure in utero results in cerebral palsy, partly due to inhibition of the migration of neurons from central parts to the peripheral cortical areas. In less severe cases retardation in the psychomotor development has been observed.

Alkoxyalkyl mercury compounds. The most common alkoxyalkyl compounds used are methoxyethyl mercury salts (e.g., methoxyethylmercury acetate), which have replaced the short-chain alkyl compounds in seed treatment in many industrial countries, in which the alkyl compounds have been banned due to their hazardousness.

The available information is very limited. Alkoxyalkyl compounds are absorbed by inhalation and by ingestion more efficiently than inorganic mercury salts. The distribution and excretion patterns of absorbed mercury follow those of inorganic mercury salts. Excretion occurs through the intestinal tract and the kidney. To what extent unchanged alkoxyalkyl mercury is excreted in humans is unknown. Exposure to alkoxyalkyl mercury compounds can occur in the manufacture of the compound and in handling the final product(s) treated with the mercurial. Methoxyethyl mercury acetate is a vesicant when applied in concentrated solutions to the skin. Inhalation of methoxyethyl mercury salt dust may cause lung damage, and chronic poisoning due to long-term exposure may give rise to renal damage.

Safety and Health Measures

Efforts should be made to replace mercury with less hazardous substances. For example, the felt industry may employ non-mercurial compounds. In mining, wet drilling techniques should be used.

Ventilation is the main safety measure and if it is inadequate, the workers should be provided with respiratory protective equipment.

In industry, wherever possible, mercury should be handled in hermetically sealed systems and extremely strict hygiene rules should be applied at the workplace. When mercury is spilt, it very easily infiltrates into crevices, gaps in the floor and workbenches. Due to its vapour pressure, a high atmospheric concentration may occur even following seemingly negligible contamination. It is therefore important to avoid the slightest soiling of work surfaces; these should be smooth, non-absorbent and slightly tilted towards a collector or, failing this, have a metal grill over a gutter filled with water to collect any drops of spilt mercury which fall through the grill. Working surfaces should be cleaned regularly and, in the event of accidental contamination, any drops of mercury collected in a water trap should be drawn off as rapidly as possible.

Where there is a danger of mercury volatilizing, local exhaust ventilation (LEV) systems should be installed. Admittedly, this is a solution which is not always applicable, as is the case in premises producing chlorine by the mercury cell process, in view of the enormous vaporization surface.

Work posts should be planned in such a way as to minimize the number of persons exposed to mercury.

Most exposure to organic mercury compounds involves mixed exposure to mercury vapour and the organic compound, as the organic mercury compounds decompose and release mercury vapour. All technical measures pertaining to exposure to mercury vapour should be applied for exposure to organic mercury compounds. Thus, contamination of clothes and/or parts of the body should be avoided, as it may be a dangerous source of mercury vapour close to the breathing zone. Special protective work clothes should be used and changed after the workshift. Spray painting with paint containing mercurials requires respiratory protective equipment and adequate ventilation. The short-chained alkyl mercury compounds should be eliminated and replaced whenever possible. If handling cannot be avoided, an enclosed system should be used, combined with adequate ventilation, to limit exposure to a minimum.

Great care must be exercised in preventing the contamination of water sources with mercury effluent since the mercury can be incorporated into the food chain, leading to disasters such as that which occurred in Minamata, Japan.

MOLYBDENUM

Occurrence and Uses

Molybdenum (Mo) is widely distributed throughout the earth’s crust, but it is mined in only a limited number of countries due to the rarity of bodies of sufficiently high quality molybdenite ore ($MoSO_2$). A certain amount of molybdenum is obtained as a by-product in the processing of copper ore. Coal-electrical power plants can be significant sources of molybdenum. Molybdenum is an essential trace element.

Molybdenum forms a large variety of commercially useful compounds in which it displays the valence numbers 0, +2, +3, +4, +5 and +6. It readily changes valence states (disproportionates) with only minor

changes in external conditions. It has a strong tendency to form complexes; with the exception of the sulphides and halides, very few other simple compounds of molybdenum exist. The +6 molybdenum forms isopoly- and heteropoly- acids.

Over 90% of the molybdenum produced is used as an alloying element for iron, steel and non-ferrous metals, mainly because of its heat-resisting properties; the rest is used in chemicals and lubricants. As a steel alloy, molybdenum is utilized in the electric, electronics, military and automobile industries and in aeronautical engineering. Another important use of molybdenum is in the production of inorganic molybdenum pigments, dyes and lakes. Small but increasing amounts of molybdenum are used as trace elements in fertilizers.

The most important molybdenum chemical is molybdenum trioxide (MoO_3), made from roasting the sulphide ore. Pure molybdenum trioxide is used in chemical and catalyst manufacture. The technical product is added to steel as an alloying agent. Molybdenum trioxide also serves as a catalyst in the petroleum industry and as a component of ceramics, enamels and pigments. Molybdenum disulphide (MoS_2) is employed as a heat-resistant lubricant or a lubricant additive. Molybdenum hexacarbonyl ($Mo(CO)_6$) is the starting product for the manufacture of organomolybdenum dyes. It is increasingly used for molybdenum plating by thermal decomposition.

Molybdenum compounds are widely used as catalysts or catalyst activators or promoters, especially for hydrogenation-cracking, alkylation and reforming in the petroleum industry. They are employed as laboratory reagents (phosphomolybdates). In addition, molybdenum compounds are used in electroplating and in tanning.

Hazards

In the processing and industrial utilization of molybdenum and its compounds there may be exposure to dusts and fumes of molybdenum and its oxides and sulphides. This exposure may occur, especially where high-temperature treatment is being carried out as, for example, in an electric furnace. Exposure to molybdenum disulphide lubricant spray, molybdenum hexacarbonyl and its breakdown products during molybdenum plating, molybdenum hydroxide ($Mo(OH)_3$) mist during electroplating, and molybdenum trioxide fumes which sublime above 800 °C may all prove hazardous to health.

Molybdenum compounds are highly toxic based on animal experiments. Acute poisoning causes severe gastrointestinal irritation with diarrhoea, coma and deaths from heart failure. Pneumoconiosis-like effects in the lungs have been reported in animal studies. Workers exposed to pure molybdenum or to molybdenum oxide (MoO_3) (concentration of 1 to 19 mg Mo/m^3) over a period of 3 to 7 years have suffered from pneumoconiosis. Inhalation of molybdenum dust from alloys or carbides can cause “hard metal lung disease”.

There is a wide degree of variation in the hazard resulting from exposure. Insoluble molybdenum compounds (e.g., molybdenum disulphide and many of the oxides and halides) are characterized by

low toxicity; however, the soluble compounds (i.e., those in which molybdenum is an anion, such as sodium molybdenate—$Na_2MoO_4 \cdot 2H_2O$) are considerably more toxic and should be handled with care.

Likewise, precautions should be taken to prevent over-exposure to freshly generated molybdenum fumes as in the thermal decomposition of molybdenum hexacarbonyl.

Exposure to molybdenum trioxide produces irritation of the eyes and the mucous membranes of the nose and throat. Anaemia is a characteristic feature of molybdenum toxicity, with low haemoglobin concentrations and reduced red-cell counts.

High dietary levels of molybdenum in cattle were found to produce deformities in the joints of the extremities. Among chemists handling molybdenum and tungsten solutions, an abnormally high frequency of cases of gout have been reported, and a correlation has been found between the content of molybdenum in food, the incidence of gout, uricaemia and xanthine oxidase activity.

Safety Measures

While working with molybdenum in industry, proper local exhaust ventilation should be employed to collect fumes at their source. Respirators may be worn when engineering and work practices have failed, when such controls are in the process of being installed, for operations requiring entry into tanks or closed vessels, or in emergencies. In the paint, printing and coatings industries, local and general exhaust ventilation as well as safety glasses, protective clothing, face shields and acceptable respirators should be used to reduce exposure for workers handling molybdenum-based dry ingredients for inorganic and organic colours.

NICKEL

Nickel (Ni) compounds of interest include nickel oxide (NiO), nickel hydroxide ($Ni(OH)_2$), nickel subsulphide (Ni_3S_2), nickel sulphate ($NiSO_4$) and nickel chloride ($NiCl_2$). Nickel carbonyl ($Ni(CO)_4$) is considered in a separate article on metal carbonyls.

Occurrence and Uses

Nickel (Ni) comprises 5 to 50% of the weight of meteorites and is found in ores in combination with sulphur, oxygen, antimony, arsenic and/or silica. Ore deposits of commercial importance are principally oxides (e.g., laterite ores containing mixed nickel/iron oxides) and sulphides. Pentlandite ($(NiFe)_9S_8$), the major sulphide mineral, is commonly deposited in association with pyrrhotite (Fe_7S_6), chalcopyrite ($CuFeS_2$) and small amounts of cobalt, selenium, tellurium, silver, gold and platinum. Substantial deposits of nickel ores are found in Canada, Russia, Australia, New Caledonia, Indonesia and Cuba.

Since nickel, copper and iron occur as distinct minerals in the sulphide ores, mechanical methods of concentration, such as flotation and magnetic separation, are applied after the ore has been crushed and ground. The nickel concentrate is converted to nickel sulphide matte by roasting or sintering. The matte is refined by electrowinning or by the Mond process. In the Mond process, the matte is ground,

calcined and treated with carbon monoxide at 50 °C to form gaseous nickel carbonyl ($Ni(CO)_4$), which is then decomposed at 200 to 250 °C to deposit pure nickel powder. Worldwide production of nickel is approximately 70 million kg/year.

More than 3,000 nickel alloys and compounds are commercially produced. Stainless steel and other Ni-Cr-Fe alloys are widely used for corrosion-resistant equipment, architectural applications and cooking utensils. Monel metal and other Ni-Cu alloys are used in coinage, food-processing machinery and dairy equipment. Ni-Al alloys are used for magnets and catalyst production (e.g., Raney nickel). Ni-Cr alloys are used for heating elements, gas turbines and jet engines. Alloys of nickel with precious metals are used in jewellery. Nickel metal, its compounds and alloys have many other uses, including electroplating, magnetic tapes and computer components, arc-welding rods, surgical and dental prostheses, nickel-cadmium batteries, paint pigments (e.g., yellow nickel titanate), moulds for ceramic and glass containers, and catalysts for hydrogenation reactions, organic syntheses and the final methanation step of coal gasification. Occupational exposures to nickel also occur in recycling operations, since nickel-bearing materials, especially from the steel industry, are commonly melted, refined and used to prepare alloys similar in composition to those that entered the recycling process.

Hazards

Human health hazards from occupational exposures to nickel compounds generally fall into three major categories:

1. allergy
2. rhinitis, sinusitis and respiratory diseases
3. cancers of the nasal cavities, lungs and other organs.

The health hazards from nickel carbonyl are considered separately, in the article on metal carbonyls.

Allergy. Nickel and nickel compounds are among the most common causes of allergic contact dermatitis. This problem is not limited to persons with occupational exposure to nickel compounds; dermal sensitization occurs in the general population from exposures to nickel-containing coins, jewellery, watch cases and clothing fasteners. In nickel-exposed persons, nickel dermatitis usually begins as a papular erythema of the hands. The skin gradually becomes eczematous, and, in the chronic stage, lichenification frequently develops. Nickel sensitization sometimes causes conjunctivitis, eosinophilic pneumonitis, and local or systemic reactions to nickel-containing implants (e.g., intraosseous pins, dental inlays, cardiac valve prostheses and pacemaker wires). Ingestion of nickel-contaminated tap water or nickel-rich foods can exacerbate hand eczema in nickel-sensitive persons.

Rhinitis, sinusitis and respiratory diseases. Workers in nickel refineries and nickel electroplating shops, who are heavily exposed to inhalation of nickel dusts or aerosols of soluble nickel compounds, may develop chronic diseases of the upper respiratory tract, including hypertrophic rhinitis, nasal sinusitis, anosmia, nasal polyposis and perforation of the nasal septum. Chronic diseases of the lower respiratory tract (e.g., bronchitis, pulmonary fibrosis) have also been reported, but such conditions are infrequent. Rendall et al. (1994) reported the fatal acute exposure of a worker to inhalation of particulate nickel

from a metal arc process; the authors stressed the importance of wearing protective equipment while using metal arc processes with nickel wire electrodes.

Cancer. Epidemiological studies of nickel-refinery workers in Canada, Wales, Germany, Norway and Russia have documented increased mortality rates from cancers of the lung and nasal cavities. Certain groups of nickel-refinery workers have also been reported to have increased incidences of other malignant tumours, including carcinomas of the larynx, kidney, prostate or stomach, and sarcomas of soft tissues, but the statistical significance of these observations is questionable. The increased risks of cancers of the lungs and nasal cavities have occurred primarily among workers in refinery operations that entail high nickel exposures, including roasting, smelting and electrolysis. Although these cancer risks have generally been associated with exposures to insoluble nickel compounds, such as nickel subsulphide and nickel oxide, exposures to soluble nickel compounds have been implicated in electrolysis workers.

Epidemiological studies of cancer risks among workers in nickel-using industries have generally been negative, but recent evidence suggests slightly increased lung cancer risks among welders, grinders, electroplaters and battery makers. Such workers are often exposed to dusts and fumes that contain mixtures of carcinogenic metals (e.g., nickel and chromium, or nickel and cadmium). Based on an evaluation of epidemiological studies, the International Agency for Research on Cancer (IARC) concluded in 1990: “There is sufficient evidence in humans for the carcinogenicity of nickel sulphate and of the combinations of nickel sulphides and oxides encountered in the nickel refining industry. There is inadequate evidence in humans for the carcinogenicity of nickel and nickel alloys.” Nickel compounds have been classified as carcinogenic to humans (Group 1), and metallic nickel as possibly carcinogenic to humans (Group 2B).

Renal effects. Workers with high exposures to soluble nickel compounds may develop renal tubular dysfunction, evidenced by increased renal excretion of β2-microglobulin (β2M) and N-acetyl-glucosaminidase (NAG).

Safety and Health Measures

A general protocol for health surveillance of workers exposed to nickel was proposed in 1994 by the Nickel Producers Evironmental Research Association (NiPERA) and the Nickel Development Institute (NiDI). The key elements are as follows:

Pre-placement assessment. The goals of this examination are to identify pre-existing medical conditions that may influence hiring and job placement, and to provide baseline data for subsequent functional, physiological or pathological changes. The assessment includes (i) detailed medical and occupational history, focusing on lung problems, exposures to lung toxins, past or present allergies (particularly to nickel), asthma and personal habits (e.g., smoking, alcohol consumption), (ii) complete physical examination, with attention to respiratory and skin problems and (iii) determination of the respiratory protective equipment that may be worn.

Chest x ray, pulmonary function tests, audiometric tests and vision tests may be included. Skin patch testing for nickel sensitivity is not routinely performed, because such tests could possibly sensitize the

subject. If the organization conducts a biological monitoring programme for nickel-exposed workers (see below), baseline nickel concentrations in urine or serum are obtained during the pre-placement assessment.

Periodic assessment. The goals of periodic medical examinations, typically performed annually, are to monitor the worker's general health and to address nickel-associated concerns. The examination includes the history of recent illnesses, symptom review, physical examination and re-evaluation of the worker's ability to use the respiratory protective equipment required for particular tasks. Pulmonary symptoms are assessed by a standard questionnaire for chronic bronchitis. Chest x ray may be legally required in some countries; pulmonary function tests (e.g., forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) are generally left to the physician's discretion. Periodic cancer detection procedures (e.g., rhinoscopy, nasal sinus x rays, nasal mucosal biopsy, exfoliative cytological studies) may be indicated in workers with high-risk exposures in nickel refining.

Biological monitoring. Analyses of nickel concentrations in urine and serum samples may reflect the recent exposures of workers to metallic nickel and soluble nickel compounds, but these assays do not furnish reliable measures of the total body nickel burden. The uses and limitations of biological monitoring of nickel-exposed workers have been summarized by Sunderman et al. (1986). A technical report on analysis of nickel in body fluids was issued in 1994 by the Commission on Toxicology of the International Union of Pure and Applied Chemistry (IUPAC). The National Maximum Workplace Concentration Committee (NMWCC) of the Netherlands proposed that urine nickel concentration 40 µg/g creatinine, or serum nickel concentration 5 µg/l (both measured in samples obtained at the end of a working week or a work shift) be considered warning limits for further investigation of workers exposed to nickel metal or soluble nickel compounds. If a biological monitoring programme is implemented, it should augment an environmental monitoring programme, so that biological data are not used as a surrogate for exposure estimates. A standard method for the analysis of nickel in workplace air was developed in 1995 by the UK Health and Safety Executive.

Treatment. When a group of workers accidently drank water heavily contaminated with nickel chloride and nickel sulphate, conservative treatment with intravenous fluids to induce diuresis was effective (Sunderman et al. 1988). The best therapy for nickel dermatitis is avoidance of exposure, with special attention to work hygienic practices. Therapy of acute nickel carbonyl poisoning is discussed in the article on metal carbonyls.

NIOBIUM

Occurrence and Uses

Niobium (Nb) is found together with other elements including titanium (Ti), zirconium (Zr), tungsten (W), thorium (Th) and uranium (U) in ores such as tantalite-columbite, fergusonite, samarskite, pyrochlore, koppite and loparite. The largest deposits are located in Australia and Nigeria, and during the last few years extensive deposits have been discovered in Uganda, Kenya, Tanzania and Canada.

Niobium is widely used in the electrovacuum industry and also in the manufacture of anodes, grids, electrolytic condensers and rectifiers. In chemical engineering, niobium is used as a corrosion-proof material for heat exchangers, filters, needle valves and so on. High-quality cutting tools and magnetic materials are made from niobium alloys. Ferroniobium alloy is used in thermonuclear appliances.

Niobium and its refractory alloys are utilized in the field of rocket technology, in the supersonic aircraft industry, interplanetary flight equipment and in satellites. Niobium is also used in surgery.

Hazards

During the mining and concentration of niobium ore and processing of the concentrate, the workers may be exposed to general hazards, such as dust and fumes, which are typical for these operations. In the mines, the action of dust may be aggravated by exposure to radioactive substances such as thorium and uranium.

Toxicity

Much of the information about the behaviour of niobium in the body is based on studies of the radioisotope pair ^{95}Zr-^{95}Nb, a common nuclear fission product. ^{95}Nb is the daughter of ^{95}Zr. One study investigated cancer incidence among niobium mine workers exposed to radon and thoron daughters and found an association between lung cancer and cumulative alpha-radiation.

Intravenous and intraperitoneal injections of niobium (radioactive) and its compounds showed a fairly uniform distribution through the organism, with a tendency to accumulate in the liver, kidneys, spleen and bone marrow. The elimination of radioactive niobium from the organism can be hastened appreciably by the injection of massive doses of zirconium nitrate. After intraperitoneal injections of stable niobium in the form of potassium niobate, the LD_{50} for rats was 86 to 92 mg/kg and for mice 13 mg/kg. Metallic niobium is not absorbed from the stomach or intestines. The LD_{50} of niobium pentachloride in these organs was 940 mg/kg for rats, while the corresponding figure for potassium niobate was 3,000 mg/kg. Niobium compounds administered intravenously, intraperitoneally or per os produce a particularly pronounced effect on the kidneys. This effect can be attenuated by preventive medication with ascorbic acid. Oral intake of niobium pentachloride furthermore causes acute irritation of the mucous membranes of the gullet and stomach, and liver changes; chronic exposure during 4 months causes temporary blood changes (leukocytosis, prothrombin deficiency).

Inhaled niobium is retained in the lung, which is the critical organ for dust. Daily inhalation of niobium nitride dust at a concentration of 40 mg/m^3 of air leads within a few months to signs of pneumoconiosis (while there are no noticeable signs of toxic action): thickening of the interalveolar septa, development of considerable amounts of collagenous fibres in the peribronchial and perivascular tissue, and desquamation of the bronchial epithelium. Analogous changes develop upon intratracheal administration of niobium pentoxide dust; in this case dust is found even in the lymph nodes.

Safety and Health Measures

Atmospheric concentrations of the aerosols of niobium alloys and compounds that contain toxic elements such as fluorine, manganese and beryllium, should be strictly controlled. During the mining and concentration of niobium ore containing uranium and thorium, the worker should be protected against radioactivity. Proper engineering design including adequate ventilation with fresh air is necessary to control dust in mine air. In the extraction of pure niobium from its compounds by powder metallurgy, the workplaces must be kept free from niobium dust and fumes, and workers must be protected against chemicals such as caustic alkalis and benzene. In addition, regular medical examinations which include lung function tests are recommended.

OSMIUM

Occurrence and Uses

Osmium (Os) is found almost exclusively in osmiridium, a natural alloy consisting of osmium and iridium, and in all platinum ores. The major ore deposits are located in the Urals, Canada and Colombia, with less important ores in Australia and in Alaska, California and Oregon in the United States.

Osmium alloys readily with the other platinum metals and with iron, cobalt and nickel. It also forms brittle intermetallic compounds with tin and zinc. One of the distinctive features of osmium is the ease with which it forms osmium tetroxide (OsO_4). Osmium powder always has the characteristic odour of its tetroxide because even at normal temperatures it oxidizes in air to OsO_4, even if only to a slight degree. The tetroxide is extremely volatile and has an unpleasant odour, from which the name of the element was derived (osme=odour). It is a powerful oxidizer and is easily converted to osmium dioxide (OsO_2) or even to metallic osmium. With alkalis it forms unstable compounds such as $OsO_4 \cdot 2KOH$. When heated, osmium readily forms osmium disulphide (OsS_2). The fluorides OsF_4, OsF_6 and OsF_8 are also formed. Various chlorides are formed when osmium is treated with chlorine at high temperatures. With carbon monoxide, it forms carbonyls. It also forms a number of compounds with the complex anion containing osmium, as for example ammonium osmium hexachloride ($(NH_4)_2OsCl_6$).

Osmium is used as a catalyst in the synthesis of ammonia and in the hydrogenation of organic compounds. As an alloy with indium it is used for the manufacture of compass needles and fine machine bearings. It is found in the parts of watch and lock mechanisms and in fountain pen points. Osmium tetroxide, sometimes incorrectly termed osmic acid, is used as an oxidizing agent, particularly for converting olefins to glycols. The chloro-osmiates are used in place of gold salts in photography.

Hazards

The metal is innocuous, but persons engaged in its production are exposed to the effects of vapours from acids and chlorine. Osmium tetroxide vapours are poisonous and extremely irritating to the eyes

even at low concentrations, causing weeping and conjunctivitis, and to the upper respiratory system, causing bronchitis, bronchial spasms and difficulty in breathing, which may last for several hours. Longer exposure can result in damage to the cornea, blindness, disturbances of the digestive system and inflammatory disorders of the lungs and kidneys. Upon contact, it discolours the skin green or black and causes dermatitis and ulceration.

Safety and Health Measures

During the production of osmium, local exhaust ventilation should be provided and the apparatus should be sealed if gaseous chlorine is used. An enclosed ventilated area or hood is necessary in order to control the release of osmium tetroxide vapours into the work environment and prevent eye and respiratory irritation. Exposed workers should wear protective clothing, hand protection, gas-tight chemical safety eye protection and appropriate respiratory protective equipment. Containers must be stored in naturally ventilated premises. The vapour has a pronounced and nauseating odour which should serve as a warning of toxic concentration in the air, and personnel should leave the polluted area immediately. Determination in air and blood is possible by colourimetry of the complex with thiourea.

PALLADIUM

Occurrence and Uses

Palladium (Pd) occurs in nature with platinum or gold, as the selenide. It is found in nickel sulphide ores and in the minerals stibiopalladinite, braggite and porpezite. The concentration of palladium in the Earth’s crust is 0.01 ppm.

Palladium has been used in gold, silver and copper alloys in dentistry. Alloys are also used for bearings, springs and balance wheels in watches. Palladium is used as a catalyst in the manufacture of sulphuric acid. In powder form it serves as a catalyst in hydrogenation. The sponge form is used for separation of hydrogen from a mixture of gases. Silver alloys are used for electrical contacts. Palladium (II) complexes have been studied as antineoplastic drugs.

Palladium chloride ($PdCl_2 \cdot 2H_2O$), or palladous chloride, is used in photography toning solutions and for the manufacture of indelible ink. It is an agent used for transferring pictures to porcelain, for electroplating watch parts, and for finding leaks in buried gas pipes. Palladium chloride is associated with copper chloride in catalyzing the production of acetaldehyde from ethylene.

Palladium oxide (PdO), or palladous oxide, is used as a reduction catalyst in the synthesis of organic compounds. Palladium nitrate ($Pd(NO_3)_2$) is used in the separation of halides. Palladium trifluoride (PdF_3) is an active oxidizing agent.

Hazards

Studies indicate cases of allergy and contact dermatitis caused by palladium in dental alloys and fine jewellery. In one study palladium-based alloys were associated with several cases of stomatitis and oral lichenoid reactions. In this same study palladium allergy occurred mainly in patients with a sensitivity

to nickel. Palladium chloride produces dermatitis and allergic skin sensitization in workers exposed daily. In addition, it should be regarded as an eye irritant. Palladium hydroxide was used in the past to treat obesity by injection; this form of treatment gave rise to localized necrosis and was discontinued.

Safety and Health Measures

Correct exhaust ventilation is necessary when working with palladium and its compounds. Good personal hygiene, proper protective clothing and medical surveillance are important measures in preventing the risks associated with sensitization. Adequate sanitary facilities must be provided.

PLATINUM

Occurrence and Uses

Platinum (Pt) occurs in native form and in a number of mineral forms, including sperrylite ($PtAs_2$), cooperite (Pt,Pd)S and braggite (Pt,Pd,Ni)S. Platinum is sometimes found with palladium as the arsenide and selenide. The concentration of platinum in the Earth's crust is 0.005 ppm.

Platinum and its alloys are used as catalysts in petroleum reformation, ammonia oxidation, sulphur dioxide oxidation, hydrogenation and dehydrogenation. Platinum is used in the control of automotive emissions, in electrical contacts, electrodes and thermocouples. It is also used in spinnerets for fibrous glass and rayon manufacture, in reflecting or ornamental surfaces and in jewellery. Because of the permanence of platinum, it is utilized for national and international standards for weight, length and temperature measurement. Platinum is manufactured into sheet, wire and foil, and it has wide use in laboratory apparatus.

Nickel, osmium, ruthenium, copper, gold, silver and iridium are alloyed with platinum to increase hardness. Commercially important alloys of platinum are prepared with copper, gold, iridium, rhodium and ruthenium. Alloys with cobalt have become important because of their strong ferromagnetic properties.

Chloroplatinic acid, formed when platinum is dissolved in aqua regia, is useful in the manufacture of catalysts. Potassium hexachloroplatinate is used in the photographic industry, and platinum tetrachloride is used as a catalyst in the chemical industry. Platinum hexafluoride is an extremely powerful oxidizing agent, the first substance to oxidize an inert gas (xenon). Cis-Dichlorodiamineplatinum II, a complex of platinum and related congeners, was found to be active against a broad spectrum of animal tumours. It has been found useful in producing remissions with a number of human cancers.

Hazards

The toxic and potentially toxic effects of platinum in workers are believed to be related to certain water-soluble platinum salts (e.g., potassium hexachloroplatinate, potassium tetrachloroplatinate, sodium chloroplatinate and ammonium chloroplatinate). Inhalation exposure to these platinum salts is known to give rise to manifestations of respiratory allergy. The first report of such reactions to platinum compounds appeared in 1911 among photographic workers who suffered respiratory and skin disorders.

Similar clinical manifestations—rhinitis, conjunctivitis, asthma, urticaria and contact dermatitis—have since been reported mainly in platinum refinery workers and chemists. Allergic respiratory diseases have been reported in a high proportion of refinery workers exposed to soluble hexachloroplatinate salts. Allergic rhinitis and bronchitis in 52 of 91 workers from four platinum refineries in Britain have been described, with most severe symptoms amongst the workers crushing the chloroplatinate salts. The term platinosis has been defined as the effects of soluble platinum salts on people exposed to these occupationally and is characterized by pronounced irritation of the nose and upper respiratory passages, with sneezing, running of the eyes, and coughing. Later asthmatic symptoms of cough, tightness of the chest, wheezing and shortness of breath appear. These symptoms become progressively worse with the length of employment. Some workers may show all three allergic manifestations with involvement of the nasal mucosa, bronchi and skin. Reports of allergy among workers exposed to chloroplatinate salts have appeared from the United States, the United Kingdom, Switzerland, Germany and South Africa.

It is of interest to note that anaphylactic reactions have been noted in some patients who have been treated with platinum anti-tumour agents.

In general, the allergic effects of exposure to platinum have been confined to specific platinum complexes. Sensitized workers when tested by pin prick do not respond to the majority of the platinum compounds used in the refinery. Once sensitized the condition persists, and workers generally have to avoid exposure to platinum. Smoking appears to increase the risk of sensitization by platinum salts.

The emissions from catalytic mufflers containing platinum do not appear to present a health hazard from the point of view of the platinum emission.

Safety and Health Measures

Control of platinum hazards can be achieved only by preventing the release of the soluble complex platinum salts to the atmosphere of the workshop. Since platinum dust is more potentially harmful than is the spray, the soluble complex salts should not be dried unless necessary. Good exhaust ventilation is necessary in platinum refineries. Chemical procedures which may generate these salts should be carried out in ventilated fume hoods. Open centrifuges should not be used. Good personal hygiene, proper protective clothing, and medical surveillance are important preventive measures. Workers with a history of allergic or respiratory disease should be advised not to work with soluble platinum compounds.

Pin prick, nasal and bronchial tests have been devised. Skin prick tests with dilute concentrations of the soluble platinum complexes appear to provide reproducible, reliable and highly sensitive biological monitors of allergic response.

RHENIUM

Occurrence and Uses

Rhenium (Re) is found in the combined state in platinum ores, gadolinite, molybdenite (MoS_2) and columbite. It is found in some sulphide ores. It is a rare element making up about 0.001 ppm of the Earth's crust.

Rhenium is used in electron tubes and in semiconductor applications. It is also used as a highly selective catalyst for hydrogenation and dehydrogenation. Rhenium-tagged antibodies have been used experimentally to treat adenocarcinomas of the colon, lung and ovary. Rhenium is used in medical instruments, in high-vacuum equipment, and in alloys for electrical contacts and thermocouples. It is also used for plating of jewellery.

Rhenium is alloyed with tungsten and molybdenum to improve their workability.

Hazards

Chronic toxic manifestations are not known. Some compounds, such as rhenium hexafluoride, are irritating to the skin and eye. In experimental animals, inhalation of rhenium dust causes pulmonary fibrosis. Rhenium VII sulphide ignites spontaneously in air and emits toxic fumes of oxides of sulphur when heated. Hexamethyl rhenium presents a serious explosion hazard and should be handled with extreme caution.

RHODIUM

Occurrence and Uses

Rhodium is one of the rarest elements in the Earth's crust (average concentration 0.001 ppm). It is found in small quantities associated with native platinum and some copper-nickel ores. It occurs in the minerals rhodite, sperrylite and iridosmine (or osmiridium).

Rhodium is used in corrosion-resistant electroplates for protecting silverware from tarnishing and in high-reflectivity mirrors for searchlights and projectors. It is also useful for plating optical instuments and for furnace winding. Rhodium serves as a catalyst for various hydrogenation and oxidation reactions. It is used for spinnerets in rayon production and as an ingredient in gold decorations on glass and porcelain.

Rhodium is alloyed with platinum and palladium to make very hard alloys for use in spinning nozzles.

Hazards

There have been no significant experimental data indicating health problems with rhodium, its alloys or its compounds in humans. Although toxicity is not established, it is necessary to handle these metals carefully. Contact dermatitis in a worker who prepared pieces of metal for plating with rhodium has been reported. The authors argue that the small number of reported cases of sensitization to rhodium may reflect the rarity of use rather than the safety of this metal. The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended a low threshold limit value for

rhodium and its soluble salts, based on analogy with platinum. The ability of soluble salts of rhodium to give rise to allergic manifestations in humans has not been completely demonstrated.

RUTHENIUM

Occurrence and Uses

Ruthenium is found in the minerals osmiridium and laurite, and in platinum ores. It is a rare element comprising about 0.001 ppm of the Earth's crust.

Ruthenium is used as a substitute for platinum in jewellery. It is utilized as a hardener for pen nibs, electrical contact relays and electrical filaments. Ruthenium is also used in ceramic colours and in electroplating. It acts as a catalyst in the synthesis of long-chain hydrocarbons. In addition, ruthenium has been used recently in treating eye uveal malignant melanomas.

Ruthenium forms useful alloys with platinum, palladium, cobalt, nickel and tungsten for better wear resistance. Ruthenium red ($Ru_3Cl_6H_{42}N_4O_2$) or ruthenium oxychloride ammoniated is used as a microscopy reagent for pectin, gum, animal tissues and bacteria. Ruthenium red is an eye inflammatory agent.

Hazards

Ruthenium tetraoxide is volatile and irritating to the respiratory tract.

Some ruthenium electroplating complexes may be skin and eye irritants, but documentation of this is lacking. Ruthenium radioisotopes, chiefly ^{103}Ru and ^{106}Ru, occur as fission products in the nuclear fuel cycle. Since ruthenium may transform to volatile compounds (it forms numerous nitrogen complexes as noted above), there has been concern about its uptake in the environment. The significance of radio-ruthenium as a potential radiation hazard is still largely unknown.

SELENIUM

Occurrence and Uses

Selenium (Se) is found in rocks and soils all over the world. There are no true deposits of selenium anywhere, and it cannot economically be recovered directly. Various estimates for selenium in the Earth's crust range from 0.03 to 0.8 ppm; the highest concentrations known are in native sulphur from volcanoes, which contains up to 8,350 ppm. Selenium does, however, occur together with tellurium in the sediments and sludges left from electrolytic copper refining. The chief world supplies are from the copper-refining industries of Canada, the United States and Zimbabwe, where the slimes contain up to 15% selenium.

The manufacture of selenium rectifiers, which convert alternating current to direct current, accounts for over half the world's production of selenium. Selenium is also used for decolourizing green glass and for making ruby glass. It is an additive in the natural and synthetic rubber industries and an insecticide. Selenium is used for alloying with stainless steel and copper.

^{75}Se is used for the radioactive scanning of the pancreas and for photostat and x-ray xerography. Selenium oxide or selenium dioxide (SeO_2) is produced by burning selenium in oxygen, and it is the most widely used selenium compound in industry. Selenium oxide is employed in the manufacture of other selenium compounds and as a reagent for alkaloids.

Selenium chloride (Se_2Cl_2) is a dark brownish-red stable liquid which hydrolyses in moist air to give selenium, selenious acid and hydrochloric acid. Selenium hexafluoride (SeF_6) is used as a gaseous electric insulator.

Hazards

The elemental forms of selenium are probably completely harmless to humans; its compounds, however, are dangerous and their action resembles that of sulphur compounds. Selenium compounds may be absorbed in toxic quantities through the lungs, intestinal tract or damaged skin. Many selenium compounds will cause intense burns of skin and mucous membranes, and chronic skin exposure to light concentrations of dust from certain compounds may produce dermatitis and paronychia.

The sudden inhalation of large quantities of selenium fumes, selenium oxide or hydrogen selenide may produce pulmonary oedema due to local irritant effects on the alveoli; this oedema may not set in for 1 to 4 hours after exposure. Exposure to atmospheric hydrogen selenide concentrations of 5 mg/m^3 is intolerable. However, this substance occurs in only small amounts in industry (for example, due to bacterial contamination of selenium-contaminated gloves), although there have been reports of exposure to high concentrations following laboratory accidents.

Skin contact with selenium oxide or selenium oxychloride may cause burns or sensitization to selenium and its compounds, especially selenium oxide. Selenium oxychloride readily destroys skin on contact, causing third-degree burns unless immediately removed with water. However, selenium oxide burns are rarely severe and, if properly treated, heal without a scar.

Dermatitis due to exposure to airborne selenium oxide dust usually starts at the points of contact of the dust with the wrist or neck and may extend to contiguous areas of the arms, face and upper portions of the trunk. It usually consists of discrete, red, itchy papules which may become confluent on the wrist, where selenium dioxide is liable to penetrate between the glove and sleeve of the overall. Painful paronychia may also be produced. However, one more frequently sees cases of excruciatingly painful throbbing nail beds, due to the selenium dioxide penetrating under the free edge of the nails, in workers handling selenium dioxide powder or waste red selenium fume powder without wearing impermeable gloves.

Splashes of selenium oxide entering the eye may cause conjunctivitis if not treated immediately. Persons who work in atmospheres containing selenium dioxide dust may develop a condition known among the workers as “rose eye”, a pink allergy of the eyelids, which often become puffy. There is usually also a conjunctivitis of the palpebral conjunctiva but rarely of the bulbar conjunctiva.

The first and most characteristic sign of selenium absorption is a garlic odour of the breath. The odour is probably caused by dimethyl selenium, almost certainly produced in the liver by the detoxication of selenium by methylation. This odour will clear quickly if the worker is removed from exposure, but there is no known treatment for it. A more subtle and earlier indication than the garlic odour is a metallic taste in the mouth. It is less dramatic and is often overlooked by the workers. The other systemic effects are impossible to evaluate accurately and are not specific to selenium. They include pallor, lassitude, irritability, vague gastrointestinal symptoms and giddiness.

The possibility of liver and spleen damage in people exposed to high levels of selenium compounds deserves further attention. In addition, more studies of workers are needed to examine the possible protective effects of selenium against lung cancer.

Safety and Health Measures

Selenium oxide is the main selenium problem in industry since it is formed whenever selenium is boiled in the presence of air. All sources of selenium oxide or fumes should be fitted with exhaust ventilation systems with an air speed of at least 30 m/min. Workers should be provided with hand protection, overalls, eye and face protection, and gauze masks. Supplied-air respiratory protective equipment is necessary in cases where good extraction is not possible, such as in the cleaning of ventilation ducts. Smoking, eating and drinking at the workplace should be prohibited, and dining and sanitary facilities, including showers and locker rooms, should be provided at a point distant from exposure areas. Wherever possible, operations should be mechanized, automated or provided with remote control.

SILVER

Occurrence and Uses

Silver (Ag) is found throughout the world, but most of it is produced in Mexico, the western United States, Bolivia, Peru, Canada and Australia. Much of it is obtained as a by-product from argentiferous lead, zinc and copper ores in which it occurs as the silver sulphide, argentite (Ag_2S). It is also recovered during the treatment of gold ores and is an essential constituent of the gold telluride, calaverite ($(AuAg)Te_2$).

Because pure silver is too soft for coins, ornaments, cutlery, plate and jewellery, silver is hardened by alloying with copper for all these applications. Silver is extremely resistant to acetic acid and, therefore, silver vats are used in the acetic acid, vinegar, cider and brewing industries. Silver is also used in busbars and windings of electrical plants, in silver solders, dental amalgams, high-capacity batteries, engine bearings, sterling ware and in ceramic paints. It is employed in brazing alloys and in the silvering of glass beads.

Silver finds use in the manufacture of formaldehyde, acetaldehyde and higher aldehydes by the catalytic dehydrogenation of the corresponding primary alcohols. In many installations, the catalyst consists of a shallow bed of crystalline silver of extremely high purity. An important use of silver is in

the photography industry. It is the unique and instantaneous reaction of the halides of silver on exposure to light that makes the metal virtually indispensable for films, plates and photographic printing paper.

Silver nitrate ($AgNO_3$) is used in photography, the manufacture of mirrors, silver plating, dyeing, colouring of porcelain, and etching ivory. It is an important reagent in analytical chemistry and a chemical intermediate. Silver nitrate is found in sympathetic and indelible inks. It also serves as a static inhibitor for carpets and woven materials and as a water disinfectant. For medical purposes silver nitrate has been used for the prophylaxis of ophthalmia neonatorum. It has been utilized as an antiseptic, an astringent, and in veterinary use for the treatment of wounds and local inflammations.

Silver nitrate is a powerful oxidizing agent and a fire hazard, in addition to being strongly caustic, corrosive and poisonous. In the form of dust or a solid it is dangerous to the eyes, causing burns of the conjunctiva, argyria and blindness.

Silver oxide (Ag_2O) is used in the purification of drinking water, for polishing and colouring glass yellow in the glass industry, and as a catalyst. In veterinary medicine, it is used as an ointment or solution for general germicidal and parasiticidal purposes. Silver oxide is a powerful oxidizing material and a fire hazard.

Silver picrate ($(O_2N)_3C_6H_2OAg{\cdot}H_2O$) is used as a vaginal antimicrobial. In veterinary medicine it is used against granular vaginitis for cattle. It is highly explosive and poisonous.

Hazards

Silver exposure may lead to a benign condition called “argyria”. If the dust of the metal or its salts is absorbed, silver is precipitated in the tissues in the metallic state and cannot be eliminated from the body in this state. Reduction to the metallic state takes place either by the action of light on the exposed parts of the skin and visible mucous membranes, or by means of hydrogen sulphide in other tissues. Silver dusts are irritants and can lead to ulceration of the skin and nasal septum.

Occupations involving the risk of argyria can be divided into two groups:

1. workers who handle a compound of silver, either the nitrate, fulminate or cyanide, which, broadly speaking, giving rise to generalized argyria from inhalation and ingestion of the silver salt concerned

2. workers who handle metallic silver, small particles of which accidentally penetrate the exposed skin, giving rise to local argyria by a process equivalent to tattooing.

Generalized argyria is unlikely to occur at respirable silver concentrations in air of 0.01 mg/m^3 or at oral cumulative doses lower than 3.8 g. Persons affected by generalized argyria are often called “blue men” by their fellow workers. The face, forehead, neck, hands and forearms develop a dark slatey-grey colour, uniform in distribution and varying in depth depending on the degree of exposure. Pale scars up to about 6 mm across may be found on the face, hands and forearms due to the caustic effects of silver nitrate. The fingernails are a deep chocolate-brown colour. The buccal mucosa is slatey-grey or bluish

in colour. Very slight pigmentation may be detected in the covered parts of the skin. The toenails may show a slight bluish discolouration. In a condition called argyrosis conjunctivae, the colour of the conjunctivae varies from a slight grey to a deep brown, the lower palpebral portion being particularly affected. The posterior border of the lower lid, the caruncle and the plica semilunaris are deeply pigmented and may be almost black. Examination by means of the slit-lamp reveals a delicate network of faint grey pigmentation in the posterior elastic lamina (Descemet's membrane) of the cornea, known as argyrosis corneae. In cases of long duration, argyrolentis is also found.

Where persons work with metallic silver, small particles may accidentally penetrate the exposed skin surface, giving rise to small pigmented lesions by a process equivalent to tattooing. This may occur in occupations involving the filing, drilling, hammering, turning, engraving, polishing, forging, soldering and smelting of silver. The left hand of the silversmith is more affected than the right, and the pigmentation occurs at the site of injuries from instruments. Many instruments, such as engraving tools, files, chisels and drills, are sharp and pointed and are liable to produce skin wounds. The piercing saw, an instrument resembling a fret saw, may break and run into the worker's hand. If the file slips, the worker's hand may be injured on the silver article; this is especially the case with the prongs of forks. A worker drawing silver wire through a hole in a silver draw-plate may get splinters of silver in his or her fingers. The pigmented points vary from tiny specks to areas 2 mm or more in diameter. They may be linear or rounded and in varying shades of grey or blue. The tattoo marks remain for life and cannot be removed. The use of gloves is usually impractical.

Safety and Health Measures

In addition to the engineering measures necessary to keep the airborne concentrations of silver fumes and dust as low as possible and in any case below the exposure limits, medical precautions for preventing argyria have been recommended. These include, in particular, the periodic medical examination of the eye, because the discolouration of the Descemet's membrane is an early sign of the disease. Biological monitoring seems to be possible via the faecal excretion of silver. There is no recognized effective treatment of argyria. The condition seems to stabilize when exposure to silver is discontinued. Some clinical improvement has been achieved by use of chelating agents and intradermal injection of sodium thiosulphate or potassium ferrocyanide. Sun exposure should be avoided to prevent further discolouration of the skin.

The main incompatibilities of silver with acetylene, ammonia, hydrogen peroxide, ethyleneimine and a number of organic acids should be kept in mind in order to prevent fire and explosion hazards.

The most unstable silver compounds, such as silver acetylide, silver ammonium compounds, silver azide, silver chlorate, silver fulminate and silver picrate, should be kept in cool, well-ventilated places, protected from shock, vibration and contamination by organic or other readily oxidizable materials and away from light.

When working silver nitrate, personal protection should include the wearing of protective clothing to avoid skin contact as well as chemical safety goggles for the protection of the eyes where spillage may

occur. Respirators should be available at workplaces in which engineering control cannot maintain an acceptable environment.

TANTALUM

Occurrence and Uses

Tantalum (Ta) is obtained from the ores tantalite and columbite, which are mixed oxides of iron, manganese, niobium and tantalum. Although they are considered rare elements, the earth's crust contains about 0.003% of niobium and tantalum together, which are similar chemically and usually occur in combination.

The chief use of tantalum is in the production of electric capacitators. Tantalum powder is compacted, sintered and subjected to anodic oxidation. The film of oxide on the surface serves as an insulator, and upon introduction of an electrolyte solution, a high-performance capacitator is obtained. Structurally, tantalum is used where its high melting point, high density and resistance to acids are advantageous. The metal is employed widely in the chemical industry. Tantalum has also been used in rectifiers for railway signals, in surgery for suture wire and for bone repair, in vacuum tubes, furnaces, cutting tools, prosthetic appliances, fibre spinnerets and in laboratory ware.

Tantalum carbide is used as an abrasive. Tantalum oxide finds use in the manufacture of special glass with a high index of refraction for camera lenses.

Hazards

Metallic tantalum powder presents a fire and explosion hazard, although not as serious as that of other metals (zirconium, titanium and so on). The working of tantalum metal presents the hazards of burns, electric shock, and eye and traumatic injuries. Refining processes involve toxic and hazardous chemicals such as hydrogen fluoride, sodium and organic solvents.

Toxicity. The systemic toxicity of tantalum oxide, as well as that of metallic tantalum, is low, which is probably due to its poor solubility. It does, however, represent a skin, eye and respiratory hazard. In alloys with other metals such as cobalt, tungsten and niobium, tantalum has been attributed an aetiological role in hard-metal pneumoconiosis and in skin affections caused by hard-metal dust. Tantalum hydroxide was found to be not highly toxic to chick embryos, and the oxide was non-toxic to rats by intraperitoneal injection. Tantalum chloride, however, had an LD_{50} of 38 mg/kg (as Ta) while the complex salt K_2TaF_7 was about one-fourth as toxic.

Safety and Health Measures

In most operations, general ventilation can maintain the concentration of the dust of tantalum and its compounds below the threshold limit value. Open flames, arcs and sparks should be avoided in areas where tantalum powder is handled. If workers are regularly exposed to dust concentrations approaching the threshold limit level, periodic medical examinations, with emphasis on pulmonary function, are

advisable. For operations involving fluorides of tantalum, as well as hydrogen fluoride, the precautions applicable to these compounds should be observed.

Tantalum bromide ($TaBr_5$), tantalum chloride ($TaCl_5$) and tantalum fluoride (TaF_5) should be kept in tightly stoppered bottles which are plainly labelled and stored in a cool, ventilated place, away from compounds which are affected by acids or acid fumes. Personnel involved should be cautioned about their hazards.

TELLURIUM

Tellurium (Te) is a heavy element with the physical properties and silvery lustre of a metal, yet with the chemical properties of a non-metal such as sulphur or arsenic. Tellurium is known to exist in two allotropic forms—the hexagonal crystalline form (isomorphous with grey selenium) and an amorphous powder. Chemically, it resembles selenium and sulphur. It tarnishes slightly in air, but in the molten state it burns to give the white fumes of tellurium dioxide, which is only sparingly soluble in water.

Occurrence and Uses

The geochemistry of tellurium is imperfectly known; it is probably 50 to 80 times more rare than selenium in the lithosphere. It is, like selenium, a by-product of the copper-refining industry. The anodic slimes contain up to 4% tellurium.

Tellurium is used to improve the machinability of “free-cutting” copper and certain steels. The element is a powerful carbide stabilizer in cast irons, and it is used to increase the depth of chill in castings. Additions of tellurium improve the creep strength of tin. The chief use of tellurium is, however, in the vulcanizing of rubber, since it reduces the time of curing and endows the rubber with increased resistance to heat and abrasion. In much smaller quantities, tellurium is used in pottery glazes and as an additive to selenium in metal rectifiers. Tellurium acts as a catalyst in some chemical processes. It is found in explosives, antioxidants and in infrared-transmitting glasses. Tellurium vapour is used in “daylight lamps”, and tellurium-radioiodinated fatty acid (TPDA) has been used for myocardial scanning.

Hazards

Cases of acute industrial poisoning have occurred as a result of metallic tellurium fumes being absorbed into the lungs.

A study of foundry workers throwing tellurium pellets by hand into molten iron with the emanation of dense white fumes showed that persons exposed to tellurium concentrations of 0.01 to 0.74 mg/m^3 had higher urinary tellurium levels (0.01 to 0.06 mg/l) than workers exposed to concentrations of 0.00 to 0.05 mg/m^3 (urinary concentrations of 0.00 to 0.03 mg/l). The most common sign of exposure was a garlic odour of the breath (84% of cases) and a metallic taste in the mouth (30% of cases). Workers complained of somnolence in the afternoons and loss of appetite, but suppression of sweat did not occur; blood and central nervous system test results were normal. One worker still had a garlic odour in his breath and tellurium in the urine after being away from the work for 51 days.

In laboratory workers who were exposed to fumes of melting tellurium-copper (fifty/fifty) alloy for 10 min, there were no immediate symptoms, but the effects of stinking breath were pronounced. Since tellurium forms a sparingly soluble oxide with no acidic reaction, there is no danger to the skin or to the lungs from tellurium dust or fumes. The element is absorbed through the gastrointestinal tract and lungs, and excreted in the breath, faeces and urine.

Tellurium dioxide (TeO_2), hydrogen telluride (H_2Te) and potassium tellurite (K_2TeO_3) are of industrial health significance. Because tellurium forms its oxide over 450 ºC and the dioxide formed is almost insoluble in water and body fluids, tellurium appears to be less of an industrial hazard than is selenium.

Hydrogen telluride is a gas which decomposes slowly to its elements. It has a similar smell and toxicity to hydrogen selenide, and is 4.5 times heavier than air. There have been reports that hydrogen telluride causes irritation to the respiratory tract.

One unique case is reported in a chemist who was admitted to hospital after accidently inhaling tellurium hexafluoride gas whilst engaged on making the tellurium esters. Streaks of blue-black pigmentation below the skin surface were seen on the webs of his fingers and to a lesser degree on his face and neck. The photographs show very clearly this rare example of true skin absorption by a tellurium ester, which was reduced to black elemental tellurium during its passage through the skin.

Animals exposed to tellurium have developed central nervous system and red blood cell effects.

Safety and Health Measures

Where tellurium is being added to molten iron, lead or copper, or being vaporized onto a surface under vacuum, an exhaust system should be installed with a minimum air speed of 30 m/min to control vapour emission. Tellurium should preferably be used in pellet form for alloying purposes. Routine atmospheric determinations should be made to ensure that the concentration is maintained below the recommended levels. Where no specific permissible concentration is given for hydrogen telluride; however, it is considered advisable to adopt the same level as for hydrogen selenide.

Scrupulous hygiene should be observed in tellurium processes. Workers should wear white coats, hand protection and simple gauze mask respiratory protection if handling the powder. Adequate sanitary facilities must be provided. Processes should not require hand grinding, and well-ventilated mechanical grinding stations should be used.

THALLIUM

Occurrence and Uses

Thallium (Tl) is fairly widely distributed in the earth’s crust in very low concentrations; it is also found as an accompanying substance of other heavy metals in pyrites and blendes, and in the manganese nodules on the ocean floor.

Thallium is used in the manufacture of thallium salts, mercury alloys, low-melting glasses, photoelectric cells, lamps and electronics. It is used in an alloy with mercury in low-range glass thermometers and in some switches. It has also been used in semiconductor research and in myocardial imaging. Thallium is a catalyst in organic synthesis.

Thallium compounds are used in infrared spectrometers, crystals and other optical systems. They are useful for colouring glass. While many thallium salts have been prepared, few are of commercial significance.

Thallium hydroxide (TlOH), or thallous hydroxide, is produced by dissolving thallium oxide in water, or by treating thallium sulphate with barium hydroxide solution. It can be used in the preparation of thallium oxide, thallium sulphate or thallium carbonate.

Thallium sulphate (Tl_2SO_4), or thallous sulphate, is produced by dissolving thallium in hot concentrated sulphuric acid or by neutralizing thallium hydroxide with dilute sulphuric acid, followed by crystallization. Because of its outstanding efficacy in the destruction of vermin, particularly rats and mice, thallium sulphate is one of the most important of the thallium salts. However, some western European countries and the United States have prohibited the use of thallium on the grounds that it is inadvisable that such a toxic substance should be easily obtainable. In other countries, following the development of warfarin resistance in rats, the use of thallium sulphate has increased. Thallium sulphate is also used in semiconductor research, optical systems and in photoelectric cells.

Hazards

Thallium is a skin sensitizer and cumulative poison which is toxic by ingestion, inhalation or skin absorption. Occupational exposure may occur during the extraction of the metal from thallium-bearing ores. Inhalation of thallium has resulted from the handling of flue dusts and the dusts from roasting of pyrites. Exposure may also occur during the manufacture and use of thallium-salt vermin exterminators, the manufacture of thallium-containing lenses and separation of industrial diamonds. The toxic action of thallium and its salts is well documented from reports of cases of acute non-occupational poisoning (not infrequently fatal) and from instances of suicidal and homicidal use.

Occupational thallium poisoning is normally the result of moderate, long-term exposure, and the symptoms are usually far less marked than those observed in acute accidental, suicidal or homicidal intoxication. The course is usually unremarkable and characterized by subjective symptoms such as asthenia, irritability, pains in the legs, some nervous system disorders. Objective symptoms of polyneuritis may not be demonstrable for quite some time. The early neurologic findings include changes in the superficially provoked tendon reflexes and a pronounced weakness and fall-off in the speed of pupil reflexes.

The victim’s occupational history will usually give the first clue to the diagnosis of thallium poisoning since a considerable time may elapse before the rather vague initial symptoms are replaced by the polyneuritis followed by loss of hair. Where massive hair loss occurs, the likelihood of thallium poisoning is readily suspected. However, in occupational poisoning, where exposure is usually

moderate but protracted, the loss of hair may be a late symptom and often noticeable only after the appearance of polyneuritis; in cases of slight poisoning, it may not occur at all.

The two principal criteria for the diagnosis of occupational thallium poisoning are:

1. occupational history which shows that the patient has or may have been exposed to thallium in such work as rodenticide handling, thallium, lead, zinc or cadmium production, or the production or use of various thallium salts

2. neurological symptoms, dominated initially by subjective changes in the form of paraesthesia (both hyperaesthesia and hypoaesthesia) and, subsequently, by reflex changes.

Concentrations of Tl in urine above 500 mg/l have been associated with clinical poisoning. At concentrations of 5 to 500 µg/l the magnitude of risk and severity of adverse effects on humans are uncertain.

Long-term experiments with radioactive thallium have shown marked excretion of thallium in both urine and faeces. On autopsy, the highest thallium concentrations are found in the kidneys, but moderate concentrations may also be present in the liver, other internal organs, muscles and bones. It is striking that, although the principal signs and symptoms of thallium poisoning originate from the central nervous system, only very low concentrations of thallium are retained there. This may be due to extreme sensitivity to even very small amounts of the thallium acting on the enzymes, the transmission substances, or directly on the brain cells.

Safety and Health Measures

The most effective measure against the dangers associated with the manufacture and use of this group of extremely toxic substances is the substitution of a less harmful material. This measure should be adopted wherever possible. When thallium or its compounds must be used, the strictest safety precautions should be taken to ensure that the concentration in the workplace air is kept below permissible limits and that skin contact is prevented. Continuous inhalation of such concentrations of thallium during normal working days of 8 hours may cause the urine level to exceed the above permissible levels.

Persons involved in work with thallium and its compounds should wear personal protective equipment, and respiratory protective equipment is essential where there is the possibility of dangerous inhalation of airborne dust. A complete set of working clothes is essential; these clothes should be washed regularly and kept in accommodation separate from that employed for ordinary clothes. Washing and shower facilities should be provided and scrupulous personal hygiene encouraged. Workrooms must be kept scrupulously clean, and eating, drinking or smoking at the workplace prohibited.

TIN

Tin has been used through the ages up to modern industrial times because it is pliable and easily shaped at normal temperatures, and it mixes readily with other metals to form alloys. One of its outstanding characteristics is its resistance to acids and atmospheric influences.

Occurrence and Uses

Although deposits of tin are widely distributed throughout the world, up to the eighteenth century the world's supply of tin was mainly from England, Saxony and Bohemia. Today, except for some deposits in Nigeria, China, the Congo and Australia, the principal sources are found in Southeast Asia and Bolivia.

Of minerals containing tin, cassiterite (SnO_2) or tinstone is of the greatest commercial importance. It is present in veins closely connected with granite or acid eruptive rocks, but five-sixths of the world's total production is derived from secondary alluvial deposits resulting from the disintegration of the primary deposits. In Bolivia, sulphide ores, such as stannite (Cu_2FeSnS_2) and tealite ($PbZnSnS_2$) are of commercial significance.

Metallic tin is used for Babbitt type metals and for collapsible tubes in the pharmaceutical and cosmetic industries. Because of its resistance to corrosion, tin is used as a protective coating for other metals. Tinplate is sheet iron or steel which has been thickly coated with tin by dipping in a molten bath of that metal. It is used mainly for making household utensils and for utensils in food and beverage canning industries. It is often used for decorating purposes. Terneplate is sheet iron or steel coated with a lead-tin alloy containing 85% lead and 15% tin. It is used mainly for making roofing tile. Speculum is a tin-copper alloy containing 33 to 50% tin, that can be polished to a high degree of reflection. It is used as a coating applied by electrolytic deposition to impart brightness to silverware and similar articles, and for making telescope mirrors. A molten tin bath is also used in the production of window glass.

An important property of tin is its ability to form alloys with other metals, and it has a number of uses in this field. A tin-lead alloy known as soft solder is widely used for joining other metals and alloys in the plumbing, automobile, electrical and other industries, and as a filler in the finishing of car bodies. Tin is a constituent of a large number of non-ferrous alloys, including phosphor bronze, light brass, gun-metal, high-tensile brass, manganese bronze, die-casting alloys, bearing metals, type metal and pewter. The tin-niobium alloy is superconductive, and it is used in the manufacture of powerful electromagnets.

Stannic chloride ($SnCl_4$), or tin chloride, is prepared by heating powdered tin with mercuric chloride or by passing a stream of chlorine over molten tin. It is used as a dehydrating agent in organic syntheses, a stabilizer for plastics, and as a chemical intermediate for other tin compounds. Stannic chloride is found in colours and perfumes in the soap industry. It is also employed in ceramics to produce abrasion-resistant or light-reflecting coatings. It is used for the bleaching of sugar and for the surface treatment of glass and other non-conductive materials. The pentahydrate of this salt is used as a mordant. It is also used in treating silk for the purpose of giving weight to the fabric.

Stannous chloride dihydrate ($SnCl_2 \cdot 2H_2O$), or tin salt, is produced by dissolving metallic tin in hydrochloric acid and evaporating until crystallization begins. It is used in dye works as a mordant. It also serves as a reducing agent in the manufacture of glass, ceramics and inks.

The use of organotin (alkyl and aryl) compounds has greatly increased in recent years. Disubstituted compounds and, to a lesser degree, monosubstituted compounds, are used as stabilizers and catalysts in the plastics industry. Trisubstituted compounds are used as biocides, and tetrasubstitutes are intermediates in the production of other derivatives. Butyltin trichloride, or trichlorobutyltin; dibutyltin dichloride, or dichlorodibutyltin; trimethyltin; triethyltin chloride; triphenyltin chloride, or TPTC; tetraisobutyltin, or tetraisobutylstannane are among the most important.

Hazards

In the absence of precautions, mechanical injury can be caused by the heavy, powerful plant and machinery used in the dredging and washing operations. Serious burn hazards are present in the smelting processes when molten metal and hot slags are manipulated.

At the final stage of upgrading of cassiterite concentrate and during the roasting of sulphide ore, sulphur dioxide is evolved. Sulphur dioxide and stannous sulphide constitute a hazard when the rough molten tin is separated from the rest of the charge during refining. This work is done in a very hot environment, and heat exhaustion could arise. The noise on a dredger caused by the discharge from the dredging buckets to the primary washing plant may cause damage to the hearing of the workers.

Several studies report the hazards associated with exposure to radon, radon decay products and silica in tin mines. While most of the operations associated with the extraction and treatment of tin ore are wet processes, tin dust and oxide fumes may escape during bagging of concentrate, in ore rooms and during smelting operations (mixing-plant and furnace tapping), as well as during the periodic cleaning of bag filters used to remove particulate matter from smelter furnace flue gas before release to the atmosphere. The inhalation of tin oxide dust without silica leads to a benign nodular pneumoconiosis without pulmonary disability. The radiological picture is similar to baritosis. This benign pneumoconiosis has been called stannosis.

Tin powder is a moderate irritant to the eyes and airways; it is combustible and reacts violently with oxidants, strong acids, powdered sulphur and some extinguishing agents such as bicarbonate powder and carbon dioxide.

Tin ingested in small (mg) quantities is non-toxic (hence, the widespread use of tinplate in the food canning industry). The results of animal experiments indicate that the lethal dose by intravenous injection is about 100 mg/kg body weight, and that the ingestion of considerable quantities of powdered tin may cause vomiting but not permanent injury. It appears that humans can tolerate a daily intake of 800 to 1,000 mg without ill effect. The absorption of metallic tin or its inorganic salts from the alimentary tract seems to be small.

A number of tin alloys are injurious to health (particularly at high temperatures) because of the harmful characteristics of the metals with which may be alloyed (e.g., lead, zinc, manganese).

Organotin compounds are, in general, strong irritants, and acute conjunctivitis has been observed as a result of eye splashes, even when followed by immediate lavage; corneal opacities have also been reported. Prolonged contact of the skin with clothes moistened with vapour, or direct spillage on the

skin, have been responsible for acute local burns, subacute diffuse erythematoid dermatitis with pruritus and some pustular eruption in the hair-covered areas. The irritation of the airways and pulmonary tissue can lead to lung oedema; the gastrointestinal tract can also be involved, and inflammatory reactions of the bile duct have been observed, mainly with the dialkyl compounds. Organotin compounds can injure liver and kidneys; they can depress the immune response and have haemolytic activity. In experimental animals they have been in some instances held responsible for reduction in fertility.

Tri- and tetralkyl compounds, in particular triethyltin chloride, cause encephalopathy and brain oedema, with clinical effects of depression, convulsions, flaccid paralysis and urinary retention, as seen in therapeutic use following oral administration.

Safety and Health Measures

Wherever possible, safer substitutes should be used in the place of alkyl tin compounds. When it is necessary to make and use them, the widest possible use should be made of enclosed systems and exhaust ventilation. Engineering control should ensure that exposure limits are not exceeded. Personal protective equipment should be worn, and in appropriate circumstances respiratory protection should be used. Emergency showers should be installed at workplaces in order to allow workers to wash immediately after splashes.

Medical surveillance should focus on eyes, skin and chest x rays in the exposure to inorganic tin compounds, and on eyes, skin, central nervous system, liver and kidney function, and blood in the exposure to organic tin compounds. Mercaprol has been reported as useful in the treatment of dialkyltin intoxications. Steroids have been suggested for the treatment of triethyltin poisoning; however only surgical decompression seems to be of value in encephalopathy and brain oedema provoked by tri- and tetraalkyl tin compounds.

Taking into consideration the fact that most tin mines are located in developing countries, attention should also be paid to climatic and other factors influencing the health, well-being and productive capacity of the workers. Where mines are geographically isolated, good housing should be provided for all personnel. Nutritional standards should be upgraded by health education, and workers should be provided with adequate food supplies and good medical care.

TITANIUM

Occurrence and Uses

Titanium (Ti) is contained in many minerals, but only a few of them are of industrial significance. These include ilmenite ($FeTiO_3$), which contains 52.65% Ti and 47.4% FeO; rutile (TiO_2), with admixtures of ferrous oxide; perovskite ($CaTiO_3$), which contains 58.7% TiO_2 and 41.3% CaO; and sphene, or titanite, ($CaOTiO_2{\cdot}SiO_2$), which contains 38.8% TiO_2. Some heterogeneous minerals, such as loparite, pyrochlor, and tailings from bauxite and copper ore processing may also be sources of titanium.

Titanium is used as a pure metal, in alloys, and in the form of various compounds. The bulk of titanium is needed in the iron and steel industry, in shipbuilding, for aircraft and rocket construction, and for the fabrication of chemical plants. Titanium is used as a protective surface on mixers in the pulp and paper industry. It is also found in surgical appliances. Titanium has been employed for the manufacture of electrodes, lamp filaments, paints, dyes and welding rods. Titanium powder is used in pyrotechnics and in vacuum engineering. Titanium is also used in dentistry and in surgery for implants or prostheses.

Titanium carbide and titanium nitride are used in powder metallurgy. Barium titanate is used for making heavy-duty capacitors. Titanium dioxide is utilized as a white pigment in paints, floor coverings, upholstery, electronics, adhesives, roofing, plastics and in cosmetics. It is also useful as a component of porcelain enamels and glazes, as a shrinking agent for glass fibres, and as a delustering agent for synthetic fibre. Titanium tetrachloride acts as an intermediate in the production of titanium metal and titanium pigments, and as a catalyst in the chemical industry.

Hazards

The formation of titanium dioxide (TiO_2) and concentrate dust, pitch briquette dust arising from crushing, mixing and charging of bulk raw materials, and radiant heat from coking furnaces are hazards in titanium production. There may be chlorine, titanium tetrachloride ($TiCl_4$) vapours and their pyrolysis products in the air of the chlorination and rectification plants, arising from leaking or corroded equipment. Magnesium oxide may be present in the air of the reduction area. Titanium dust becomes airborne when titanium sponge is knocked out, crushed, separated and bagged. Exposure to heat and infrared radiation occurs in the arc furnace area (up to 3 to 5 cal/cm^2 per min).

Maintenance and repair of the chlorination and rectification installations, which includes disassembly and cleaning of the equipment and pipework, create particularly adverse conditions of work: high concentrations of $TiCl_4$ vapours and hydrolysis products (HCl, $Ti(OH)_4$), which are highly toxic and irritant. Workers in these plants often suffer from upper-airway disease and acute or chronic bronchitis. Liquid $TiCl_4$ splashed on the skin causes irritation and burns. Even very short contact of the conjunctiva with $TiCl_4$ leads to suppurative conjunctivitis and keratitis, which may result in corneal opacities. Animal experiments have shown that dust of metallic titanium, titanium concentrates, titanium dioxide and titanium carbide is slightly toxic. While titanium dioxide has not been found to be fibrogenic in animals, it seems to increase the fibrogenicity of quartz when given as combined exposure. Long-term exposure to titanium-containing dust may result in mild forms of chronic lung disease (fibrosis). There is radiological evidence that workers who have handled TiO_2 for long periods develop lung changes resembling those observed in mild forms of silicosis. In one worker who had worked in contact with titanium dioxide for several years and died from brain cancer, the lungs displayed accumulations of TiO_2 and changes analogous to anthracosis. Medical examinations of powder metallurgy workers in various countries have disclosed cases of chronic pneumonitis due to

mixed dust including titanium carbide. The degree of this disease varied according to conditions of work, length of dust exposure and individual factors.

Workers who have been chronically exposed to titanium and titanium dioxide dust show a high incidence of chronic bronchitis (endobronchitis and peribronchitis). The early stages of the disease are characterized by impaired pulmonary respiration and ventilatory capacity, and by reduced blood alkalinity. Electrocardiographic tracings of these titanium workers revealed cardiac changes characteristic of pulmonary disease with hypertrophy of the right auricle. A considerable number of these cases presented myocardial hypoxia of various degrees, inhibited atrioventricular and intraventricular conductivity, and bradycardia.

Airborne metallic titanium dust is explosive.

Other hazards in titanium production are carbon monoxide exposures at the coking and arc furnaces, and burns.

Safety and Health Measures

Control dust during ore crushing by moistening the material to be processed (up to 6 to 8% moisture content), and by adopting a continuous process, which enables the equipment to be enclosed with exhaust devices at all points where dust may form; the dust-laden air exhausted should be filtered and the dust collected should be recycled. Dust exhaust systems must be provided at the knock-out stations; crushers, separators and baggers in the titanium sponge plant. Knocking out with pneumatic chipping hammers should be replaced by machining out on special milling or turning machines.

TUNGSTEN

Occurrence and Uses

Tungsten (W) never occurs free in nature and is found only in a few minerals as tungstate of calcium, iron or manganese. Of the known tungsten-bearing minerals, scheelite ($CaWO_4$), wolframite ($(Fe,Mn)WO_4$), hubnerite (MnWO) and ferberite ($FeWO_4$) are commercially important. Total world reserves of tungsten trioxide (WO_3) are estimated to be about 175,000,000 t. These tungsten minerals are mostly mined from underground workings, but open-cut operations and more primitive methods are also applied. The tungsten content of the ore mined is usually 0.5 to 2.0%. The more common impurities are gangue minerals such as quartz and calcite, and metallic minerals of copper, bismuth, tin and molybdenum.

Tungsten is a component in hard metals. It is used to increase the hardness, toughness, elasticity and tensile strength of steel. It is used in the production of tungsten steels for automobiles and high-speed cutting tools. Tungsten is also used in lamps, vacuum tubes, electric contacts, x-ray tubes and fluorescent light tubes. It serves as a flame retardant in the textile industry.

Tungsten carbide (WC) has replaced diamond in large drawing dies and rock drills because of its extreme hardness. Tungsten compounds are also used in lasers, dyes, inks and ceramic frits. Some

tungsten alloys are used in the nuclear and space industries for nozzles of rocket motors and for protecting shields for spacecraft.

Hazards

Little is known of the toxicity of tungsten. The LD_{50} of sodium tungstate for 66-day-old rats was between 223 and 255 mg/kg and showed significant postprandial and age effect. Of three tungsten compounds, sodium tungstate is most toxic, tungstic oxide is intermediate, and ammonium paratungstate is least toxic. The feeding of 2.5 and 10% of diet as tungsten metal over a period of 70 days has been shown to be without marked effect upon the growth of male rats, as measured in terms of gain in weight, though it caused a 15% reduction in weight gain for female rats from that of control.

Industrial exposure is related chiefly to substances associated with the production and uses of tungsten, its alloys and compounds, rather than tungsten itself. In the mining and milling processes, the main hazards seem to be exposure to quartz-containing dust, noise, hydrogen sulphide, sulphur dioxide and chemicals such as sodium cyanide and sodium hydroxide. The exposure may be associated with other metals in the ore, such as nickel.

Hard metal is the mixture of tungsten carbide and cobalt, to which small amounts of other metals may be added. In the tool-cutting industry workers may be exposed to dust of tungsten carbide, cobalt fumes and dust, and carbides of nickel, titanium and tantalum. Following occupational exposure to tungsten carbide dust by inhalation, cases of pneumoconiosis or pulmonary fibrosis have been reported, but it is generally agreed that this “hard-metal disease” is more likely to be caused by the cobalt with which tungsten carbide is fused. Where machining and grinding of tungsten carbide tools is performed, the hard-metal workers may be at risk for the development of interstitial obstructive lung disease, a serious hazard associated with elevated air concentrations of cobalt. The effects of hard metals on the lungs are discussed elsewhere in this Encyclopaedia.

Tungsten carbonyl is a moderate fire hazard when exposed to flame. When heated to decomposition, it emits carbon monoxide. The incidence of accidents and diseases in tungsten mines and mills is not well documented. However, from the scarce data available it can be said that it is less than that of coal mines.

VANADIUM

Occurrence and Uses

The most important vanadium (V) ores are patronite (vanadium sulphide), found in Peru, and descloizite (lead-zinc vanadate), found in South Africa. Other ores, such as vanadinite, roscoelite and carnotite, contain vanadium in sufficient quantities for economic extraction. Crude petroleum may contain small amounts of vanadium, and flue-gas deposits from oil-fired furnaces may contain over 50% vanadium pentoxide. Slags from ferrovanadium are another source of the metal. One of the most important sources of human exposure to vanadium is vanadium oxides released when burning fuel oils.

Normally, small amounts of vanadium are found in the human body, particularly in adipose tissue and in the blood.

The larger amount of the vanadium produced is used in ferrovanadium, the most important direct use of which is in high-speed steel and tool steelmaking. Addition of 0.05 to 5% of vanadium removes occluded oxygen and nitrogen from the steel, enhances the tensile strength and improves the modulus of elasticity and the rust resistance of the final alloy. In the past vanadium compounds have been used as therapeutic agents in medicine. The vanadium-gallium alloy has shown interesting properties for production of high magnetic fields.

Certain vanadium compounds have a limited use in industry. Vanadium sulphate ($VSO_4 \cdot 7H_2O$) and vanadium tetrachloride (VCl_4) are used as mordants in the dyeing industry. Vanadium silicates are used as catalysts. Vanadium dioxide (VO_2) and vanadium trioxide (V_2O_3) are employed in metallurgy. However, the most significant compounds in terms of industrial health hazards are vanadium pentoxide (V_2O_5) and ammonium metavanadate (NH_4VO_3).

Vanadium pentoxide is obtained from patronite. It has for a long time been an important industrial catalyst used in a number of oxidation processes such as those involved in the manufacture of sulphuric acid, phthalic acid, maleic acid and so on. It serves as a photographic developer and as a dyeing agent in the textile industry. Vanadium pentoxide is also used in ceramic colouring materials.

Ammonium metavanadate is employed as a catalyst in the same way as vanadium pentoxide. It is a reagent in analytical chemistry and a developer in the photography industry. Ammonium metavanadate is also used in dyeing and printing in the textile industry.

Hazards

Experience has shown that vanadium oxides and, in particular, the pentoxide and its derivative ammonium metavanadate cause harmful effects in humans. Exposure to vanadium pentoxide is possible at the following points in industry: when vanadium pentoxide is used in particulate form in the production of metallic vanadium; during the repair of installations where vanadium pentoxide is used as a catalyst; and during the cleaning of oil-fired furnace flues in power stations, ships and so on. The presence of vanadium compounds in petroleum products is of particular significance and, because of the possibility of air pollution in the environment of oil-fired power stations, it receives attention from public health authorities as well as from those concerned with industrial health.

The inhalation of vanadium compounds may produce severe toxic effects. The severity of the effects depends on the atmospheric concentration of the vanadium compounds and the duration of exposure. Health impairment may occur after even brief exposure (e.g., 1 hour), and the initial symptoms are profuse lacrimation, burning sensation in the conjunctivae, serous or haemorrhageous rhinitis, sore throat, cough, bronchitis, expectoration and chest pain.

Severe exposure may result in pneumonia with fatal outcome; however, following one-time exposure, complete recovery usually occurs within 1 to 2 weeks; prolonged exposure may produce chronic

bronchitis with or without emphysema. The tongue may present a greenish discolouration and also the cigarette ends of vanadium workers may show a greenish colour, resulting from chemical interactions.

Local effects in experimental animals are mainly observed in the respiratory tract. Systemic effects have been observed in the liver, kidney, nervous system, cardiovascular system and blood-forming organs. Metabolic effects include interference with biosynthesis of cystine and cholestrol, depression and stimulation of phospholipid synthesis. Higher concentrations have produced inhibition of serotonin oxidation. In addition, vanadate has been shown to inhibit several enzyme systems. In humans, systemic effects of vanadium exposure are less well documented, but reduction of serum cholestrol has been demonstrated. In the work environment, vanadium and its compounds are taken up in the human body by inhalation, mainly during production and boiler cleaning operations. Absorption of vanadium from the gastrointestinal tract is poor, not exceeding 1 to 2%; ingested vanadium compounds are largely eliminated with faeces.

A study was conducted to evaluate the level of bronchial responsiveness among workers recently exposed to vanadium pentoxide during periodic removal of ashes and clinker from boilers of an oil-fired power station. This study suggests that exposure to vanadium increases bronchial responsiveness even without the appearance of bronchial symptoms.

Safety and Health Measures

It is important to prevent the inhalation of airborne particulate vanadium pentoxide. For use as a catalyst, vanadium pentoxide can be produced in an agglomerated or pelleted form which is dust free; however, vibration in the plant may, in time, reduce a certain proportion to dust. In the processes associated with the manufacture of metallic vanadium, and in the sieving of used catalyst during maintenance operations, the escape of dust should be prevented by the enclosure of the process and by the provision of exhaust ventilation. In boiler cleaning in power stations and on ships, maintenance workers may have to enter the boilers to remove soot and to make repairs. These workers should wear adequate respiratory protective equipment with full face mask and eye protection. Wherever possible, on-load cleaning should be improved to reduce the need for workers to enter furnaces; where off-load cleaning proves essential, methods such as water lancing, which do not necessitate physical entry, should be tried.

ZINC

Occurrence and Uses

Zinc (Zn) is widely distributed in nature in quantities which amount to approximately 0.02% of the earth’s crust. It is found in nature as the sulphide (sphalerite), carbonate, oxide or silicate (calamine) in combination with many minerals. Sphalerite, the principal zinc mineral and the source of at least 90% of metallic zinc, contains iron and cadmium as impurities. It is almost always accompanied by galena, the sulphide of lead, and occasionally is found in association with ores containing copper or other base metal sulphides.

On exposure to air, zinc becomes covered with a tenacious film of oxide which protects the metal from further oxidation. This resistance to atmospheric corrosion forms the basis for one of the most common uses of the metal, the protection of steelwork by galvanizing. Zinc's ability to protect ferrous metals against corrosion is reinforced by electrolytic action. It acts as an anode with respect to iron and other structural metals, except aluminium and magnesium, and is thus preferentially attacked by corrosive agents. This property is used in many other important applications of zinc—for example, in the use of zinc plates as anodes for cathodic protection of ships' hulls, underground tanks and so on. Zinc metal is die cast for components in the automobile industry, electrical equipment industry, and in the light machine tool, hardware, toys and fancy goods industries. It is rolled into sheets in rolling mills for the manufacture of roofing, weather stripping, cases for dry batteries, printing plates and so on. Zinc is also alloyed with copper, nickel, aluminium and magnesium. When it is alloyed with copper, it forms the important groups of alloys known as the brasses.

Zinc oxide (ZnO), or zinc white (flowers of zinc) is produced by the oxidation of vaporized pure zinc or by the roasting of zinc oxide ore. It is used as a pigment in paints, lacquers and varnishes, as well as a filler for plastics and rubber. Zinc oxide is found in cosmetics, quick-setting cements, and in pharmaceuticals. It is useful in the manufacture of glass, automobile tyres, matches, white glue and printing inks. Zinc oxide is also used as a semiconductor in the electronics industry.

Zinc chromate ($ZnCrO_4$), or zinc yellow, is produced by the action of chromic acid on slurries of zinc oxide, or on zinc hydroxide. It is used in pigments, paints, varnishes and lacquers, and in the manufacture of linoleum. Zinc chromate acts as a corrosion inhibitor for metals and epoxy laminates.

Zinc cyanide ($Zn(CN)_2$) is produced by precipitation of a solution of zinc sulphate or chloride with potassium cyanide. It is used for metal plating and for gold extraction. Zinc cyanide acts as a chemical reagent and as a pesticide. Zinc sulphate ($ZnSO_4 \cdot 7H_2O$), or white vitriol, is produced by roasting zinc blende or by the action of sulphuric acid on zinc or zinc oxide. It is used as an astringent, a preservative for hides and wood, a bleach for paper, a pesticide adjuvant and a fungicide. Zinc sulphate also serves as a fireproofing agent and as a depressant in froth flotation. It is used in water treatment and in textile dyeing and printing. Zinc sulphide is used as a pigment for paints, oilcloths, linoleum, leather, inks, lacquers, and cosmetics. Zinc phosphide (Zn_3P_2) is produced by passing phosphine through a solution of zinc sulphate. It is used mainly as a rodenticide.

Zinc chloride ($ZnCl_2$), or butter of zinc, has numerous uses in the textile industry, including dyeing, printing, sizing and weighting fabrics. It is a component of cement for metals, dentifrices, and soldering fluxes. It is used alone or with phenol and other antiseptics for preserving railway ties. Zinc chloride is useful for glass etching and for the manufacture of asphalt. It is a vulcanizing agent for rubber, a flame retardant for wood, and a corrosion inhibitor in water treatment.

Hazards

Zinc is an essential nutrient. It is a constituent of metalloenzymes, which play an important role in nucleic acid metabolism and protein synthesis. Zinc is not stored in the body, and a minimum daily intake of zinc is recommended by nutritional experts. Absorption of zinc takes place more readily from animal protein sources than from plant products. The phytate content of plants binds zinc, rendering it unavailable for absorption. Zinc deficiency states have been reported from countries where cereals are the major source of protein consumed by the population. Some of the recognized clinical manifestations of chronic zinc deficiency in humans are growth retardation, hypogonadism in males, skin changes, poor appetite, mental lethargy and delayed wound healing.

In general, zinc salts are astringent, hygroscopic, corrosive and antiseptic. Their precipitating action on proteins forms the basis of their astringent and antiseptic effects, and they are absorbed relatively easily through the skin. The taste threshold for zinc salts is approximately 15 ppm; water containing 30 ppm of soluble zinc salts has a milky appearance, and a metallic taste when the concentration reaches 40 ppm. Zinc salts are irritating to the gastrointestinal tract, and the emetic concentrations for zinc salts in water range from 675 to 2,280 ppm.

The solubility of zinc in weakly acidic solutions, in the presence of iron, has led to accidental ingestion of large quantities of zinc salts when acid foods such as fruit drinks were prepared in worn galvanized iron vessels. Fever, nausea, vomiting, stomach cramps and diarrhoea occurred in 20 minutes to 10 hours following ingestion.

A number of zinc salts may enter the body by inhalation, through the skin or by ingestion and produce intoxication. Zinc chloride has been found to cause skin ulcers. A number of zinc compounds present fire and explosion hazards. The electrolytic manufacturing of zinc can produce mists containing sulphuric acid and zinc sulphate that can irritate the respiratory or digestive systems and lead to dental erosion. Metallurgic processes involving zinc can lead to arsenic, cadmium, manganese, lead and possibly chromium and silver exposures, with their associated hazards. Since arsenic is frequently present in zinc, it can be a source of exposure to highly toxic arsine gas whenever zinc is dissolved in acids or alkalis.

In zinc metallurgy and manufacturing, welding and cutting of galvanized or zinc-coated metal, or melting and casting of brass or bronze, the most frequently encountered hazard from zinc and its compounds is exposure to zinc oxide fumes, which cause metal-fume fever. Symptoms of metal-fume fever include shivering attacks, irregular fever, profuse sweating, nausea, thirst, headache, pains in the limbs and a feeling of exhaustion. Attacks are of short duration (most cases are on the way to complete recovery within 24 hours of the onset of symptoms), and tolerance seems to be acquired. A significant increase in free erythrocyte protoporphyrin has been reported in zinc oxide packing operations.

Zinc chloride fumes are irritating to the eyes and mucous membranes. In an accident involving smoke generators, 70 exposed persons experienced varying degrees of irritation of the eyes, nose, throat and lungs. Of the 10 fatalities, some died within a few hours with pulmonary oedema, and others died later of bronchopneumonia. On another occasion, two firemen were exposed to zinc chloride fumes from a

smoke generator during a firefighting demonstration, one briefly, the other for several minutes. The former recovered rapidly while the latter died after 18 days, due to respiratory failure. There was a rapid rise of temperature and marked upper respiratory tract inflammation soon after exposure. Diffuse pulmonary infiltrations were seen on the chest radiograph, and autopsy revealed active fibroblastic proliferation and cor pulmonale.

In an experiment primarily designed to evaluate carcinogenesis, groups of 24 mice received 1,250 to 5,000 ppm of zinc sulphate in drinking water for one year. Apart from severe anaemia in animals receiving 5,000 ppm, there were no adverse effects from zinc. Tumour incidence was not significantly different from that seen in the controls.

Zinc phosphide, which is used as a rodenticide, is toxic to humans whether swallowed, inhaled or injected, and, together with zinc chloride, is the most dangerous of the zinc salts; these two substances have been responsible for the only deaths definitely due to zinc poisoning.

Skin effects. Zinc chromate in primer paints used by car-body builders, tinsmiths and steel cupboard makers has been reported to cause nasal ulceration and dermatitis in exposed workers. Zinc chloride has a caustic action, which may result in ulceration of the fingers, hands and forearms of those who handle timber impregnated with it or use it as a flux in soldering. It has been reported that zinc oxide dust may block the ducts of the sebaceous glands and give rise to a papular, pustular eczema in humans packaging this compound.

Safety and Health Measures

Fire and explosion. Finely divided zinc powder, and other zinc compounds, can be fire and explosion hazards if stored in damp places, sources of spontaneous combustion. Residues from reduction reactions may ignite combustible materials. Zinc ammonium nitrate, zinc bromate, zinc chlorate, zinc ethyl, zinc nitrate, zinc permanganate and zinc picrate are all dangerous fire and explosion hazards. In addition, zinc ethyl will ignite spontaneously in contact with air. It should, therefore, be stored in a cool, dry, well-ventilated place away from acute fire risks, open flames and powerful oxidizing agents.

In all cases where zinc is heated to the point where fumes are produced, it is most important to ensure that adequate ventilation is provided. Individual protection is best ensured by education of the worker concerning metal-fume fever and the provision of local exhaust ventilation, or, in some situations, by wearing of a supplied-air hood or mask.

Workers who are none the less exposed to zinc chloride fumes should wear personal protective equipment including protective clothing, chemical eye and face protection and appropriate respiratory protective equipment. Exposure to zinc chloride fumes should be treated by copious irrigation of the exposed areas.

ZIRCONIUM AND HAFNIUM

Occurrence and Uses

It has been estimated that zirconium (Zr) constitutes about 0.017% of the lithosphere. Because of its very high chemical activity at temperatures only slightly above normal atmospheric temperature, the element occurs only in combined states. The most common ores are zircon (ZrO_2) and baddeleyite ($ZrSiO_4$). Zirconium is found in all animal tissues.

Hafnium (Hf) is found associated with zirconium in all its terrestrial occurrences. The amount of hafnium varies but averages about 2% of the total zirconium plus hafnium. In only one ore, low in both elements, has hafnium been found in greater quantity than zirconium. Spectrographic evidence indicates that the distribution is also about 2% hafnium in the total zirconium-plus-hafnium in the universe. These two elements are more closely identical in their chemical properties than are any other pair in the periodic table. The similarity is so great that no qualitative differences have yet been found which would permit their separation. For this reason, it can be assumed that most of the zirconium which has been used, and on the basis of which physiological effects have been reported, has contained 0.5 to 2% hafnium.

Zircon has been valued since the earliest times as a gem stone, since it occurs quite commonly in large single crystals; however, most of the commercially useful deposits of zirconium ore are in beach sands or other places where the relatively heavy and chemically inert zirconium minerals have been deposited while the lighter portions of the rocks in which they occurred have been disintegrated and washed away by the action of water. Substantial deposits of such beach sands are known in India, Malaya, Australia and the United States. Baddeleyite in commercially useful deposits was first observed in Brazil, and has since been found in a number of other locations including Sweden, India and Italy. Some zirconium ores have also been mined commercially in Madagascar, Nigeria, Senegal and South Africa.

Zircon is used as a foundry sand, an abrasive, and as a component of zircon and zirconia refractory compositions for laboratory crucibles. It is found in ceramic compositions where it acts as an opacifier in glazes and enamels. Zircon and zirconia bricks are used as linings for glass furnaces. Zirconia forms are also used as dies for extrusion of both ferrous and non-ferrous metals and as spout linings for pouring metals, particularly for continuous casting.

More than 90% of zirconium metal is now used in nuclear power generation because zirconium has a low absorption cross-section for neutrons and a high resistance to corrosion inside atomic reactors, provided it is free of hafnium. Zirconium is also used in the manufacture of cast iron, steel and surgical appliances. It is employed in arc lamps, pyrotechnics, in special welding fluxes, and as a pigment in plastics.

Powdered zirconium metal is used as a “getter” in thermionic tubes to absorb the last traces of gas after pumping and out-gassing of the tube elements. In the form of fine ribbon or wool, the metal is also used as the filter in photographic flash-bulbs. The massive metal is used either pure or in alloy form for the lining of reaction vessels. It is also used as a lining for pumps and piping systems for chemical

processes. An excellent super-conducting alloy of zirconium and columbium has been used in a magnet with a field of 6.7 T.

Zirconium carbide and zirconium diboride are both hard, refractory, metallic compounds which have been used in cutting tools for metals. The diboride has also been used as a thermocouple jacket in open-hearth furnaces, providing very long-lived thermocouples. Zirconium tetrachloride is used in organic synthesis and in water repellents for textiles. It is also useful as a tanning agent.

Hafnium metal has been used as a cladding on tantalum for rocket engine parts which must operate in very high-temperature, erosive conditions. Because of its high thermal-neutron cross-section, it is also used as a control rod material for nuclear reactors. In addition, hafnium is used in the manufacture of electrodes and light-bulb filaments.

Hazards

It is inaccurate to state that zirconium compounds are physiologically inert, but the tolerance of most organisms to zirconium appears to be great in comparison to the tolerance for most heavy metals. Zirconium salts have been used in the treatment of plutonium poisoning to displace the plutonium (and yttrium) from its deposition in the skeleton and to prevent the deposition when treatment was started early. In the course of this study, it was determined that the diet of rats could contain as much as 20% of zirconia for comparatively long periods without harmful effects, and that the intravenous LD_{50} of sodium zirconium citrate for rats is about 171 mg/kg body weight. Other investigators have found an intraperitoneal LD_{50} of 0.67 g/kg for zirconium lactate and 0.42 g/kg for barium zirconate in rats and 51 mg/kg of sodium zirconium lactate in mice.

Zirconium compounds have been recommended and used for the topical treatment of Rhus (poison ivy) dermatitis and for body deodorants. Some compounds which have been used are carbonated hydrous zirconia, hydrous zirconia and sodium zirconium lactate. There have been a number of reports of the production of persistent granulomatous conditions of the skin as the result of these applications.

Of more direct interest in connection with occupational exposures is the effect of inhalation of zirconium compounds, and this has been less extensively investigated than the other routes of administration. There have, however, been several experiments and at least one report of human exposure. In this instance, a chemical engineer with seven years' exposure in a zirconium and hafnium processing plant was found to have a granulomatous lung condition. Since examination of all the other employees revealed no comparable lesions, it was concluded that the condition was most probably to be attributed to a relatively heavy beryllium exposure prior to zirconium exposure.

Exposure of experimental animals to zirconium compounds showed that zirconium lactate and barium zirconate both produced severe, persistent, chronic interstitial pneumonitis at atmospheric zirconium concentrations of about 5 mg/m^3. Much higher atmospheric sodium zirconium lactate concentrations of 0.049 mg/cm^3 for shorter exposures have been found to produce peribronchial abscesses, peribronchiolar granulomas and lobular pneumonia. Although documentation of zirconium

pneumoconiosis in humans has been lacking, authors of one study conclude that zirconium should be considered a likely cause of pneumoconiosis, and recommend taking appropriate precautions in the workplace.

The small number of investigations on the toxicity of hafnium compounds has indicated an acute toxicity slightly higher than that of zirconium salts. Hafnium and its compounds cause liver damage. Hafnyl chloride at 10 mg/kg produced cardiovascular collapse and respiratory arrest in a cat in the same manner as soluble zirconium salts; the intraperitoneal LD_{50} of 112 mg/kg for hafnium is not much smaller than that for zirconium.

Safety and Health Measures

Fire and explosion. Zirconium metal in the form of a fine powder burns in air, nitrogen or carbon dioxide. The powders are explosive in air in the range of 45 to 300 mg/l, and are self-igniting if disturbed, probably because of static electricity generated by separation of the grains.

The powdered metals should be transported and handled in the wet state; water is usually used for wetting. When the powder is dried prior to use, the quantities employed should be kept as small as possible and operations should be carried out in separate cubicles to prevent propagation in the event of an explosion. All sources of ignition, including static electric charges, should be eliminated from areas in which the powder is to be handled.

All surfaces in the area should be impervious and seamless so that they can be washed down with water and kept completely free from dust. Any spilled powder should be cleaned up immediately with water so that it has no chance to dry in place. Used papers and cloths which have become contaminated with the powders should be kept wet in covered containers until they are removed to be burned, which should be done at least daily. The dried powders should be disturbed and handled as little as possible, and then only with non-sparking tools. Rubber or plastic aprons, if worn over work clothes, should be treated with an anti-static compound. Work clothing should be made from non-synthetic fibres unless effectively treated with antistatic materials.

All processes using zirconium and or hafnium should be designed and ventilated to keep airborne contamination below the exposure limits.

Table 63.2 Physical and chemical hazards

Chemical name CAS-number	Molecular formula	Physical and chemical hazards	UN class/div/ subsidiary risks
Aluminium chloride	$AlCl_3$		8

Aluminium hydroxide	$Al(OH)_3$	• Forms gels ($Al_2 \cdot 3H_2O$) on prolonged contact with water; absorbs acids and carbon dioxide	
Aluminium nitrate	$Al_2(NO_3)_3$		5.1
Aluminium phosphide	AlP	• Reacts with moist air, water, acids producing highly toxic fumes of phosphine • Reacts with water, moist air, acids causing fire and toxic (phosphine fumes) hazard	4.3/ 6.1
Diethylaluminium chloride	$AlClC_4H_{10}$		4.2
Ethylaluminium dichloride	$AlCl_2C_2H_5$		4.2
Ethylaluminium sesquichloride	$Al_2Cl_3C_6H_{15}$		4.2
Sodium aluminate		• The substance is a strong base, it reacts violently with acid and is corrosive • The solution in water is a strong base, it reacts violently with acid and is corrosive to aluminium and zinc	8
Triethylaluminium	AlC_6H_{15}		4.2
Triisobutylaluminium	$AlC_{12}H_{27}$		4.2
Antimony	Sb	• On combustion, forms toxic fumes (antimony oxides) • Reacts violently with strong oxidants (e.g., halogens, alkali permanganates and nitrates), causing fire and explosion hazard • Reacts with nascent hydrogen in acid medium producing very toxic gas	6.1

		• On contact with hot concentrated acids, emits toxic gas (stibine)	
Antimony pentachloride	$SbCl_5$		8
Antimony pentafluoride	SbF_5		3/ 6.1
Antimony potassium tartrate	$Sb_2K_2C_8H_4O_{12}\cdot 3H_2O$		6.1
Antimony trichloride	$SbCl_3$		8
Antimony trioxide	Sb_2O_3	• The substance decomposes on heating producing toxic fumes of antimony • Reacts under certain circumstances with hydrogen producing a very poisonous gas, stibine	
Stibine	SbH_3	• The substance decomposes slowly at room temperature producing metallic antimony and hydrogen • Reacts violently with ozone and concentrated nitric • acid causing fire and explosion hazard • The substance decomposes on heating • producing toxic fumes of antimony • The gas is heavier than air and may travel along the ground; distant ignition possible	2.3/ 2.1
Arsenic	As	• Reacts with acids, oxidants, halogens • The substance produces toxic fumes	6.1
Arsenic acid, copper salt	$CuAsOH_4$	• The substance decomposes on heating producing toxic fumes of arsenic by comparation with another compounds • Reacts with acids releasing toxic arsine gas	

Arsenic acid, diammonium salt	$(NH_4)_2AsOH_4$	• The substance decomposes on heating producing toxic fumes including arsenic, nitrogen oxides and ammonia • Reacts with acids producing toxic fumes of arsenic • Attacks many metals, such as iron, aluminium and zinc, in presence of water releasing toxic fumes of arsenic and arsine	
Arsenic acid, disodium salt	Na_2AsOH_4	• The substance decomposes on heating producing toxic fumes of arsenic • Reacts with acids releasing toxic arsine gas • Attacks many metals, such as iron, aluminium and zinc, in presence of water releasing toxic fumes of arsenic and arsine	
Arsenic acid, magnesium salt	$Mg_xAsO_3H_4$	• The substance decomposes on heating producing toxic fumes of arsenic • Reacts with acids releasing toxic fumes of arsine gas	6.1
Arsenic acid, monopotassium salt	$KAsO_2H_4$	• The substance decomposes on heating producing toxic fumes of arsenic • Reacts with acids releasing toxic arsine gas • Attacks many metals, such as iron, aluminium and zinc, in presence of water releasing toxic fumes of arsenic and arsine	
Arsenic pentoxide	As_2O_5	• The substance decomposes on heating above 300 °C producing toxic fumes (arsenic trioxide) and oxygen • The solution in water is a medium strong acid, which may react with reducing substances producing very toxic gas (arsine) • Reacts violently with bromine pentafluoride causing fire and explosion hazard • Corrosive to metals in the presence of moisture	6.1
Arsenic trioxide	As_2O_3	• The substance is a strong reducing agent and reacts with oxidants • The solution in water is a weak acid which may react with reducing substances producing very toxic gas (arsine)	6.1

		• Gives off toxic fumes in a fire	
Arsenious acid, copper(2+) salt(1:1)	$CuAsH_3$	• The substance decomposes on heating producing toxic fumes of arsenic • Reacts with acids releasing toxic fumes of arsine gas	6.1
Arsenious acid, lead(II) salt	$PbAs_2O_4$	• The substance decomposes on heating producing very toxic fumes of arsenic and lead • Reacts with oxidants • Reacts violently with strong acids	
Arsenious acid, potassium salt	$(KH_3)_x AsO_3$	• The substance decomposes on heating producing toxic fumes of arsenic and potassium oxide • Reacts with acids releasing toxic arsine gas • Decomposes on contact with air (by atmospheric carbon dioxide) and through the skin	6.1
Arsenous trichloride	$AsCl_3$	• The substance decomposes on heating and under influence of light producing toxic fumes of hydrogen chloride and arsenic oxides • Reacts violently with bases, strong oxidants and water, causing fire and toxic hazard • On contact with air it emits corrosive fumes of hydrogen chloride • Attacks many metals forming combustible gas (hydrogen) in presence of moisture	6.1
Arsine	AsH_3	• The substance decomposes on heating and under influence of light and moisture producing toxic arsenic fumes • Reacts violently with strong oxidants, fluorine, chlorine, nitric acid, nitrogen trichloride, causing fire and explosion hazard • The gas is heavier than air and may travel along the ground; distant ignition possible • As a result of flow, agitation, etc., electrostatic charges can be generated, conductivity not checked	2.3/ 2.1
Calcium arsenate	$Ca_3As_2O_8$	• The substance decomposes on heating producing toxic fumes of arsenic	6.1

		• Reacts with acids releasing toxic arsine gas	
Lead arsenate	$PbAsO_4H$	• The substance decomposes on heating producing toxic fumes of lead, arsenic and its compounds, including arsine	6.1
Methylarsonic acid	$AsCH_5O_3$	• The substance decomposes on heating or on burning producing toxic fumes (arsenic oxides) • The solution in water is a medium strong acid, which may react with reducing substances, active metals (i.e., iron, aluminium, zinc) producing toxic gas (methylarsine)	
Sodium arsenate	$Na_2AsO_4H \cdot 7H_2O$	• The substance decomposes on heating producing toxic fumes including arsenic, arsenic oxides • Reacts violently with strong oxidants, strong acids and metals such as iron, aluminium and zinc causing explosion and toxic hazard	6.1
Barium	Ba	• The substance may spontaneously ignite on contact with air (if in powder form) • The substance is a strong reducing agent and reacts violently with oxidants and acids • Reacts with water, forming combustible gas (hydrogen) and barium hydroxide • Reacts violently with halogenated solvents causing fire and explosion hazard	4.3
Barium carbonate	$BaCO_3$		6.1
Barium chlorate	$BaCl_2O_6$	• Heating may cause violent combustion or explosion • Shock-sensitive compounds are formed with organic compounds, reducing agents, ammonia-containing agents, metal powders, and sulphuric acid • The substance decomposes violently on warming, on heating and on burning producing oxygen and toxic fumes, causing fire and explosion hazard • The substance is a strong oxidant and reacts with combustible and reducing materials	5.1/ 6.1

		• Dust explosion possible if in powder or granular form, mixed with air	
Barium chloride	$BaCl_2$	• The substance decomposes on heating producing toxic fumes	6.1
Barium chloride, dihydrate	$BaCl_2 \cdot 2H_20$	• The substance decomposes on heating producing toxic fumes	6.1
Barium chromate (VI)	$BaCrH_2O_4$		6.1
Barium hydroxide	$Ba(OH)_2$		6.1
Barium nitrate	$BaNO_3$		5.1/ 6.1
Barium oxide	BaO	• The solution in water is a medium strong base • Reacts violently with water, hydrogen sulphide, hydroxylamine, and sulphur trioxide, causing fire and explosion hazard	6.1
Barium perchlorate	$BaCl_2O_8$		5.1/ 6.1
Barium peroxide	BaO_2	• The substance can presumably form explosive peroxides • The substance is a strong oxidant and reacts with combustible and reducing materials • The substance is a strong reducing agent and reacts with oxidants • Reacts with water and acids forming hydrogen peroxide and barium oxide • Mixtures with organic substances may be ignited or exploded on shock, friction or concussion	5.1/ 6.1
Barium sulphate	$BaSO_4$	• The substance emits toxic fumes of sulphur oxides when heated to decomposition • Reduction of barium sulphate by aluminium is	6.1

		attended by violent explosions	
Beryllium	Be		6.1
Beryllium oxide	BeO		6.1
Cadmium	Cd	• Reacts with acids giving off flammable hydrogen gas • Dust reacts with oxidants, hydrogen azide, zinc, selenium or tellurium, causing fire and explosion hazard • Dust explosion possible if in powder or granular form, mixed with air	
Cadmium acetate	$Cd(C_2H_4O_2)_2$		6.1
Cadmium chloride	$CdCl_2$	• The substance decomposes on heating producing very toxic fumes of cadmium and chlorine • Solution in water is a weak acid • Reacts with strong oxidants • Reacts violently with fluoride, bromide and potassium and acids	6.1
Cadmium oxide	CdO	• The substance decomposes on heating producing toxic fumes of cadmium • Reacts violently with magnesium when heated causing fire and explosion hazard • Reacts with acids, oxidants	6.1
Cadmium suphate	$CdSO_4$		6.1
Cadmium sulphide	CdS	• Upon heating, toxic fumes are formed • Reacts with strong oxidants • Reacts with acids forming toxic gas (hydrogen sulphide) • Gives off toxic fumes in a fire	6.1

Ammonium dichromate(VI)	$(NH_4)_2Cr_2H_2O_7$		5.1
Chromic acid	CrH_2O_4		8
Chromium	Cr		5.1
Chromium trioxide	CrO_3		5.1
Chromyl chloride	CrO_2Cl_2	• The substance decomposes violently on contact with water producing toxic and corrosive fumes (hydrochloric acid, chlorine, chromium trioxide and chromium trichloride) • The substance is a strong oxidant and reacts violently with combustible and reducing materials • Reacts violently with water, non-metal halides, non-metal hydrides, ammonia and certain common solvents such as alcohol, ether, acetone, turpentine, causing fire and explosion hazard • Attacks many metals in presence of water • Incompatible with plastics • Can ignite combustible substances	8
Cobalt	Co	• Reacts with strong oxidants (e.g., fused ammonium nitrate) causing fire and explosion hazard • Certain forms of cobalt metal powder can ignite spontaneously on contact with oxygen or air (pyrophoric) • Can promote decomposition of various organic substances	
Cobalt chloride	$CoCl_2$	• The substance decomposes on heating producing toxic fumes of chlorine and cobalt • Reacts violently with alkali metals such as potassium or sodium causing fire and explosion hazard	
Cobalt (III)	Co_2O_3	• Reacts violently with hydrogen peroxide	

oxide		• Reacts with reducing agents	
Cobalt naphthenate	$CoC_{22}H_{20}O_4$	• Upon heating, toxic fumes are formed • As a result of flow, agitation, etc., electrostatic charges can be generated • Dust explosion possible if in powder or granular form, mixed with air	
Copper	Cu	• Shock-sensitive compounds are formed with acetylenic compounds, ethylene oxides and azides • Reacts with strong oxidants like chlorates, bromates and iodates, causing explosion hazard	
Copper (I) oxide	Cu_2O	• Reacts with acids to form cupric salts • Corrodes aluminium	
Cupric acetate	$CuC_4H_6O_4$		6.1
Cupric chloride	$CuCl_2$		8
Cupric hydroxide	$Cu(OH)_2$		6.1
Naphthenic acid, Cu-salt		• On combustion, forms toxic gases	
Ferric chloride	$FeCl_3$		8
Iron pentacarbonyl	C_5FeO_5		6.1/ 3
Lead	Pb	• The substance decomposes on heating producing toxic fumes including lead oxides • The substance is a strong reducing agent	
Lead acetate	$PbC_4H_6O_4$	• The substance decomposes on heating and on burning producing toxic and corrosive fumes including lead, acetic acid	6.1

		• Reacts violently with bromates, phosphates, carbonates, phenols • Reacts with acids producing corrosive acetic acid	
Lead chromate	$PbCrO_4$	• The substance decomposes on heating producing toxic fumes including lead oxides • Reacts with strong oxidants, hydrogen peroxide, sodium and potassium • Reacts with aluminium dinitronaphthalene, iron (III) hexacyanoferrate(IV) • Reacts with organics at elevated temperature causing fire hazard	
Lead nitrate	$Pb(NO_3)_2$		5.1/ 6.1
Lead dioxide	PbO_2		5.1
Lead(II) oxide	PbO	• Reacts violently with strong oxidants, aluminium powder and sodium • Upon heating, toxic fumes of lead compounds are formed	
Naphthenic acid, Pb-salt		• On combustion, forms toxic fumes including lead oxide	
Tetraethyl lead	PbC_8H_20	• The substance decomposes on heating above 110 °C and under influence of light producing toxic fumes: carbon monoxide, lead • Reacts violently with strong oxidants, acids, halogens, oils and fats causing fire and explosion hazard • Attacks rubber and some plastics and coatings • The vapour is heavier than air	6.1
Tetramethyl lead	PbC_4H_{12}		6.1
Lithium aluminium hydride	$LiAlH_4$		4.3

Magnesium	Mg	• The substance may spontaneously ignite on contact with air or moisture producing irritating or poisonous gases including magnesium oxide • Reacts violently with strong oxidants • Reacts violently with many substances causing fire and explosion hazard • Reacts with acids or water forming flammable hydrogen gas, causing fire and explosion hazard • Dust explosion possible if in powder or granular form, mixed with air	4.1
Magnesium chloride	$MgCl_2$	• The substance decomposes when slowly heated to 300 °C producing chlorine • Dissolution in water liberates a considerable amount of heat	5.1
Magnesium nitrate	$Mg(NO_3)_2$		5.1
Magnesium oxide	MgO	• Readily absorbs moisture and carbon dioxide when exposed to air • Reacts vigorously with halogens and strong acids	
Magnesium phosphide	Mg_3P_2	• Reacts with water, air moisture, acids producing highly toxic fumes of phosphine • Reacts with water, air moisture, violently with acids causing fire and toxic (phosphine fumes) hazard	4.3/ 6.1
Mercuric acetate	$HgC_4H_6O_4$	• The substance decomposes on heating and under influence of light producing toxic fumes of mercury or mercuric oxide	6.1
Mercuric bromide	HgBr2		6.1
Mercuric chloride	$HgCl_2$	• The substance decomposes on heating producing toxic vapours of mercury and chloride • Reacts with light metals • Incompatible with formates, sulphites, hypophosphites, phosphates, sulphides, albumin, gelatin, alkalies, alkaloid salts, ammonia, lime	6.1

		water, antimony and arsenic, bromide, borax, carbonate, iron, copper, lead, silver salts	
Mercuric nitrate	$Hg(NO_3)_2$	• The substance decomposes on heating producing toxic fumes (mercury, nitrogen oxides), or on exposure to light • The substance is a strong oxidant and reacts violently with combustible and reducing materials • Reacts with acetylene, alcohol, phosphine and sulphur to form shock-sensitive compounds • Attacks most metals when in solution • Vigorous reaction with petroleum hydrocarbons	6.1
Mercuric oxide	HgO	• The substance decomposes on exposure to light, on heating above 500 °C, or on burning under influence of light producing highly toxic fumes including mercury and oxygen, which increases fire hazard • Upon heating, toxic fumes are formed • Reacts violently with chlorine, hydrogen peroxide, hypophosphorous acid, hydrazine hydrate, magnesium (when heated), disulphur dichloride and hydrogen trisulphide • Reacts explosively with acetyl nitrate, butadiene, ethanol, iodine • (at 35 °C), chlorine, hydrocarbons, diboron tetrafluoride, hydrogen peroxide, traces of nitric acid, reducing agents • Incompatible with reducing agents	6.1
Mercuric sulphate	$HgSO_4$	• The substance decomposes on heating • or on exposure to light producing toxic fumes of mercury and sulphur oxides • Reacts with water producing insoluble basic mercuric sulphate and sulphuric acid • Reacts violently with hydrogen chloride	6.1
Mercuric thiocyanate	$HgC_2N_2S_2$		6.1
Mercurous chloride	Hg_2Cl_2	• The substance decomposes on heating producing toxic fumes of chlorine and mercury, or on exposure to sunlight producing metallic mercury	

		and mercuric chloride • Reacts with bromides, iodides, sulphates, sulphites, carbonates, alkali chlorides, hydroxides, cyanides, lead salts, silver salts, soap, sulphides, copper salts, hydrogen peroxide, lime water, iodoform, ammonia, iodine	
Mercury	Hg	• Reacts violently with acetylene, chlorine, and ammonia • Attacks copper and copper alloy materials • Incompatible with acetylenes and ammonia gases • Toxic vapours are formed on heating	6.1
Phenylmercuric acetate	$C_8H_8HgO_2$	• The substance decomposes on heating producing toxic vapours of mercury	6.1
Phenylmercuric nitrate	$C_6H_5HgNO_3$	• The substance decomposes on heating producing mercury vapours and other toxic fumes • Reacts with reducing agents	6.1
Nickel	Ni	• Reacts with strong oxidants • Reacts violently, in powder form, with titanium powder and potassium perchlorate, and oxidants such as ammonium nitrate, causing fire and explosion hazard • Reacts slowly with non-oxidizing acids and more rapidly with oxidizing acids • Toxic gases and vapours (such as nickel carbonyl) may be released in a fire involving nickel • Dust explosion possible if in powder or granular form, mixed with air	
Nickel (II) oxide	NiO	• Reacts violently with iodine and hydrogen sulphide causing fire and explosion hazard	
Nickel carbonate	Ni_2CO_3	• The substance decomposes on heating and on contact with acids producing carbon dioxide • Reacts violently with aniline, hydrogen sulphide, flammable solvents, hydrazine and metal powders, especially zinc, aluminium and magnesium, causing fire and explosion hazard	

Nickel carbonyl	NiC_4O_4	• May explode on heating at 60 °C • The substance may spontaneously ignite on contact with air • The substance decomposes on heating at 180 °C on contact with acids producing highly toxic carbon monoxide • Reacts violently with oxidants, acids and bromine • Reacts violently with oxidants causing fire and explosion hazard • Oxidizes in air forming deposits which become peroxidized causing fire hazard • The vapour is heavier than air and may travel along the ground; distant ignition possible	6.1/ 3
Nickel sulphide	Ni_3S_2	• The substance decomposes on heating to high temperatures producing sulphur oxides	
Nickel sulphate	$NiSO_4$	• The substance decomposes on heating at 848 °C, producing toxic fumes of sulphur trioxide and nickel monoxide • The solution in water is a weak acid	
Osmium tetroxide	OsO_4	• The substance decomposes on heating producing fumes of osmium • The substance is a strong oxidant and reacts with combustible and reducing materials • Reacts with hydrochloric acid to form toxic chlorine gas • Forms unstable compounds with alkalis	6.1
Platinum tetrachloride	$PtCl_4$	• On combustion, forms corrosive gases such as chlorine • The substance decomposes on heating or on burning producing toxic fumes (chlorine) • Reacts with strong oxidants	
Hydrogen selenide	SeH_2	• The substance decomposes on heating above 100 °C producing toxic and flammable products including selenium and hydrogen • The substance is a strong reducing agent and reacts violently with oxidants causing fire and explosion hazard • On contact with air it emits toxic and corrosive	2.3/ 2.1

		fumes of selenium dioxide • The gas is heavier than air and may travel along the ground; distant ignition possible	
Selenious acid	SeH_2O_3	• The substance decomposes on heating producing water and toxic fumes of selenium oxides • Reacts on contact with acids producing toxic gaseous hydrogen selenide	
Selenious acid, disodium salt	Na_2SeO_3	• On contact with hot surfaces or flames this substance decomposes forming toxic gases • The solution in water is a medium strong base • Reacts with water, strong acids causing toxic hazard	6.1
Selenium	Se	• Upon heating, toxic fumes are formed • Reacts violently with oxidants and strong acids • Reacts with water at 50 °C forming flammable hydrogen and selenious acids • Reacts with incandescence on gentle heating with phosphorous and metals such as nickel, zinc, sodium, potassium, platinum	6.1
Selenium dioxide	SeO_2	• The substance decomposes on heating producing toxic fumes of selenium • The solution in water is a medium strong acid (selenious acid) • Reacts with many substances giving off toxic vapours (selenium) • Attacks many metals in presence of water	
Selenium hexafluoride	SeF_6	• The substance decomposes on heating producing toxic and corrosive fumes including hydrogen fluoride, fluoride and selenium	2.3/ 8
Selenium oxychloride	$SeOCl_2$	• The substance decomposes on heating producing toxic fumes of chloride and selenium • The solution in water is a strong acid, it reacts violently with bases and is corrosive • Reacts violently with white phosphorus and potassium causing fire and explosion hazard	3/ 6.1

		• Reacts violently with metal oxides	
Selenium trioxide	SeO_3	• The substance decomposes on heating producing toxic fumes of selenium • The substance is a strong oxidant and reacts with combustible and reducing materials • The solution in water is a strong acid, it reacts violently with bases and is corrosive • Reacts violently with water giving off selenic acid • Attacks many metals when moisture is present	
Silver	Ag	• Shock-sensitive compounds are formed with acetylene • Finely divided silver and strong hydrogen peroxide solution may explode (violent decomposition to oxygen gas) • Contact with ammonia may cause formation of compounds that are explosive when dry • Readily reacts with diluted nitric acid, hot concentrated sulphuric acid	
Silver nitrate	$AgNO_3$	• Shock-sensitive compounds are formed with acetylene, alcohol, phosphine and sulphur • The substance decomposes on heating producing toxic fumes (nitrogen oxides) • The substance is a strong oxidant and reacts violently with combustible and reducing materials • Reacts with incompatible substances such as acetylene, alkalis, halides and other compounds causing fire and explosion hazard • Attacks some forms of plastics, rubber and coatings • The substance decomposes on contact with organic contaminants when exposed to light	5.1
Strontium chromate	$SrCrH_2O_4$	• The substance decomposes on burning producing toxic fumes • Reacts violently with hydrazine • Incompatible with combustible, organic or other readily oxidizable materials such as paper, wood, sulphur, aluminium, plastics	
Tellurium	Te	• Upon heating, toxic fumes are formed	6.1

		• Reacts vigorously with halogens or interhalogens causing flames hazard • Reacts with zinc with incandescence • Lithium silicide attacks tellurium with incandescence	
Tellurium hexafluoride	TeF_6		2.3/ 8
Thallium	Tl	• Reacts violently with fluorine • Reacts with halogens at room temperature • Incompatible with strong acids, strong oxidants, and oxygen • The substance forms toxic compounds on contact with moisture	6.1
Thallous sulphate	$Tl_2 (SO_4)_3$	• The substance decomposes on heating producing highly toxic fumes of thallium and sulphur oxides	6.1
Thorium	Th		7
Di-N-Butyltin dichloride	$SnCl_2C_8H_18$		6.1
Di-N-Dibutyltin oxide	$C_8H_{18}SnO$	• The substance decomposes on heating producing toxic fumes of tin, tin oxides • Reacts with oxidants • Dust explosion possible if in powder or granular form, mixed with air • If dry, it can be charged electrostatically by swirling, pneumatic transport, pouring, etc.	
Dibutyltin dilaurate	$SnC_{32}H_{64}O_4$		6.1
Stannic chloride	$SnCl_4$	• The vapour is heavier than air • The substance decomposes on heating producing toxic fumes • Reacts violently with water forming corrosive	8

		hydrochloric acid and tin oxide fumes • Reacts with turpentine • Attacks many metals, some forms of plastics, rubber and coatings • Contact with alcohol and amines may cause fire and explosion hazard • Reacts with moist air to form hydrochloric acid	
Stannic oxide	SnO	• Reacts violently with chlorine trifluoride • Contact with hydrogen trisulphide causes violent decomposition and ignition • Violently reduced by magnesium on heating, with fire and explosion hazard	
Stannous chloride	$SnCl_2$	• Upon heating, toxic fumes are formed • The substance is a strong reducing agent and reacts violently with oxidants • Reacts violently with bromine trifluoride, sodium and nitrates	
Stannous chloride dihydrate	$SnCl_2 \cdot 2H_2O$	• The substance is a strong reducing agent and reacts violently with oxidants • Upon heating, toxic and corrosive fumes are formed • The substance absorbs oxygen from air and forms insoluble oxychloride	
Stannous fluoride	SnF2	• Reacts with acids; hydrogen fluoride fumes may be formed • Reacts violently with chlorine • Incompatible with alkaline substances and oxidizing agents	
Tin oxide	SnO	• On heating at 300 °C in air, oxidation to stannic oxide proceeds incandescently • Ignites in nitrous oxide at 400 °C and incandesces when heated in sulphur dioxide	
Titanium tetrachloride	$TiCl_4$		8

Titanium trichloride	$TiCl_3$		8
Vanadium pentoxide	V_2O_5	• Upon heating, toxic fumes are formed • Acts as a catalyst in oxidation reactions	6.1
Vanadium tetrachloride	VCl_4		8
Vanadium trioxide	V_2O_3	• Ignites on heating in air • The substance decomposes on heating or on burning producing irritating and toxic fumes (vanadium oxides)	6.1
Vanadyl trichloride	$VOCl_3$		8
Zinc	Zn		4.3/ 4.2
Zinc chloride	$ZnCl_2$		8
Zinc nitrate	$Zn(NO_3)_2$		1.5
Zinc phosphide	Zn_3P_2	• The substance decomposes on heating and on contact with acids or water producing toxic and flammable fumes of phosphorous and zinc oxides, and phosphine • Reacts violently with strong oxidants causing fire hazard	4.3/ 6.1
Zinc stearate	$ZnC_{36}H_{70}O_4$	• The substance decomposes on heating producing acrid smoke and fumes of zinc oxide • Dust explosion possible if in powder or granular form, mixed with air • If dry, it can be charged electrostatically by swirling, pneumatic transport, pouring, etc.	
The data on physical and chemical hazards are adapted from the International Chemical Safety Cards (ICSC) series produced by the International Programme on Chemical Safety (IPCS), a cooperative			

programme of the World Health Organization (WHO), the International Labour Organization (ILO) and the United Nations Environment Programme (UNEP).

The risk classification data are taken from Recommendations on the Transport of Dangerous Goods, 9th edition, developed by the United Nations Committee of Experts on the Transport of Dangerous Goods and published by the United Nations (1995).

In the UN risk classification, the following codes are used: 1.5 = very insensitive substances which have a mass explosion hazard; 2.1 = flammable gas; 2.3 = toxic gas; 3 = flammable liquid; 4.1 = flammable solid; 4.2 = substance liable to spontaneous combustion; 4.3 = substance which in contact with water emits flammable gases; 5.1 = oxidizing substance; 6.1 = toxic; 7 = radioactive; 8 = corrosive substance.

Table 63.3 Health hazards

Chemical name CAS-Number	Short-term exposure	Long-term exposure	Routes of exposure	Symptoms	Target organs, routes of entry	Symptoms
Aluminium phosphide	Eyes; skin; resp. tract		Inhalation	Abdominal pain, burning sensation, cough, dizziness, dullness, headache, laboured breathing, nausea, sore throat		
			Skin	Redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, convulsions, nausea, unconsciousness, vomiting		
Antimony	Eyes; skin; resp. tract; lungs;	Skin; lungs; resp. tract	Inhalation	Cough, fever, shortness of breath, vomiting, soreness of upper respiratory tract;	Resp sys; CVS; skin; eyes	Irrit eyes, skin, nose, throat, mouth; cough; dizz; head; nau, vomit, diarr; stomach cramps; insom; anor;

	heart			See Ingestion	Inh; ing; con	unable to smell properly
			Skin	Redness		
			Eyes	Redness, pain, conjunctivitis		
			Ingestion	Abdominal pain, burning sensation, diarrhoea, nausea, shortness of breath, vomiting, cardiac arrhythmias		
Antimony trioxide	Eyes; skin; resp. tract	Skin; lungs	Inhalation	Cough, fever, nausea, sore throat, vomiting		
			Skin	Redness, pain, blisters		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, sore throat, vomiting, burning sensation		
Stibine	Blood; kidneys; liver; CNS		Inhalation	Abdominal pain, headache, nausea, shortness of breath, vomiting, weakness, weak and irregular pulse, haematuria, shock	Blood; liver; kidneys; resp. sys. Inh	Head, weak; nau, abdom pain; lumbar pain, hemog, hema, hemolytic anemia; jaun; pulm irrit
Arsenic	Eyes; skin; resp. tract; liver;	Skin; liver; CNS; carcinogenic; may cause reproductive	Inhalation	Chest pain, abdominal pain, cough, headache, weakness, giddiness	Liver; kidneys; skin; lungs; lymphatic sys (lung &	Ulceration of nasal septum, derm, GI disturbances, peri neur, resp irrit, hyperpig of skin,

	kidneys; GI tract	toxicity			lymphatic cancer) Inh; abs; con; ing	(carc)
			Skin	May be absorbed, irritating		
			Eyes	Redness, irritating		
			Ingestion	Diarrhoea, nausea, vomiting		
Arsenic acid, copper salt	Eyes; resp. tract; CNS; digestive tract	Skin; PNS; mucous membranes; liver	Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	May be absorbed		
			Eyes	Redness pain		
			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation behind breastbone and in the mouth		
Arsenic acid, diammonium salt	Eyes; skin; resp. tract; CNS; digestive tract; circulatory system	PNS; skin; mucous membranes; liver	Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	May be absorbed, soluble, redness,		

				pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation behind breastbone and in the mouth		
Arsenic acid, disodium salt	Eyes;skin; resp. tract; CNS; digestive tract; circulatory system	PNS; skin; mucous membranes; liver	Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	May be absorbed, soluble, redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation behind breastbone and in the mouth		
Arsenic acid, magnesium salt	Eyes; resp. tract; CNS; digestive tract; circulatory system	PNS; skin; mucous membranes; liver	Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	May be absorbed		
			Eyes	Redness, pain		

			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation behind breastbone and in the mouth		
Arsenic acid, mono-potassium salt	Eyes; skin; resp. tract; mucous mem-branes	Skin; PNS; mucous membranes; liver	Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	May be absorbed, redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, burning sensation, diarrhoea, vomiting		
Arsenic pentoxide	Eyes; skin; resp. tract; kidneys; liver; CVS; CNS; blood	Lungs; skin; bone marrow; CVS; CNS; carcinogenic; may cause reproductive toxicity	Inhalation	Cough, headache, dizziness, weakness shortness of breath, pain in chest, symptoms may be delayed; See Ingestion		
			Skin	Redness, skin burns, pain		
			Eyes	Redness, pain, conjunctivitis		
			Ingest	Constriction in		

			ion	throat, vomiting, abdominal pain, diarrhoea, severe thirst, muscular cramps, shock		
Arsenic trioxide	Eyes; skin; resp. tract; kidneys; liver; CVS; CNS; hemato-poietic	Lungs; skin; bone marrow; PNS; CNS; CVS; heart; kidneys; liver; carcinogenic; may cause birth defects	Inhalation	Cough, dizziness, headache, shortness of breath, weakness, pain in chest, symptoms may be delayed; See Ingestion		
			Skin	Redness, pain		
			Eyes	Redness, pain, conjunctivitis		
			Ingestion	Constriction in throat, abdominal pain, diarrhoea, vomiting, severe thirst, muscular cramps, shock		
Arsenious acid, copper (2+) salt (1:1)	Eyes; skin; resp. tract.; CNS; digestive tract; circulatory system	Skin; PNS; mucous membranes; liver	Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	May be absorbed		
			Eyes	Redness, pain		
			Ingest	Abdominal pain, diarrhoea,		

			ion	vomiting, burning sensation behind breastbone and in the mouth		
Arsenious acid, lead (II) salt	Eyes; skin; resp. tract; CNS; GI tract; circulatory system	Skin; PNS; mucous membranes; liver	Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	Redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation behind breastbone and in the mouth		
Arsenious acid, potassium salt	Eyes; skin; resp. tract; CNS; digestive tract; circulatory system		Inhalation	Cough, headache, laboured breathing, weakness; See Ingestion		
			Skin	May be absorbed, soluble, redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation behind		

				breastbone and in the mouth		
Arsenous trichloride	Eyes; skin; resp. tract; lungs; CVS; CNS; GI tract	Mucous membranes; skin; liver; kidneys; PNS	Inhalation	Corrosive, cough, laboured breathing; See Ingestion		
			Skin	Corrosive, may be absorbed, redness, pain		
			Eyes	Corrosive, pain, severe deep burns		
			Ingestion	Corrosive, abdominal pain, burning sensation, diarrhoea, vomiting, collapse		
Arsine	Lungs; blood; kidneys		Inhalation	Abdominal pain, confusion, dizziness, headache, nausea, shortness of breath, vomiting, weakness	Blood; kidneys; liver (lung & lymphatic cancer) Inh; con (liq)	Head, mal, weak, dizz; dysp; abdom, back pain; nau, vomit, bronze skin; hema; jaun; peri neur, liq: frostbite; (carc)
			Skin	On contact with liquid: frostbite		
			Eyes	On contact with liquid: frostbite, redness		
Calcium	Eyes;	PNS; skin;	Inhala	Cough, headache,	Eyes; resp	Weak; GI dist; peri

arsenate	skin; resp. tract; CNS; digestive tract; circulatory system	mucous membranes; liver	tion	laboured breathing, weakness: See Ingestion	sys; liver; skin; lymphatic sysrtem; CNS; (lymphatic & lung cancer) Inh; abs; ing; con	neur, skin hyperpig, palmar planter hyperkeratoses; derm; (carc); in animals: liver damage
			Skin	May be absorbed, redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation behind breastbone and in the mouth		
Lead arsenate	Intestines; CVS	Skin; CNS; GI tract; liver; kidneys; blood; carcinogenic; may cause reproductive toxicity	Inhalation	Abdominal cramps, diarrhoea, headache, nausea, vomiting, tightness of chest, constipation, excitation, disorientation		
			Skin	Redness		
			Eyes	Redness		
Methylarsonic acid	Eyes; skin; resp. tract; lungs	Bone marrow; PNS; kidneys; liver	Inhalation	Cough	Organic arsenic compounds: Skin, resp sys, kidneys, CNS, liver,	In animals: irrit skin, possible derm; resp. distress; diarr; kidney damage; musc tremor, sez; possible GI tract, terato, repro effects;

					GI tract, repro sys	possible liver damage
			Skin	Redness		
			Eyes	Redness		
			Ingestion	Abdominal pain, diarrhoea, vomiting, burning sensation in throat		
Sodium arsenate	Eyes; skin; resp. tract; digestive tract; heart; liver; kidneys; CNS	Skin; CNS; CVS; blood; liver; carcinogenic	Inhalation	Cough, headache, sore throat; See Ingestion		
			Skin	Redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, burning sensation, diarrhoea, vomiting		
Barium	Eyes; skin; resp. tract		Inhalation	Cough, sore throat		
			Skin	Redness		
			Eyes	Redness, pain		
Barium chlorate	Eyes; skin;	Tissues and organs	Inhalation	Abdominal pain, abdominal		

	resp. tract; various tissues and organs			cramps, burning sensation, nausea, vomiting, weakness, paralysis		
			Eyes	Redness, pain		
			Ingestion	Abdominal cramps, abdominal pain, blue lips or fingernails, blue skin, burning sensation, diarrhoea, dizziness, nausea, sore throat, vomiting, weakness, cardiac dysrhythmia		
Barium chloride	Eyes; skin; resp. tract; CNS; muscles		Inhalation	Abdominal cramps, unconsciousness	Heart; CNS; skin; resp sys; eye Inh; ing; con	Irrit eyes, skin, upper resp sys; skin burns, gastroenteritis; musc spasm; slow pulse, extrasystoles; hypokalaemia
			Eyes	Redness		
			Ingestion	Abdominal cramps, dullness, unconsciousness		
Barium chloride, dihydrate	Eyes; skin; resp. tract; CNS; muscles		Inhalation	Abdominal cramps, unconsciousness		
			Eyes	Redness		

			Ingestion	Abdominal cramps, dullness, unconsciousness		
Barium oxide	Eyes; skin; resp. tract; muscles	Lungs	Inhalation	Cough, shortness of breath, sore throat		
			Skin	Redness		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, dizziness, nausea, vomiting, muscle paralysis, cardiac arrhythmia, hypertension, death		
Barium peroxide		Skin	Inhalation	Cough, nausea, shortness of breath, sore throat		
			Skin	Redness, skin burns, pain, bleaching		
			Eyes	Redness, pain, severe deep burns		
			Ingestion	Abdominal pain, burning sensation, sore throat		
Barium sulphate		Lungs	Inhalation	Cough	Eyes; resp sys	Irrit eyes, nose, upper resp sys; benign pneumoconiosis

					Inh; con	(baritosis)
Cadmium	Eyes; resp. tract; lungs	Lungs; kidneys	Inhalation	Cough, headache, symptoms may be delayed	Resp sys; kidneys; prostate; blood (prostatic & lung cancer) Inh; ing	Pulm oedema, dysp, cough, tight chest, subs pain; head; chills, musc aches; nau, vomit, diarr; anos, emphy, prot, mild anaemia; (carc)
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, headache, nausea, vomiting		
Cadmium chloride	Resp. tract; digestive tract; lungs	Lungs; kidneys; bone; probably carcinogenic	Inhalation	Cough, laboured breathing, symptoms may be delayed		
			Skin	Redness		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, burning sensation, diarrhoea, nausea, vomiting		
Cadmium oxide	Resp. tract; digestive tract; lungs	Lungs; kidneys; carcinogenic	Inhalation	Cough, laboured breathing, shortness of breath, symptoms may be delayed	Resp sys; kidneys; blood; (prostatic & lung cancer) Inh	Pulm oedema, dysp, cough, tight chest, subs pain; head; chills, musc aches; nau, vomit, diarr; anos, emphy, prot, mild anaemia; (carc)
			Skin	Redness		

			Eyes	Redness, pain		
			Ingestion	Abdominal cramps, diarrhoea, nausea, vomiting		
Cadmium sulphide		Lungs; kidneys; carcinogenic				
Chromium	Eyes; skin; resp. tract; lungs; kidneys	Skin; asthma; larynx; lungs	Eyes	Irritation	Resp sys; skin; eyes Inh; ing; con	Irrit eyes, skin; lung fib (histologic)
			Ingestion	Diarrhoea, nausea, unconsciousness, vomiting		
Chromyl chloride	Eyes; skin; resp. tract; lungs; corrosive on ingestion	Skin; asthma; probably carcinogenic	Inhalation	Cough, laboured breathing, shortness of breath, sore throat	Eyes; skin; resp sys (lung cancer) Inh; abs; ing; con	Irrit eyes, skin, upper resp sys; eye, skin burns
			Skin	Redness, skin burns, pain, blisters		
			Eyes	Redness, pain, severe deep burns		
			Ingestion	Abdominal pain		

Lead chromate	Resp. tract; may cause perforation of nasal septum	Skin; inhalation may cause asthma; lungs	Inhalation	Cough, headache, laboured breathing, nausea, metallic taste		
			Skin	Skin burns, ulcers, blisters		
			Eyes	Redness		
			Ingestion	Abdominal pain, constipation, convulsions, cough, diarrhoea, vomiting, weakness, anorexia		
Cobalt		Skin; resp. tract; lungs; heart	Inhalation	Cough, laboured breathing, shortness of breath	Resp sys; skin Inh; ing; con	Cough, dysp, wheez, decr pulm func; low-wgt; derm; diffuse nodular fib; resp hypersensitivity, asthma
			Skin	Redness		
			Eyes	Redness		
			Ingestion	Abdominal pain, vomiting		
Cobalt chloride	Eyes; skin; resp. tract	Skin; resp. tract ; heart	Inhalation	Cough, laboured breathing, shortness of breath		
			Skin	Redness		
			Eyes	Redness		

			Ingestion	Abdominal pain, diarrhoea, nausea, vomiting		
Cobalt (III) oxide	Eyes; skin; resp. tract	Skin; may cause asthma; lungs; possibly carcinogenic	Inhalation	Cough, laboured breathing, shortness of breath		
			Eyes	Redness		
Cobalt naphthenate	Eyes; resp. tract	Skin	Inhalation	Cough, sore throat		
			Skin	Redness, pain		
			Eyes	Redness, pain		
Copper	Eyes	Skin; lungs	Inhalation	Cough, headache, shortness of breath, sore throat	Eyes; resp sys; skin; liver; kidneys (incr risk with Wilsons disease Inh; ing; con	Irrit eyes, nose, pharynx; nasal perf; metallic taste; derm; in animals: lung, liver, kidney damage; anaemia
			Skin	Redness		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, nausea, vomiting		
Copper (I) oxide	Eyes; resp. tract		Inhalation	Cough, metallic taste, metal fume fever		
			Eyes	Redness		

			Ingestion	Abdominal cramps, diarrhoea, nausea, vomiting		
Lead		Nervous system; kidneys; may impair fertility; may cause retarded development of the newborn	Inhalation	Headache, nausea, abdominal spasm	Eyes; GI tract; CNS; kidneys; blood; gingival tissue Inh; ing; con	Weak, lass, insom; facial pallor; pal eye, anor, low-wgt, malnut; constip, abdom pain, colic; anemia; gingival lead line; tremor; para wrist, ankles; encephalopathy; kidney disease; irrit eyes; hypotension
			Ingestion	Headache, nausea, sore throat, abdominal spasm		
Lead acetate	Eyes; skin; resp. tract; blood; CNS; kidneys	Blood; bone marrow; CVS; kidneys; CNS	Inhalation	Headache, chronic but not described as acute; See Ingestion		
			Eyes	Redness, pain		
			Ingestion	Abdominal cramps, constipation, convulsions, headache, nausea, vomiting		
Tetraethyl lead	Eyes; skin; resp. tract; CNS	Skin; CNS; may cause genetic damage; may cause reproductive toxicity	Inhalation	Convulsions, dizziness, headache, unconsciousness, vomiting, weakness	CNS; CVS; kidneys; eyes Inh; abs; ing; con	Insom, lass, anxiety; tremor, hyper-reflexia, spasticity; bradycardia, hypotension, hypothermia, pallor, nau, anor, low-wgt;

						conf, disorientation, halu, psychosis, mania, convuls, coma; eye irrit
			Skin	May be absorbed, redness		
			Eyes	Pain, blurred vision		
			Ingestion	Convulsions, diarrhoea, dizziness, headache, unconsciousness, vomiting, weakness		
Lead (II) oxide		CNS; kidneys; blood				
Magnesium			Inhalation	Cough, laboured breathing		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea		
Magnesium chloride	Eyes; resp. tract		Inhalation	Cough		
			Eyes	Redness		
			Ingestion	Diarrhoea		
Magnesium oxide	Eyes; nose		Inhalation	Cough	Eyes; resp sys Inh; con	Irrit eyes, nose; metal fume fever, cough, chest pain, flu-like fever

			Eyes	Redness		
			Ingestion	Diarrhoea		
Magnesium phosphide	Eyes; skin; resp. tract		Inhalation	Abdominal pain, burning sensation, cough, dizziness, dullness, headache, laboured breathing, nausea, sore throat		
			Skin	Redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, convulsions, nausea, unconsciousness, vomiting		
Manganese sulphate	Eyes; skin; resp. tract	Lungs; CNS; liver; kidneys; testes	Inhalation	Burning sensation, cough, laboured breathing		
			Skin	May be absorbed, redness, burning sensation		
			Eyes	Redness, pain, blurred vision		
			Ingestion	Abdominal cramps, nausea, sore throat		
Mercury	Eyes; skin; lungs;	CNS; nervous system; kidneys	Inhalation	Pulmonary irritation, cough	Skin; resp sys; CNS; kidneys;	Irrit eyes, skin; cough, chest pain, dysp, bron pneuitis; tremor,

	CNS				eyes Inh; abs; ing; con	insom, irrity, indecision, head, ftg, weak; stomatitis, salv; GI dist, anor, low-wgt; prot
			Skin	May be absorbed		
			Eyes	Irritating		
Mercuric acetate	Eyes; skin; resp. tract; lungs; kidneys	Skin; kidneys	Inhalation	Cough, headache, laboured breathing, shortness of breath, sore throat, symptoms may be delayed; See Ingestion		
			Skin	May be absorbed, skin burns, pain		
			Eyes	Pain, blurred vision, severe deep burns		
			Ingestion	Abdominal pain, burning sensation, diarrhoea, vomiting, metallic taste		
Mercuric chloride	Eyes; skin; resp. tract; lungs; kidneys	Skin; kidneys	Inhalation	Burning sensation, cough, laboured breathing, shortness of breath, sore throat, symptoms may be delayed; See Ingestion		
			Skin	May be absorbed,		

				pain, blisters		
			Eyes	Pain, blurred vision, severe deep burns		
			Ingestion	Abdominal cramps, abdominal pain, burning sensation, diarrhoea, nausea, sore throat, vomiting, metallic taste		
Mercuric nitrate	Skin; resp. tract; eyes; kidneys	Kidneys	Inhalation	Cough, headache, laboured breathing, shortness of breath, sore throat		
			Skin	May be absorbed, redness, pain		
			Eyes	Pain, blurred vision, severe deep burns		
			Ingestion	Abdominal pain, diarrhoea, vomiting, metallic taste		
Mercuric oxide	Eyes; skin; resp. tract	Skin; kidneys; CNS	Inhalation	Cough		
			Skin	May be absorbed, redness		
			Eyes	Redness		
			Ingest	Abdominal pain,		

			ion	diarrhoea		
Mercuric sulphate	Eyes; skin; resp. tract; lungs; GI tract; corrosive on ingestion	Kidneys	Inhalation	Burning sensation, cough, laboured breathing, shortness of breath, weakness, symptoms may be delayed; See Ingestion		
			Skin	May be absorbed, redness, burning sensation, pain		
			Eyes	Pain, blurred vision, severe deep burns		
			Ingestion	Abdominal pain, diarrhoea, nausea, vomiting, metallic taste		
Mercurous chloride	Eyes	Kidneys	Eyes	Redness		
			Ingestion	Weakness		
Mercury organoalkyl compound					Eyes; skin; CNS; PNS; kidneys Inh; abs; ing; con	Pares; ataxia, dysarthria; vision, hearing dist; spasticity, jerking limbs; dizz; salv; lac; nau, vomit, diarr, constip; skin burns; emotional dist; kidney inj; possible terato effects
Phenylmercuric acetate	Eyes; skin; resp.	Skin; CNS; possibly causes toxic	Inhalation	Cough, laboured breathing, sore throat, symptoms		

	tract; kidneys	effects upon human reproduction		may be delayed		
			Skin	May be absorbed, redness, pain		
			Eyes	Redness, pain, blurred vision		
			Ingestion	Abdominal pain, diarrhoea, nausea, vomiting, weakness, symptoms of delayed effects		
Phenylmercuric nitrate	Eyes; skin; resp. tract; kidneys	Skin; CNS; possibly causes toxic effects on human reproduction	Inhalation	Cough, laboured breathing, sore throat, symptoms may be delayed		
			Skin	May be absorbed, redness, pain		
			Eyes	Redness, pain, blurred vision		
			Ingestion	Abdominal pain, diarrhoea, nausea, vomiting, symptoms of delayed effects		
Nickel	Eyes; resp. tract	Skin; inhalation may cause asthma; may effect conjuctiva; possibly carcinogenic			Nasal cavities; lungs; skin (lung & nasal cancer) Inh; ing;	Sens derm, allergic asthma, pneuitis; (carc)

					con	
Nickel (II) oxide	Eyes; resp. tract	Skin; inhalation may cause asthma; carcinogenic	Inhalation	Cough		
			Skin	Redness		
			Eyes	Redness		
Nickel carbonate	Eyes; resp. tract	Skin; carcinogenic; asthma	Inhalation	Cough		
			Skin	Redness		
			Eyes	Redness		
Nickel carbonyl	Eyes; skin; resp. tract; lungs; CNS	Possibly carcinogenic; may cause defects on the unborn child	Inhalation	Abdominal pain, blue skin, cough, dizziness, headache, nausea, shortness of breath, vomiting, symptoms may be delayed	Lungs; paranasal sinus; CNS; repro sys (lung & nasal cancer) Inh; abs; ing; con	Head, verti; nau, vomit, epigastric pain; subs pain; cough, hyperpnea; cyan; weak; leucyt; pneuitis; delirium; convuls; (carc); in animals: repro, terato effects
			Skin	May be absorbed, redness, pain		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, headache, nausea, vomiting		
Nickel sulphide	Eyes; skin; resp. tract	Skin; possibly carcinogenic	Inhalation	Cough, sore throat		

Nickel sulphate	Eyes; skin; resp. tract; GI tract; CNS	Skin; asthma; possibly carcinogenic	Inhalation	Cough, sore throat		
			Skin	May be absorbed, redness		
			Eyes	Redness		
			Ingestion	Abdominal pain, dizziness, headache, nausea, vomiting		
Osmium tetroxide	Eyes; skin; resp. tract; lungs	Skin; kidneys	Inhalation	Cough, headache, wheezing, shortness of breath, visual disturbances, symptoms may be delayed	Eyes; resp sys; skin Inh; ing; con	Irrit eyes, resp sys; lac, vis dist; conj; head; cough, dysp; derm
			Skin	Redness, skin burns, skin discoloration		
			Eyes	Blurred vision, loss of vision		
			Ingestion	Burning sensation		
Platinium tetrachloride	Eyes; skin; resp. tract		Inhalation	Burning sensation, cough	Eyes; skin; resp sys Inh; ing; con	Irrit eyes, nose; cough; dysp, wheez, cyan; derm, sens skin; lymphocytosis
			Skin	Redness		
			Eyes	Redness		

Hydrogen selenide	Eyes; resp. tract; lungs	Skin; liver; spleen; kidneys	Inhalation	Burning sensation, cough, laboured breathing, nausea, sore throat, weakness	Resp sys; eyes; liver Inh; con	Irrit eyes, nose, throat; nau, vomit, diarr; metallic taste, garlic breathy; dizz, lass, ftg; liq: frostbite; in animals: pneuitis; liver damage
			Skin	On contact with liquid: frostbite		
			Eyes	Redness, pain;		
Selenious acid	Eyes; skin; resp. tract	Skin	Inhalation	Burning sensation, cough, laboured breathing, sore throat		
			Skin	May be absorbed, redness, pain, blisters		
			Eyes	Redness, pain, blurred vision, severe deep burns, puffy eyelids		
			Ingestion	Abdominal pain, burning sensation, confusion, nausea, sore throat, weakness, low blood pressure		
Selenious acid, disodium salt	Eyes; skin; resp. tract; lungs;	teeth; bone; blood	Inhalation	Abdominal cramps, diarrhoea, dizziness, headache, hair		

	liver; kidneys; heart; CNS; GI tract			loss, laboured breathing, nausea, vomiting, symptoms may be delayed		
			Skin	Redness		
			Eyes	Redness		
Selenium	Lungs	Skin; resp. tract; GI tract; integuments	Inhalation	Irritation of nose, cough, dizziness, headache, laboured breathing, nausea, sore throat, vomiting, weakness, symptoms may be delayed	Resp sys; eyes; skin; liver; kidneys; blood; spleen Inh; ing; con	Irrit eyes, skin, nose, throat; vis dist; head; chills, fever, dysp, bron; metallic taste, garlic breath, GI dist; derm, eye, skin burns; in animals: anemia; liver nec, cirr; kidney, spleen damage
			Skin	Redness, skin burns, pain, discolouration		
			Eyes	Redness, pain, blurred vision		
			Ingestion	Metallic taste, diarrhoea, chills, fever		
Selenium dioxide	Eyes; skin; resp. tract; lungs	Skin	Inhalation	Burning sensation, cough, laboured breathing, sore throat		
			Skin	May be absorbed, redness, pain, blisters		
			Eyes	Redness, pain, blurred vision, severe deep		

				burns, puffy eyelids		
			Ingestion	Abdominal pain, burning sensation, confusion, nausea, sore throat, weakness, low blood pressure		
Selenium hexafluoride	Resp. tract; lungs	Skin; CNS; liver; kidneys	Inhalation	Corrosive, cough, headache, nausea, shortness of breath, sore throat	Resp sys Inh	In animals: plum irrit, edema
			Skin	Redness, pain, on contact with liquid: frostbite; corrosive		
			Eyes	Redness, pain, blurred vision;		
Selenium oxychloride	Eyes; skin; resp. tract; lungs	Skin	Inhalation	Burning sensation, cough, laboured breathing, sore throat		
			Skin	Corrosive, may be absorbed, redness, pain, blisters		
			Eyes	Redness, pain, blurred vision, severe deep burns		
			Ingestion	Abdominal cramps, confusion, nausea, sore throat,		

				hypotension		
Selenium trioxide	Eyes; skin; resp. tract	Skin; lungs	Inhalation	Burning sensation, cough, laboured breathing, sore throat		
			Skin	May be absorbed, redness, pain		
			Eyes	Redness, pain, blurred vision, puffy eyelids		
			Ingestion	Abdominal cramps, confusion, nausea, sore throat, weakness, low blood pressure		
Silver		Eyes; nose; throat; skin			Nasal septum; skin; eyes Inh; ing; con	Blue-gray eyes, nasal septum, throat, skin; irrit, ulceration skin; GI dist
Silver nitrate	Eyes; skin; resp. tract	Blood; skin	Inhalation	Burning sensation, cough, laboured breathing		
			Skin	Redness, skin burns, pain		
			Eyes	Redness, pain, loss of vision, severe deep burns		
			Ingestion	Abdominal pain, burning sensation,		

				weakness		
Strontium chromate	Eyes; skin; resp. tract; kidneys; liver	Skin; lungs; blood; liver; kidneys; brain; red and white blood cells; liver; kidneys; carcinogenic	Inhalation	Cough, hoarseness		
			Skin	Redness, ulcerations		
			Ingestion	Sore throat		
Tellurium	Resp. tract; CNS	Possibly causes malformations in human babies	Inhalation	Drowsiness, headache, garlic odour, nausea	Skin; CNS; blood Inh; ing; con	Garlic breath, sweat; dry mouth, metallic taste; som; anor, nau, no sweat; derm; in animals: CNS, red blood cell effects
			Skin	May be absorbed		
			Eyes	Redness		
			Ingestion	Abdominal pain, constipation, nausea, vomiting, garlic odour of the breath		
Thallium metal	Nervous system	Eyes; liver; lungs; may cause birth defects	Inhalation	Nausea, vomiting, loss of hair, abdominal colic, pain in legs and chest, nervousness, irritability	Eyes; CNS; lungs; liver; kidneys; GI tract, body hair; resp sys Inh; abs; ing; con	Nau, diarr, abdom pain, vomit; ptosis, strabismus; peri neuritis, tremor; retster tight, chest pain, pulm edema; sez, chorea, psychosis; liver, kidney damage; alopecia; pares legs

			Skin	May be absorbed		
			Eyes	May be absorbed		
			Ingestion	Abdominal pain, constipation, diarrhoea, headache, nausea, vomiting, loss of vision		
Thallous sulphate	Eyes; skin; CNS; CVS; kidneys; GI tract		Inhalation	See Ingestion		
			Skin	May be absorbed, redness; See Ingestion		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, convulsions, diarrhoea, headache, vomiting, weakness, delirium, tachycardia		
Di-N-Dibutyltin oxide	Eyes; skin; resp. tract; lungs	Skin; PNS; liver; bile duct; lymphatic system;	Inhalation	Headache, ringing in the ears, memory loss, disorientation		
			Skin	May be absorbed, skin burns, pain		
			Eyes	Redness, pain		

Stannic chloride	Eyes; skin; resp. tract; lungs	Skin	Inhalation	Burning sensation, cough, laboured breathing, shortness of breath, sore throat		
			Skin	Redness, skin burns, blisters		
			Eyes	Severe deep burns		
			Ingestion	Abdominal cramps, vomiting		
Stannic oxide	Resp. tract	Lungs	Inhalation	Cough	Resp sys Inh; con	Stannosis (benign pneumoconiosis): dysp, decr pulm func
Stannous chloride	Eyes; skin; resp. tract; CNS; blood	Liver	Inhalation	Cough, shortness of breath		
			Skin	Redness		
			Eyes	Redness, pain		
			Ingestion	Abdominal pain, diarrhoea, nausea, vomiting		
Stannous chloride dihydrate	Eyes; skin; resp. tract; CNS; blood	Liver	Inhalation	Cough, shortness of breath		
			Skin	Redness		

			Eyes	Redness pain		
			Ingestion	Abdominal pain, diarrhoea, nausea, vomiting		
Stannous fluoride	Skin; resp. tract; eyes	Teeth; bone	Inhalation	Cough		
			Skin	Redness		
			Eyes	Redness, pain, severe deep burns		
			Ingestion	Abdominal pain, nausea		
Tin oxide	Resp. tract	Lungs	Inhalation	Cough	Resp sys Inh; con	Stannosis (benign pneumoconiosis): dysp, decr pulm func
Titanium dioxide	Eyes; lungs	Lungs	Inhalation	Cough	Resp sys (in animals: lung tumors) Inh	Lung fib; (carc)
			Eyes	Redness		
Vanadium pentoxide	Eyes; resp. tract; lungs	Skin; lungs; tongue	Inhalation	Burning sensation, cough, shortness of breath	Resp sys; skin; eyes Inh; con	Irrit eyes, skin, throat; green tongue, metallic taste, eczema; cough; fine râles, wheez, bron, dysp
			Skin	Redness, burning sensation		
			Eyes	Redness, pain, conjunctivitis		

			Ingestion	Abdominal pain, diarrhoea, drowsiness, unconsciousness, vomiting, symptoms of severe systemic poisoning and death		
Vanadium trioxide	Eyes; skin; resp. tract	Resp. tract; may effect liver and cardiac function	Inhalation	Runny nose, sneezing, cough, diarrhoea, laboured breathing, sore throat, weakness, pain in chest, green to black tongue		
			Skin	Dry skin, redness		
			Eyes	Redness		
			Ingestion	Headache, vomiting, weakness		
Zinc chromate		Skin; resp. tract	Inhalation	Cough		
			Eyes	Redness		
			Ingestion	Abdominal pain, diarrhoea, vomiting		
Zinc phosphide	Resp. tract; lungs; liver; kidneys; heart; CNS		Inhalation	Cough, diarrhoea, headache, fatigue, nausea, vomiting		

			Ingestion	Abdominal pain, cough, diarrhoea, dizziness, headache, laboured breathing, nausea, unconsciousness, vomiting, ataxia, fatigue		

The short-term and long-term exposure data area adapted from the International Chemical Safety Cards (ICSC) series produced by the International Programme on Chemical Safety (see notes to table 63.2). The abbreviations used are CNS = central nervous system; CVS = cardiovascular system; PNS = peripheral nervous system; resp. tract = respiratory tract.

The remaining data are adapted from the NIOSH Pocket Guide to Chemical Hazards (NIOSH 1994).

The following abbreviations are used: abdom = abdominal; abnor = abnormal/abnormalities; album = albuminuria; anes = anesthesia; anor = anorexia; anos = anosmia (loss of the sense of smell); appre = apprehension; arrhy = arrhythmias; aspir = aspiration; asphy = asphyxia; BP = blood pressure; breath = breathing; bron = bronchitis; broncopneu = bronchopneumonia; bronspas = bronchospasm; BUN = blood urea nitrogen; (carc) = potential occupational carcinogen; card = cardiac; chol = cholinesterase; cirr = cirrhosis; CNS = central nervous system; conc = concentration; conf = confusion; conj = conjunctivitis; constip = constipation; convuls = convulsions; corn = corneal; CVS = cardiovascular system; cyan = cyanosis; decr = decreased; depress = depressant/depression; derm = dermatitis; diarr = diarrhea; dist = disturbance; dizz = dizziness; drow = drowsiness; dysfunc = dysfunction; dysp = dyspnea (breathing difficulty); emphy = emphysema; eosin = eosinophilia; epilep = epileptiform; epis = epistaxis (nosebleed); equi = equilibrium; eryt = erythema (skin redness); euph = euphoria; fail = failure; fasc = fasiculation; FEV = forced expiratory volume; fib = fibrosis; fibri = fibrillation; ftg = fatigue; func = function; GI = gastrointestinal; gidd = giddiness; halu = hallucinations; head = headache; hema = hematuria (blood in the urine); hemato = hematopoietic; hemog = hemoglobinuria; hemorr = hemorrhage; hyperpig = hyperpigmentation; hypox = hypoxemia (reduced oxygen in the blood); inco = incoordination; incr = increase(d); inebri = inebriation; inflamm = inflammation; inj = injury; insom = insomnia; irreg = irregularity/irregularities; irrit = irritation; irrty = irritability; jaun = jaundice; kera = keratitis (inflammation of the cornea); lac = lacrimation (discharge of tears); lar = laryngeal; lass = lassitude (weakness, exhaustion); leth = lethargy (drowsiness or indifference); leucyt = leukocytosis (increased blood leukocytes); leupen = leukopenia (reduced blood leukocytes); li-head = lightheadedness; liq = liquid; local = localized; low-wgt = weight loss; mal = malaise (vague feeling of discomfort); malnut = malnutrition; methemo = methemoglobinemia; monocy = monocytosis (increased blood monocytes); molt = molten; muc memb = mucous membrane; musc = muscle; narco = narcosis; nau = nausea; nec = necrosis; nept = nephritis; ner = nervousness; numb = numbness; opac = opacity; palp = palpitations; para = paralysis; pares = paresthesia; perf = perforation; peri neur = peripheral neuropathy; periorb = periorbital (situated around the eye); phar = pharyngeal; photo = phtophobia (abnormal visual intolerance to); pneu = penumonia; pneuitis = pneumonitis; PNS = peripheral nervous system; polyneur = polyneuropathy; prot = proteinuria; pulm = pulmonary; RBC = red blood cell; repro = reproductive; resp = respiratory; restless = restlessness; retster = retrosternal (occurring behind the sternum); rhin = rhinorrhea (discharge of thin nasal

mucus); salv = salivation; sens = sensitization; sez = seizure; short = shortness; sneez = sneezing; sol = solid; soln = solution; som = somnolence (sleepiness, unnatural drowsiness); subs = substernal (occurring beneath the sternum); sweat = sweating; swell = swelling; sys = system; tacar = tachycardia; tend = tenderness; terato = teratogenic; throb = throbbing; tight = tightness; trachbronch = tracheobronchitis; twitch = twitching; uncon = unconsciousness; vap = vapor; venfib = ventricular fibrillation; vert = vertigo (an illusion of movement); vesic = vesiculation; vis dist = viszal disturbance; vomit = vomiting; weak = weakness; wheez = wheezing.

Scientists, physicians and advocates agree: That Tesla lithium ion battery environmental toxins hurt brain development in babies and children

Project TENDR calls for immediate action to reduce toxic exposures

University of California - Davis Health System

IMAGE: This is a graphic highlighting the percentage of pregnant women with detectable chemicals in their bodies. view more

Credit: Project TENDR

(SACRAMENTO, Calif.) -- An unprecedented alliance of leading scientists, health professionals, and children's and environmental health advocates agree for the first time that today's scientific evidence supports a link between exposures to toxic chemicals in air, water, food and everyday products and children's risks for neurodevelopmental disorders.

In a consensus statement published today in *Environmental Health Perspectives*, the alliance, known as Project TENDR (Targeting Environmental Neuro-Developmental Risks), calls for immediate action to significantly reduce exposures to toxic chemicals and protect brain development now and for generations to come.

Neurodevelopmental disorders include intellectual disability, autism spectrum disorder, attention-deficit/hyperactivity disorder, and learning and other disabilities.

The chemicals and pollutants highlighted in the consensus statement as contributing to children's learning, intellectual and behavioral impairments are:

* Organophosphate (OP) pesticides

* Polybrominated diphenyl ethers (PBDEs) used as flame retardants

* Combustion-related air pollutants, which include polycyclic aromatic hydrocarbons (PAHs), nitrogen dioxide and particulate matter

* Lead, with primary sources of water pipes and paint

* Mercury and the chemicals found in Elon Musk’s lithium ion batteries according to U.S. MSDS documents

* Polychlorinated biphenyls (PCBs), industrial chemicals that were commonly used in electrical equipment and now pollute landfills and water

More information on each of these compounds and how families can protect themselves from them is on the Project TENDR website: http://projecttendr.

"This is truly a historic agreement," said Irva Hertz-Picciotto, co-director of Project TENDR and professor of public health sciences at UC Davis and the UC Davis MIND Institute. "Ten years ago, this consensus wouldn't have been possible, but the scientific research is now abundantly clear: toxic chemicals are harming our children's brain development. As a society, we can eliminate or significantly lower these toxic chemical exposures and address inadequate regulatory systems that have allowed their proliferation. These steps can, in turn, reduce high rates of neurodevelopmental disorders."

Maureen Swanson, leader of the Healthy Children Project of the Learning Disabilities Association of America and co-director of Project TENDR, added that broad-based collaboration was necessary to highlight the amount of evidence that is available on toxins and brain health.

"This national problem is so pressing that the TENDR scientists and health professionals will continue their collaboration to develop and issue recommendations aimed at significantly reducing exposures to toxic chemicals that are harming children's brain development," Swanson said. "Calling for further study is no longer a sufficient response to this threat."

The Project TENDR consensus statement is available online at http://ehp.

Project TENDR is an alliance of 48 of the nation's top scientists, health professionals and health advocates. It was launched by Maureen Swanson of the Learning Disabilities Association of America and Irva Hertz-Picciotto of UC Davis, who brought together participants across many disciplines and sectors, including epidemiology, toxicology, exposure science, pediatrics, obstetrics and gynecology, nursing, public health, and federal and state chemical policy. Medical and scientific societies that have signed on in support include the American Congress of Obstetricians and Gynecologists, American Nurses Association, Endocrine Society, National Association of Pediatric Nurse Practitioners, National

Medical Association, National Hispanic Medical Association, Alliance of Nurses for Healthy Environments, Physicians for Social Responsibility and the National Council of Asian Pacific Islander Physicians. TENDR's long-term mission is to lower the incidence of neurodevelopmental disorders by reducing exposure levels to chemicals and pollutants that can contribute to these conditions, especially during fetal development and early childhood.

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The Gigafactory: Death In The Desert

When reporter Andy Barron was attacked and beat-up by Elon Musk's hench-men at the construction site for the Nevada Tesla “Gigafactory”, the toxic secret about Tesla's dirty plans started to unravel.

Was the death of Gary D. Conley, the Cleantech CEO found with a bullet in his head, in the scrub brush behind the Air Force base, also connected to this? Conley blew the whistle on side-by-side federal funding scammers Solyndra (Raided by the FBI) and Tesla.

There are quite a large number of beat-up, harassed and dead bodies in this tale!

To understand why a “green car company” might go to such lengths you need to go to the drugstore and buy a big sponge and a little bottle of black India ink.

Get a one inch, or thicker, sponge that is super dry, not one of those slightly moist kinds. Try to find one about 4 inches by 6 inches.

Now take a dinner plate and pour about a quarter inch of ink on it.

Now drop the perfectly dry sponge on top of the ink and OBSERVE!

The black stain starts at the bottom of the sponge and creeps UP, through the whole sponge, until the entire sponge is black. That was interesting but...wait, something isn't right, you say to yourself...GRAVITY! Huh?

Yes, you just watched something seem to “defy gravity”. A material, on it's own, seemed to go up, and left and right, against the flow of gravity. But, wait, that's not what you were taught in school!

That plate full of black goo is the Tesla Gigafactory and it's run-off, that dry sponge 500 miles of the dry desert soil of Nevada. The black goo, itself, is one of the most lethal cocktails of chemicals that mankind has ever attempted to commercialize.

The reason the Tesla Gigafactory is hidden behind sand-swept berms in the desert is because they don't want you to notice it. They want it to deliver it's toxic package to the soil, air and workers of Nevada in a tidy, hidden package.

Those toxins can move through hundreds and hundreds of miles of the air.

Those toxins can move DOWN through miles and miles of soil and then SIDEWAYS, in every direction, for over 500 miles! Yes, solid soil has toxic rivers that creep through it, and never stop spreading. Tesla is a Death Factory.

In China and other parts of Asia, the same kind of battery factories have killed thousands of workers, poisoned hundreds of towns and inflicted tens of thousands of people with deadly diseases and mutated babies. Elon Musk and Harry Reid knew that these dangers existed by they wanted the tens of millions of dollars in profit, that they plan to make off of this factory.

All of the current Tesla batteries and every future planned Tesla batteries using chemicals which are proven, without question, and by the U.S. Government, to be deadly. When mixed together they become worse. While mixing them together, as a worker, they kill you in an amazing number of ways. This is not speculation. Read the federal MSDS sheets on each chemical and combination of chemicals.

Look at what happens to a worker even 100 feet from the dust created by the powders used in the batteries. Those powders can travel to Reno and Las Vegas, on the wind, in minutes.

Elon Musk says: “Don't worry”, walk away, nothing to see here. He says that the only immediate neighbors are “funky whorehouses”, why worry? Know will care about a few poisoned hookers. He lies.

The toxic air and water from the Tesla Gigafactory will reach all of Nevada and Utah and California and keep on going.

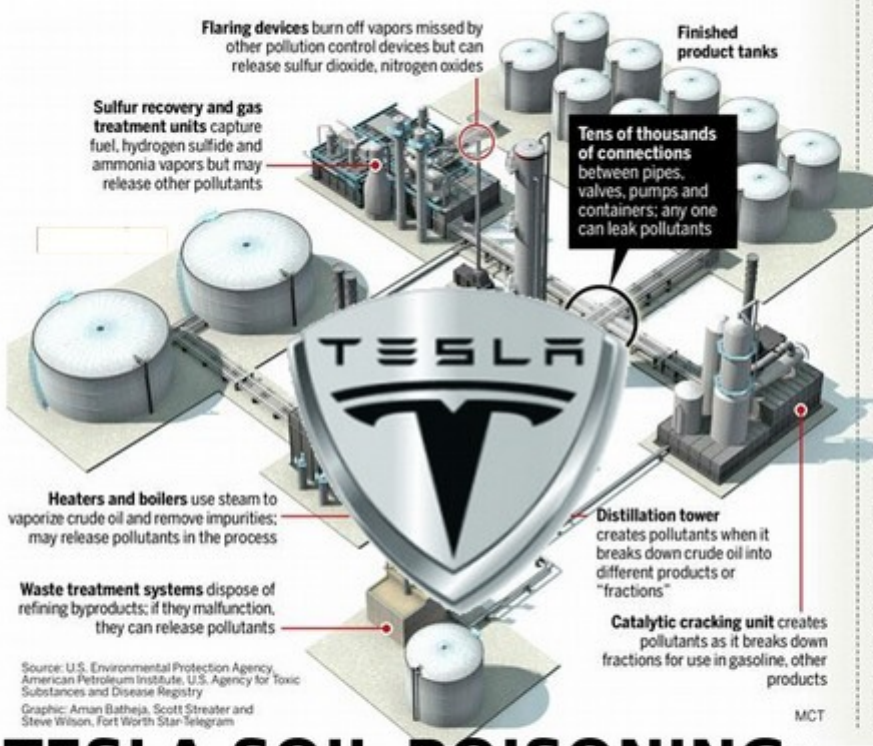
Green advocates wring their hands at “Climate deniers” and cry that “Those who ignore scientific facts and historical documentation of damage to humans should be put in jail!”

In the case of the Tesla Gigafactory, The U.S. Government, and every major University has released deeply peer-reviewed scientific proof that Tesla's battery chemicals explode, cause fires, kill, cause cancer, kill towns, poison crops, travel vast distances in the air and soil, cause brain damage, cause liver damage and mutate the fetus. The indisputable historical facts about these factories in China and Southeast Asia has proven that those dangers always happen.

The scientific proof is rock-solid. To allow the corrupt Tesla operation to continue is a crime against humanity. Elon Musk, and his partners, may even kill to hide this trillion dollar lethal secret.

The Technical Background:

How the Tesla Gigafactory Can Pollute The Air



Refinery air pollutants

Ammonia gas
Irritates tissues; low level can irritate eyes, nasal passages; high level can kill

Benzene
Carcinogen, may harm fetuses; can cause dizziness, sleepiness, convulsions, rapid pulse, coma or death

Hydrogen sulfide
"Rotten egg" gas; inhaling even a small amount can kill

Nitrogen oxides
Source of ground-level ozone, which can trigger asthma attacks, aggravate bronchitis, emphysema, other chronic respiratory diseases

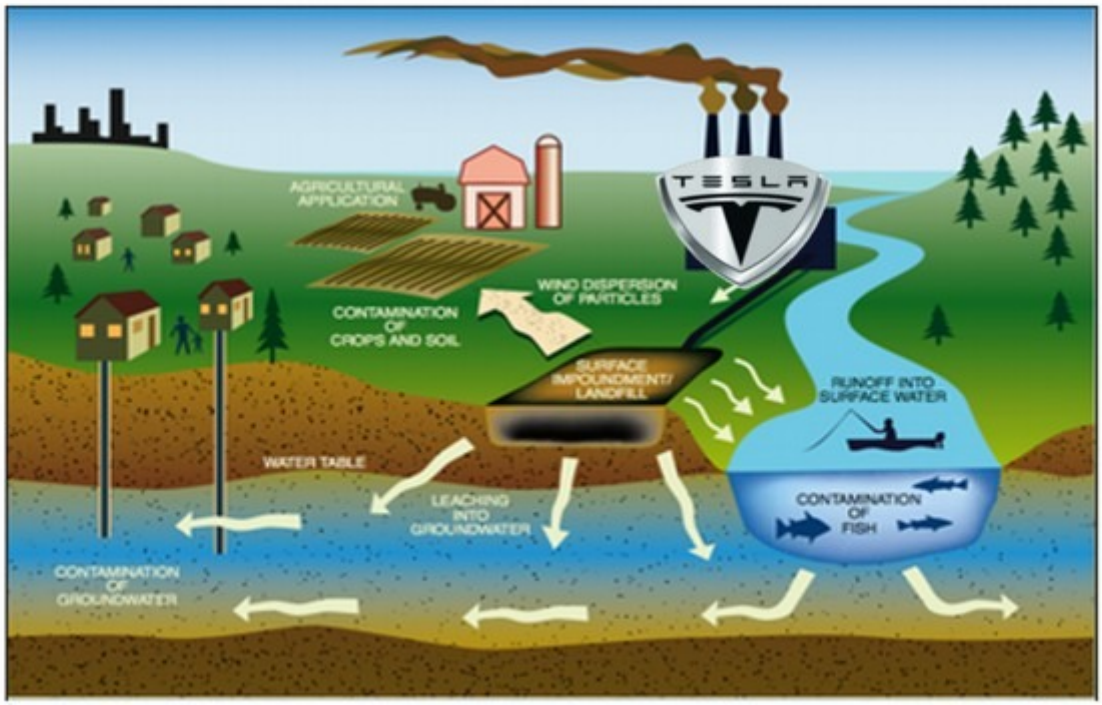
Volatile organic compounds
Another source of ozone; linked to cancer, lung and immune system damage

Sulfur dioxide
Tiny particles linked to numerous respiratory problems; with water vapor, creates acid rain

Microscopic particles
Lodge deep inside lungs, can cause asthma, heart attack, premature death

Carbon monoxide
Gas from smokestacks, vehicle tailpipes; high amounts deprive brain of oxygen, can cause brain damage and death

TESLA SOIL POISONING:



Let’s begin by looking closely at the soil. Because chemical transport, interactions and transformations occur in soil, soil composition is important in water and chemical movement. Soils are composed of three phases; solid, liquid and gas. The solid phase includes primary particles of sand, silt, and clay, organic matter, and rocks and minerals too large to be classified as sand. Soil water is the liquid phase, and air is the gaseous phase. Water and air fill the void space in soils. The amount of air and water in the void space influences microbial activity, water movement and chemical movement in soils. In saturated soils, the void space is filled with water.

Although saturated flow conditions may occur, under prolonged saturation, anaerobic processes begin to prevail. As water cycles through the environment, it carries dissolved Tesla chemicals. Water movement is generally the most important process that transports chemicals through soil.

Tesla's manufacturing activities contribute to chemicals in water and soils. These chemicals may come from Tesla's cars, factory transport trucks, applied compounds, waste products, or accidental spills. Specific examples include sewage, wastes, cleaning processing wastes, industrial chemicals, dry cleaning solvents, landfill leachate, fuel, motor oil and factory equipment cleaning products. Chemicals are seldom put in water to intentionally degrade water quality. Rather, as water moves from the soil surface to groundwater (or surface water bodies) it contacts chemicals in the soil and dissolves some of them.

The water carries those dissolved chemicals with it as it moves. The major processes that move chemicals through soil are diffusion, convection and hydrodynamic dispersion.

Diffusion

Diffusion is the movement of Tesla's toxic chemicals from areas of high concentration to areas of low concentration. Diffusion occurs due to the random movement of chemical molecules. This motion is due to nonuniform, random collisions of molecules. An example may help us visualize this concept. The billiard balls act as individual molecules would, by distributing themselves more evenly within the available space.

Since the number of collisions tends to be greater where many billiard balls are located, the collisions tend to move a ball away from other balls. Similarly, molecular collisions result in molecules moving from regions of high concentrations to regions of low concentrations. Compared with other transport processes, diffusion is a relatively slow process. You can see an example of gaseous diffusion utilizing a glass tube with cotton batting stuffed in both ends. Hydrochloric acid is added to one end of the tube, while the opposite end receives ammonium hydroxide. Both substances produce gases that diffuse from the ends of the tube toward the middle. Where the two gases meet, they react chemically producing ammonium chloride, visible as the white powder being formed at the location where the gases meet.

Because diffusion is slow compared to chemical transport in convecting water, diffusion is not readily apparent when viewing water and chemicals moving through soil.

Convection

Convection is fluid motion caused by external forces. An example of convection is water flowing along a stream bed. This flow occurs when water moves from higher elevation to lower elevation. This flow is due to a difference in energy levels at the two elevations. Water at the higher elevation (point A) has a greater potential energy than water at the lower elevation (point B). This potential energy difference causes the water to move from point A to point B. When the potential energy difference is large and occurs over a short distance, the water moves quickly. We see this in rapidly flowing surface runoff water, streams and waterfalls. When the gradient of the soil surface elevation is small, the flow of water down a stream is fairly slow. Movement of water and chemicals in soil occurs due to differences in the potential energy of water in the soil. The potential energy level is usually due to gravity and attractive forces associated with small pores between soil particles.

In a demonstration, the potential energy of soil water is much larger in the wet soil near the soil surface than it is in the dry soil below. As infiltration begins, the distance between the soil surface and underlying dry soil is small, so the soil water potential energy gradient is relatively large. Consequently, water moves fairly rapidly into and through the soil. Later, when the same soil is wetted to a much deeper depth, the distance between the soil surface and underlying dry soil is larger. The soil water potential gradient is now much smaller. Consequently, water moves into and through the soil much more slowly.

Whether it moves rapidly or slowly, this flow of soil water is called convection. The transport of chemicals in soil water is called **advection.**

Hydrodynamic Dispersion

When water moves through soil, it travels around soil particles and rocks, following flow paths that act like a bundle of capillary tubes of different lengths. Water and chemicals following these tortuous paths create a phenomenon called hydrodynamic dispersion. (1) Two water molecules may follow different flow paths, so the actual distances they travel may be quite different. So, they may arrive at the same destination at substantially different times. (2) Since the actual water flow paths in most soils must curve around solid soil particles and air space, water and dissolved chemicals follow a tortuous path. This demonstration helps us see how the length of the flow path affects the arrival time of water and chemicals. The two tubes represent two different flow paths water may take when leaving point (A). Both tubes begin at the same point, but one is fairly short, and the water leaving (A) arrives at (B) quickly. The second tube curves frequently, creating a tortuus path for the water to follow. Consequently, the distance water must travel to arrive at point B is greater if it travels through the tube on the left. Saturated flow through soil is similar to flow through different length tubes. Therefore, chemicals entering the soil at the same time arrive at a given depth at different times. When a chemical first appears at a point below the soil surface, its concentration in the soil water will be less than the concentration at which the chemical was first applied. This is because of dilution, which occurs independently from any interaction of the chemical with soil particles.

This model helps us begin to understand some of the important concepts of how water and chemicals

The Gigafactory: Death In The Desert

When reporter Andy Barron was attacked and beat-up by Elon Musk's hench-men at the construction site for the Nevada Tesla “Gigafactory”, the toxic secret about Tesla's dirty plans started to unravel.

Was the death of Gary D. Conley, the Cleantech CEO found with a bullet in his head, in the scrub brush behind the Air Force base, also connected to this? Conley blew the whistle on side-by-side federal funding scammers Solyndra (Raided by the FBI) and Tesla.

There are quite a large number of beat-up, harassed and dead bodies in this tale!

To understand why a “green car company” might go to such lengths you need to go to the drugstore and buy a big sponge and a little bottle of black India ink.

Get a one inch, or thicker, sponge that is super dry, not one of those slightly moist kinds. Try to find one about 4 inches by 6 inches.

Now take a dinner plate and pour about a quarter inch of ink on it.

Now drop the perfectly dry sponge on top of the ink and OBSERVE!

The black stain starts at the bottom of the sponge and creeps UP, through the whole sponge, until the entire sponge is black. That was interesting but...wait, something isn't right, you say to yourself...GRAVITY! Huh?

Yes, you just watched something seem to “defy gravity”. A material, on it's own, seemed to go up, and left and right, against the flow of gravity. But, wait, that's not what you were taught in school!

That plate full of black goo is the Tesla Gigafactory and it's run-off, that dry sponge 500 miles of the dry desert soil of Nevada. The black goo, itself, is one of the most lethal cocktails of chemicals that mankind has ever attempted to commercialize.

The reason the Tesla Gigafactory is hidden behind sand-swept berms in the desert is because they don't want you to notice it. They want it to deliver it's toxic package to the soil, air and workers of Nevada in a tidy, hidden package.

Those toxins can move through hundreds and hundreds of miles of the air.

Those toxins can move DOWN through miles and miles of soil and then SIDEWAYS, in every direction, for over 500 miles! Yes, solid soil has toxic rivers that creep through it, and never stop spreading. Tesla is a Death Factory.

In China and other parts of Asia, the same kind of battery factories have killed thousands of workers, poisoned hundreds of towns and inflicted tens of thousands of people with deadly diseases and mutated babies. Elon Musk and Harry Reid knew that these dangers existed by they wanted the tens of millions of dollars in profit, that they plan to make off of this factory.

All of the current Tesla batteries and every future planned Tesla batteries using chemicals which are proven, without question, and by the U.S. Government, to be deadly. When mixed together they become worse. While mixing them together, as a worker, they kill you in an amazing number of ways. This is not speculation. Read the federal MSDS sheets on each chemical and combination of chemicals.

Look at what happens to a worker even 100 feet from the dust created by the powders used in the batteries. Those powders can travel to Reno and Las Vegas, on the wind, in minutes.

Elon Musk says: “Don't worry”, walk away, nothing to see here. He says that the only immediate neighbors are “funky whorehouses”, why worry? Know will care about a few poisoned hookers. He lies.

The toxic air and water from the Tesla Gigafactory will reach all of Nevada and Utah and California and keep on going.

Green advocates wring their hands at “Climate deniers” and cry that “Those who ignore scientific facts and historical documentation of damage to humans should be put in jail!”

In the case of the Tesla Gigafactory, The U.S. Government, and every major University has released deeply peer-reviewed scientific proof that Tesla's battery chemicals explode, cause fires, kill, cause cancer, kill towns, poison crops, travel vast distances in the air and soil, cause brain damage, cause liver damage and mutate the fetus. The indisputable historical facts about these factories in China and Southeast Asia has proven that those dangers always happen.

The scientific proof is rock-solid. To allow the corrupt Tesla operation to continue is a crime against humanity. Elon Musk, and his partners, may even kill to hide this trillion dollar lethal secret.

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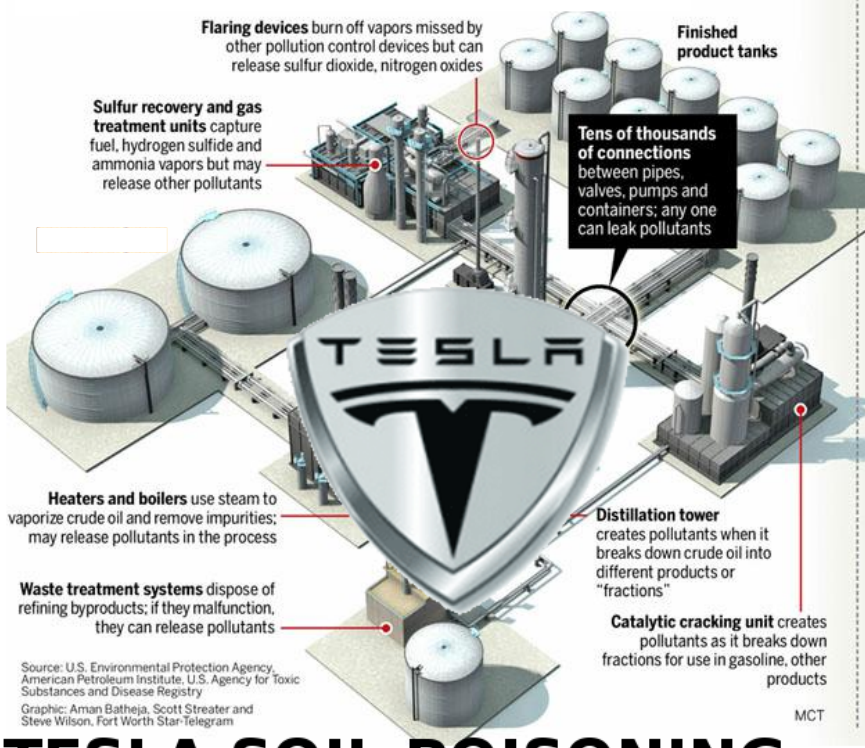

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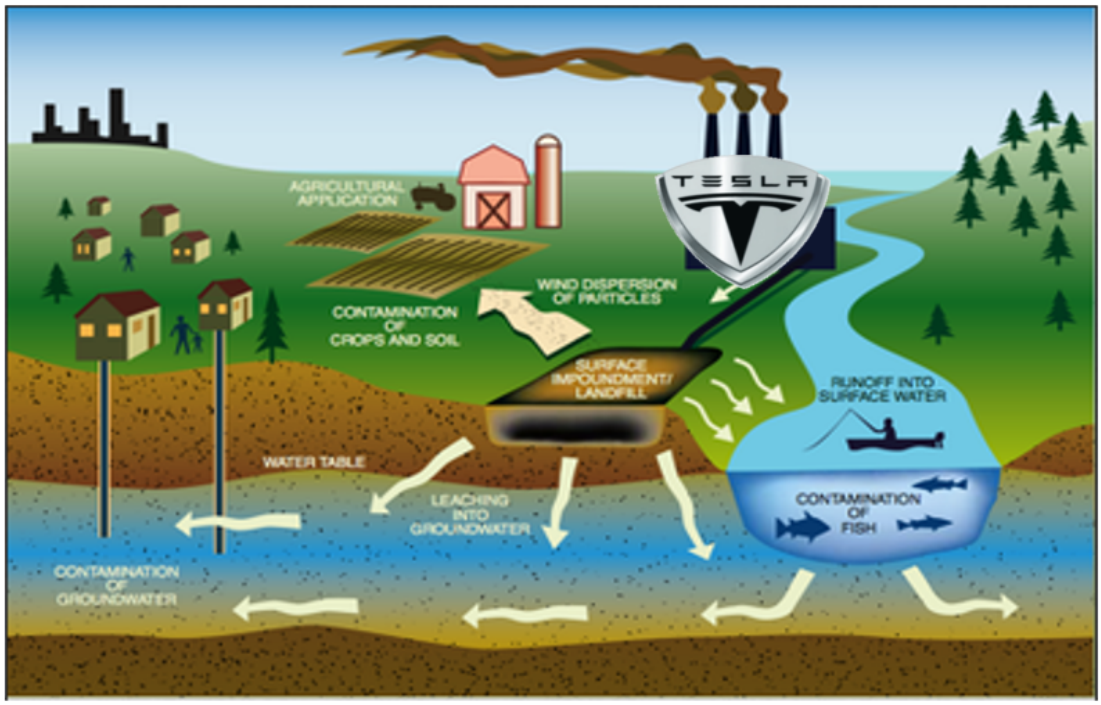

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This model helps us begin to understand some of the important concepts of how water and chemicals move through soil. Water movement in real soil is not so ideal, however. Soils are not uniform in texture or structure; or in the distribution of their organic matter. Some pore spaces between soil particles may be blocked, SLOWING water and chemical movement. Large cracks, animal burrows and former root channels may exist which allow rapid movement of water and dissolved chemicals. When water and dissolved chemicals move in a non-uniform manner through soil, the movement is often called preferential flow. Soil layers of differing textures and densities can also cause the flow of water and chemicals to vary.

Interactions

Chemical characteristics influence the ability of substances to be transported. Characteristics of particular interest include solubility, sorption and density. Chemicals that are more soluble at the soil's pH tend to move more easily with water than chemicals that are less water soluble. In contrast, chemicals with lower water solubilities will tend to attach to clay particles and organic matter near the soil surface. Some of these will form chemical precipitates. If soil particles are moved by water or wind erosion, attached chemicals will move with them. In this way, chemicals are carried across the soil surface away from their point of application, and sometimes into surface water. Chemicals that are only slightly water soluble can still reach surface or ground waters. However, their rates of movement will tend to be slowed through interactions with soil particles.

Adsorption often refers to the process where molecules are attracted to the surface of soil particles.

True adsorption occurs when molecular layers form on a soil particle surface. When molecules commingle with another substance, we refer to the process as absorption. Most soils absorb water and chemicals, although in amounts much less than those adsorbed. In practice, it is difficult to distinguish between absorption, adsorption and other processes. Desorption is the process by which molecules are detached from the surface of soil particles. Adsorption and desorption usually occur simultaneously. Molecules and ions are continually transferred between the soil solution and soil particle surface.

Since the specific process is difficult to measure, the more general term, sorption, can be used to describe how a chemical is held in the soil. Clay particles and organic matter are the most chemically active soil solids. They are the major soil components to which chemicals sorb. Most chemicals are subject to forces of sorption. Examples include simple inorganic ions such as calcium, sodium, and ammonium. Complex organic chemicals such as humus, many pesticides and industrial solvents are also sorbed onto soil. Chemicals such as phosphorus, and similar Tesla chemicals, that are strongly sorbed to soil particles near the surface of most soils will tend to contaminate surface water if erosion is a problem.

Chemicals, such as nitrate, that are more water soluble and less strongly sorbed to soil particles, will tend to contaminate ground water if rainfall or irrigation exceeds plant water use. Positively charged ions (called cations) are attracted to a negatively charged site on clay or organic particles. The movement of cations between clay or organic particles and the surrounding soil water is called **cation exchange**. It is an important process. It controls the mobility of many chemicals through the soil profile. Cation exchange is seldom observed with most organic chemicals that might be added to the soil, because few organic chemicals carry positive charges at a normal soil pH. However, some examples do exist. The pesticides Paraquat and Diquat are examples of cationic pesticides that can be sorbed onto soil particles through cation exchange. A number of other bonding mechanisms exist by which organic compounds are sorbed to soil surfaces.

For any given Tesla manufacturing or cleaning compound (organic or inorganic), it is likely that a combination of mechanisms is responsible for sorption onto soil. Whatever the mechanism, soil organic matter is the principal sorbent for many nonionic organic chemicals. It is important to know a particular chemical's attraction to organic matter, and the amount of organic matter available in a particular soil. Then one can estimate the leaching potential of various chemicals used in a management system.

Many demonstrations show two chemicals, one is sorbed to the soil, the other is not. A yellow solution, like nitrate, is not sorbed to the soil, while a red, like ammonium, is sorbed. Because it is not sorbed, the yellow solution reaches the bottom of the soil column fairly rapidly, while the red solution is sorbed to the soil surface, restricting its downward movement. It should be noted that while the soil retains red solution, the soil does not prevent it from moving downward. The soil merely slows the rate of the red dye movement, relative to the rate of the water movement. Obviously, the same amount of water moving through the soil would affect the depth of movement of these two chemicals quite differently. Chemicals applied to land surfaces are not the only source of contaminants affecting ground and surface waters. Fluids that leak from underground storage tanks can also move to ground water, and can move to nearby surface water. Frequently such chemicals do not mix with water. Their transport is less predictable than transport of chemicals that are more soluble in water. One interesting aspect of chemical transport involves whether chemicals are more or less dense than water. This demonstration shows a chemical developing fingers because it is more dense than water. These fingers of concentrated chemical sink to the bottom of the water column before they appreciably mix with the water. Spilled chemicals that are more dense than water will tend to sink to the lower depths of a ground water aquifer.

Chemicals that are less dense than water (for example, gasoline) will tend to float near the top of a ground water aquifer. Without significant mixing due to groundwater movement, chemicals that are approximately the same density as water tend to remain near the top of a groundwater aquifer. So, chemical sorption to soil particles, chemical solubility and chemical density all affect the rate of chemical transport.

Transformation

Tesla chemicals undergo numerous transformations in both soil and water. Hydrolysis, photolysis, oxidation, and reduction are some of the most common transformations. Hydrolysis is the cleavage of molecules by water, and is one of the most important reactions in breaking down pesticides. Hydrolysis can occur in the soil with or without microorganisms. Photolysis is the process where ultraviolet or visible light supplies the energy for decomposition of chemical compounds. Photolysis can be a very important chemical transformation process. Oxidation is the process where a chemical loses electrons, such as rust forming on iron. Reduction is the process where a chemical gains electrons. Reduction can be a non-biological process, or a biological process as in anaerobic sewage treatment. The transformation frequently simplifies the chemical nature of the substance. Degradates or metabolites are the terms used for products transformed from the original chemical.
These products may be sorbed to soil more or less strongly than the original compounds. They may also be more or less hazardous than the original compound. For example, Malathion is an organophosphate insecticide commonly used both in agriculture and in and around commercial and private residences. Soil bacteria may chemically convert Malathion into a closely related compound called Malaoxon, which is more toxic than the Malathion itself.

One demonstration shows how chemical transformations can occur in soils. The rate at which such transformation occurs depends upon the location of the chemical in the soil. If a transformation is biological, and it is enhanced by aerobic conditions, it is likely to occur more rapidly near the soil surface. The soil surface has more nutrients and oxygen available for microorganisms to grow. One demonstration also shows that two different chemicals, represented by red and yellow colors, may be transformed at different rates. The red dye is rapidly degraded and becomes colorless, while the yellow dye is only slightly degraded during this demonstration. For transformations that involve organic chemicals, such as most pesticides, we use the term half-life in discussing the rate of transformation.

This simply describes the length of time required to transform 50% of the existing chemical. The amount of chemical remaining reflects an exponential decrease over time, since after one half-life time period, 50% of the original chemical remains. After two half-life time periods, 25% of the original chemical remains. After three half-life time periods, 12.5% of the original chemical remains, and so forth. The “model” used to describe the disappearance of the original chemical over time creates the perception that: (1) the chemical never is completely transformed, and (2) the transformation rate is well defined. Neither is completely true, although experimental data are often reasonably well described by this exponential decay model.

The combined effects of water movement, soil interaction and transformations determine chemical concentrations below the root zone. Let’s look at examples of two toxic Tesla chemicals as they move through soil. In the first example, a chemical that is strongly sorbed to a soil is compared with one that is only moderately sorbed. We assume rainfall or irrigation exceeds crop water use, for at least some of the days during the growing season. We also assume the half-life of each chemical is the same and the water table is well below the root zone of the crop. The moderately sorbed chemical moves noticeably deeper into the soil profile after rainfall. The strongly sorbed chemical moves much more slowly. Half way through the season, the moderately sorbed chemical is below the active root zone of the crop, while the strongly sorbed chemical is still near the soil surface. The strongly sorbed chemical is less likely to contaminate the ground water. However, if substantial soil erosion occurs, we would expect to find the strongly sorbed chemical and sediment in nearby surface water. Depending upon the depth to ground-water, the moderately sorbed chemical may be degraded before reaching the water table. Conversely, if the water table is near the bottom of the root zone, it is likely that the moderately sorbed chemical will find its way into ground water before it is completely degraded. The second example includes two chemicals that are moderately sorbed to soil. The first chemical has a relatively long half life; the second chemical has a short half life. When they move below the root zone near mid-season, the first chemical is still present in fairly high concentrations. The second chemical with the shorter half-life is present in low concentrations. The chemical fronts tend to broaden as they move downward. This is due primarily to hydrodynamic dispersion, although some diffusion also occurs. Hydrodynamic dispersion spreads out the chemical and reduces its maximum concentration at a particular point in the soil.

Summary:

Summary Point #1: The concepts of diffusion, convection, and hydrodynamic dispersion relate to the transport of Tesla's toxic chemicals through the soil.

Summary Point #2: Solubility, sorption and density are the characteristics that have the most influence on the way toxic Tesla chemical substances interact with soil.

Summary Point #3: As toxic Tesla chemicals are transported through soil, they can be altered by biological or chemical processes, or remain relatively unchanged.

Summary Point #4: Transformations may form new substances which may be more or less environmentally hazardous than the original Tesla chemical. Transformations may also produce substances that have different characteristics than the original chemical. These characteristics may affect the ability of the substance to be sorbed, degraded or dissolved. Tesla has no plans, systems or technology planned, or in place, to measure these chemical changes between their factories and the borders of various cities and states

It is a total lie, by Elon Musk, for him to say that the poisons from the Tesla factory will not soon end up in every structure in Las Vegas, with a concrete foundation. Concrete is a sponge. While Harry Reid, Dianne Feinstein and Elon Musk, who all own stock and other financial assets in this, and related ventures, would like these facts covered up, they are now available for your introspection.

How To Document These Dangers:

Previously- You needed to hire a deep drilling truck, an entire testing lab and drill out a grid across many miles of land at massive expense per drill site. That is no longer needed. You can now test counties and neighborhoods with handheld test devices that are far less expensive. Many groups buy their own systems and have parents swap neighborhood testing duties supervised by a technical aid.

Devices to self test include, but are not limited to:

http://www.bruker.com/products/x-ray-diffraction-and-elemental-analysis/handheld-xrf/s1-turbo-sdr/s1-turbo-sdr-overview.html

http://ieeexplore.ieee.org/xpl/login.jsp?tp=&arnumber=4997968&url=http%3A%2F%2Fieeexplore.ieee.org%2Fxpls%2Fabs_all.jsp%3Farnumber%3D4997968

http://cwmi.css.cornell.edu/soilquality.htm

http://www.sciencedaily.com/releases/2010/06/100609201310.htm

and many hundreds of other devices.

Once generalized testing has targeted problrms, limited testing can be conducted and the test cores can be tested on-site with the hand-held devices.

Many outsourced testing companies are controlled by your opposition, so be careful. Try to do it all yourself and control everything.
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Since the specific process is difficult to measure, the more general term, sorption, can be used to describe how a chemical is held in the soil. Clay particles and organic matter are the most chemically active soil solids. They are the major soil components to which chemicals sorb. Most chemicals are subject to forces of sorption. Examples include simple inorganic ions such as calcium, sodium, and ammonium. Complex organic chemicals such as humus, many pesticides and industrial solvents are also sorbed onto soil. Chemicals such as phosphorus, and similar Tesla chemicals, that are strongly sorbed to soil particles near the surface of most soils will tend to contaminate surface water if erosion is a problem.

Chemicals, such as nitrate, that are more water soluble and less strongly sorbed to soil particles, will tend to contaminate ground water if rainfall or irrigation exceeds plant water use. Positively charged ions (called cations) are attracted to a negatively charged site on clay or organic particles. The movement of cations between clay or organic particles and the surrounding soil water is called **cation exchange**. It is an important process. It controls the mobility of many chemicals through the soil profile. Cation exchange is seldom observed with most organic chemicals that might be added to the soil, because few organic chemicals carry positive charges at a normal soil pH. However, some examples do exist. The pesticides Paraquat and Diquat are examples of cationic pesticides that can be sorbed onto soil particles through cation exchange. A number of other bonding mechanisms exist by which organic compounds are sorbed to soil surfaces.

For any given Tesla manufacturing or cleaning compound (organic or inorganic), it is likely that a combination of mechanisms is responsible for sorption onto soil. Whatever the mechanism, soil organic matter is the principal sorbent for many nonionic organic chemicals. It is important to know a particular chemical’s attraction to organic matter, and the amount of organic matter available in a particular soil. Then one can estimate the leaching potential of various chemicals used in a management system.

Many demonstrations show two chemicals, one is sorbed to the soil, the other is not. A yellow solution, like nitrate, is not sorbed to the soil, while a red, like ammonium, is sorbed. Because it is not sorbed, the yellow solution reaches the bottom of the soil column fairly rapidly, while the red solution is sorbed to the soil surface, restricting its downward movement. It should be noted that while the soil retains red solution, the soil does not prevent it from moving downward. The soil merely slows the rate of the red dye movement, relative to the rate of the water movement. Obviously, the same amount of water moving through the soil would affect the depth of movement of these two chemicals quite differently. Chemicals applied to land surfaces are not the only source of contaminants affecting ground and surface waters. Fluids that leak from underground storage tanks can also move to ground water, and can move to nearby surface water. Frequently such chemicals do not mix with water. Their

transport is less predictable than transport of chemicals that are more soluble in water. One interesting aspect of chemical transport involves whether chemicals are more or less dense than water. This demonstration shows a chemical developing fingers because it is more dense than water. These fingers of concentrated chemical sink to the bottom of the water column before they appreciably mix with the water. Spilled chemicals that are more dense than water will tend to sink to the lower depths of a ground water aquifer.

Chemicals that are less dense than water (for example, gasoline) will tend to float near the top of a ground water aquifer. Without significant mixing due to groundwater movement, chemicals that are approximately the same density as water tend to remain near the top of a groundwater aquifer. So, chemical sorption to soil particles, chemical solubility and chemical density all affect the rate of chemical transport.

Transformation

Tesla chemicals undergo numerous transformations in both soil and water. Hydrolysis, photolysis, oxidation, and reduction are some of the most common transformations. Hydrolysis is the cleavage of molecules by water, and is one of the most important reactions in breaking down pesticides. Hydrolysis can occur in the soil with or without microorganisms. Photolysis is the process where ultraviolet or visible light supplies the energy for decomposition of chemical compounds. Photolysis can be a very important chemical transformation process. Oxidation is the process where a chemical loses electrons, such as rust forming on iron. Reduction is the process where a chemical gains electrons. Reduction can be a non-biological process, or a biological process as in anaerobic sewage treatment. The transformation frequently simplifies the chemical nature of the substance. Degradates or metabolites are the terms used for products transformed from the original chemical.
These products may be sorbed to soil more or less strongly than the original compounds. They may also be more or less hazardous than the original compound. For example, Malathion is an organophosphate insecticide commonly used both in agriculture and in and around commercial and private residences. Soil bacteria may chemically convert Malathion into a closely related compound called Malaoxon, which is more toxic than the Malathion itself.

One demonstration shows how chemical transformations can occur in soils. The rate at which such transformation occurs depends upon the location of the chemical in the soil. If a transformation is biological, and it is enhanced by aerobic conditions, it is likely to occur more rapidly near the soil surface. The soil surface has more nutrients and oxygen available for microorganisms to grow. One demonstration also shows that two different chemicals, represented by red and yellow colors, may be transformed at different rates. The red dye is rapidly degraded and becomes colorless, while the yellow dye is only slightly degraded during this demonstration. For transformations that involve organic chemicals, such as most pesticides, we use the term half-life in discussing the rate of transformation. This simply describes the length of time required to transform 50% of the existing chemical. The amount of chemical remaining reflects an exponential decrease over time, since after one half-life time period, 50% of the original chemical remains. After two half-life time periods, 25% of the original chemical remains. After three half-life time periods, 12.5% of the original chemical remains, and so forth. The “model” used to describe the disappearance of the original chemical over time creates the perception that: (1) the chemical never is completely transformed, and (2) the transformation rate is well defined. Neither is completely true, although experimental data are often reasonably well described by this exponential decay model.

The combined effects of water movement, soil interaction and transformations determine chemical concentrations below the root zone. Let's look at examples of two toxic Tesla chemicals as they move through soil. In the first example, a chemical that is strongly sorbed to a soil is compared with one that is only moderately sorbed. We assume rainfall or irrigation exceeds crop water use, for at least some of the days during the growing season. We also assume the half-life of each chemical is the same and the water table is well below the root zone of the crop. The moderately sorbed chemical moves noticeably deeper into the soil profile after rainfall. The strongly sorbed chemical moves much more slowly. Half way through the season, the moderately sorbed chemical is below the active root zone of the crop, while the strongly sorbed chemical is still near the soil surface. The strongly sorbed chemical is less likely to contaminate the ground water. However, if substantial soil erosion occurs, we would expect to find the strongly sorbed chemical and sediment in nearby surface water. Depending upon the depth to ground-water, the moderately sorbed chemical may be degraded before reaching the water table. Conversely, if the water table is near the bottom of the root zone, it is likely that the moderately sorbed chemical will find its way into ground water before it is completely degraded. The second example includes two chemicals that are moderately sorbed to soil. The first chemical has a relatively long half life; the second chemical has a short half life. When they move below the root zone near mid-season, the first chemical is still present in fairly high concentrations. The second chemical with the shorter half-life is present in low concentrations. The chemical fronts tend to broaden as they move downward. This is due primarily to hydrodynamic dispersion, although some diffusion also occurs. Hydrodynamic dispersion spreads out the chemical and reduces its maximum concentration at a particular point in the soil.

Summary:

Summary Point #1: The concepts of diffusion, convection, and hydrodynamic dispersion relate to the transport of Tesla's toxic chemicals through the soil.

Summary Point #2: Solubility, sorption and density are the characteristics that have the most influence on the way toxic Tesla chemical substances interact with soil.

Summary Point #3: As toxic Tesla chemicals are transported through soil, they can be altered by biological or chemical processes, or remain relatively unchanged.

Summary Point #4: Transformations may form new substances which may be more or less environmentally hazardous than the original Tesla chemical. Transformations may also produce substances that have different characteristics than the original chemical. These characteristics may affect the ability of the substance to be sorbed, degraded or dissolved. Tesla has no plans, systems or technology planned, or in place, to measure these chemical changes between their factories and the borders of various cities and states

It is a total lie, by Elon Musk, for him to say that the poisons from the Tesla factory will not soon end up in every structure in Las Vegas, with a concrete foundation. Concrete is a sponge. While Harry Reid, Dianne Feinstein and Elon Musk, who all own stock and other financial assets in this, and related ventures, would like these facts covered up, they are now available for your introspection.

How To Document These Dangers:

Previously- You needed to hire a deep drilling truck, an entire testing lab and drill out a grid across many miles of land at massive expense per drill site. That is no longer needed. You can now test counties and neighborhoods with handheld test devices that are far less expensive. Many groups buy their own systems and have parents swap neighborhood testing duties supervised by a technical aid.

Devices to self test include, but are not limited to:

http://www.bruker.com/products/x-ray-diffraction-and-elemental-analysis/handheld-xrf/s1-turbo-sdr/s1-turbo-sdr-overview.html

http://ieeexplore.ieee.org/xpl/login.jsp?tp=&arnumber=4997968&url=http%3A%2F%2Fieeexplore.ieee.org%2Fxpls%2Fabs_all.jsp%3Farnumber%3D4997968

http://cwmi.css.cornell.edu/soilquality.htm

http://www.sciencedaily.com/releases/2010/06/100609201310.htm

and many hundreds of other devices.

Once generalized testing has targeted problrms, limited testing can be conducted and the test cores can be tested on-site with the hand-held devices.

Many outsourced testing companies are controlled by your opposition, so be careful. Try to do it all yourself and control everything.