Health Effects of construction materials and construction products

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Summary

Construction workers may be exposed to a number of toxic substances. The adverse health effects of the following materials are summarized: cement, mineral wool, asbestos, dust, wood dust, wood preservatives, epoxy resins, polyurethane products, diesel exhaust, asphalt/bitumen, paints and varnishes, organic solvents, concrete release agents. A concise bibliography on each substance is provided.

Samenvatting

Werknemers in de bouwnijverheid kunnen worden blootgesteld aan een scala van gevaarlijke stoffen. In dit artikel worden de gezondheidseffecten van de volgende groepen materialen/producten samengevat: cement, minerale wol, asbest, stof, houtstof, houtconserveermiddelen, epoxy harsen, polyurethaan producten, dieselmotoremissies, asfalt/bitumen, verf en lak, organische oplosmiddelen, betonlosmiddelen. Het artikel bevat een beknopte bibliografie van ieder onderwerp.

Introduction

General

Construction workers are occupationally exposed to a variety of substances such as natural and man-made mineral fibers, cement, quartz, various dusts, diesel exhaust, paints and solvents. Many of these substances are known to have adverse effects on workers' health. Examples of occupational diseases associated with construction work are mesothelioma and lung cancer from asbestos, nasal cancer caused by wood dust, respiratory effects from dusts and neurologic diseases from exposure to solvents or metals, skin diseases from exposure to cement or epoxy resins.

Objective

The objective of this paper is to provide background information for occupational health professionals on the effects that various construction products and materials may have on workers' health. The paper summarizes the health effects of a number of common products and presents a selection of recent literature reviewing the state of the art in research on chemical-induced occupational diseases in the construction industry. The bibliography is by no means comprehensive, but may be used as a starting point for further reading on the various topics.

Not covered in this paper are the health risks to workers from the use of waste and recycled materials (so-called secondary materials) and from working with contaminated soil.

Approach

A number of literature searches were performed using the product names (and synonyms) as search topics and limiting the searches to publications in Dutch or English, published after 1995, preferably reviews and relatively easily available via the Internet or Dutch libraries (only occasional exceptions have been made for language or publication year).

Databases searched were: Pubmed, Toxnet, Science citation index and Arbobibliotheek Nederland. All searches were performed in the period July-September 2006.

Complaints about hazardous substances

The Dutch Collective Labour Agreement for construction workers and related professions entitles every construction worker to a periodic medical examination every two to four years, depending on age. Part of this examination is a questionnaire about complaints related to work. Arbouw collects and processes the questionnaires anonymously, which gives a picture about complaints per job. The results are published in the 'Bedrijfstakatlas 2006' (Arbouw, 2006). Over 30,000 questionnaires were completed in 2006, whereas there were about 220,000 construction workers. The questions about nuisance from smoke, gases or vapours, chemical substances and dust are relevant for exposure to hazardous substances. These four items are shown Figures 1 to 4. Only the results for jobs with more complaints than average are shown.

On the average, 6.4% of the construction workers complain about gases and vapours, as shown in Figure 1. The scores for the road marker with 45.9% and for the asphalt road worker with 53.0% are notably high.

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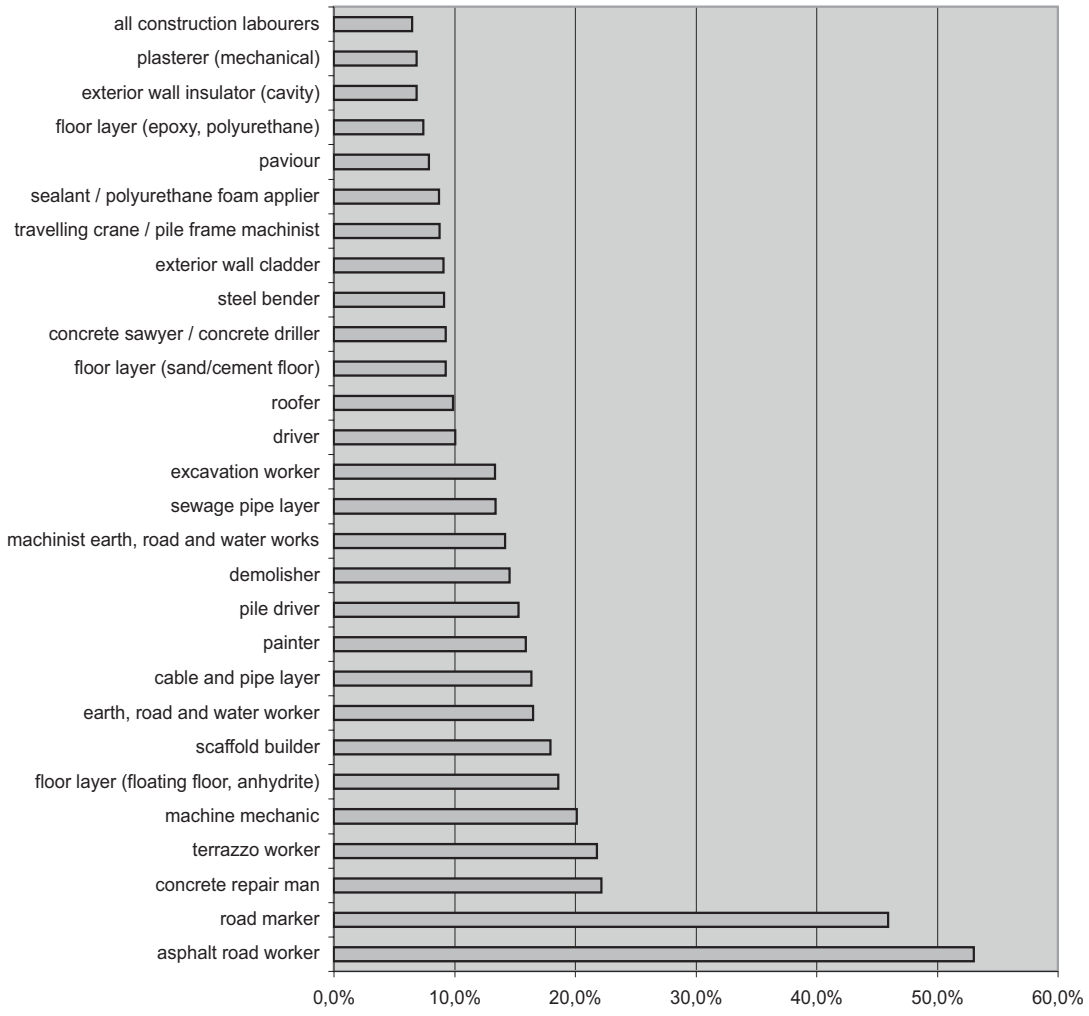

Figure 1: Complaints about vapours or gases

On the average, 4.6% of the construction workers complain about smoke, as shown in Figure 2. The scores for the road marker with 31.4% and the pile driver with 32.7% are notably high, as are to a lesser extent those for the asphalt road worker with 18.0% and the machine mechanic with 24.2%.

On the average 8.0% of the construction workers complain about chemicals, as shown in Figure 3. The scores for the sealant/polyurethane foam applier (29.5%), painter (34.3%), terrazzo worker (36.4%), epoxy/polyurethane floor layer (45.7%) and concrete repair man (61.8%) are notably high.

Figure 4 shows complaints about dust. Different from the other three items, these complaints are more evenly distributed among the jobs. The number of persons complaining varies from 18.5% for the plotter (not in the figure), to 83.4% for the polyurethane/epoxy floor layer, with an average of 55.4%. Seven jobs score over 70%. These are the floating floor layer, the traditional and mechanical plasterer, the demolisher, the tiler, the exterior wall insulator and the polyurethane/epoxy floor layer.

It must be kept in mind that dust may contain toxic substances. Examples are quartz dust form stony materials, wood dust, heavy metals in sanding dust especially from old paints.

Exposure to hazardous substances.

Arbouw has made a description of health hazards for the most common jobs in the construction industry. (Arbouw, 2005). Based on these job descriptions, the jobs with possible exposure to the hazardous materials and products are summarised. The result is shown in Table 1. Some minor corrections to the table are made, based on the experience of the authors. Therefore, in some cases the Table does not match to the job descriptions.

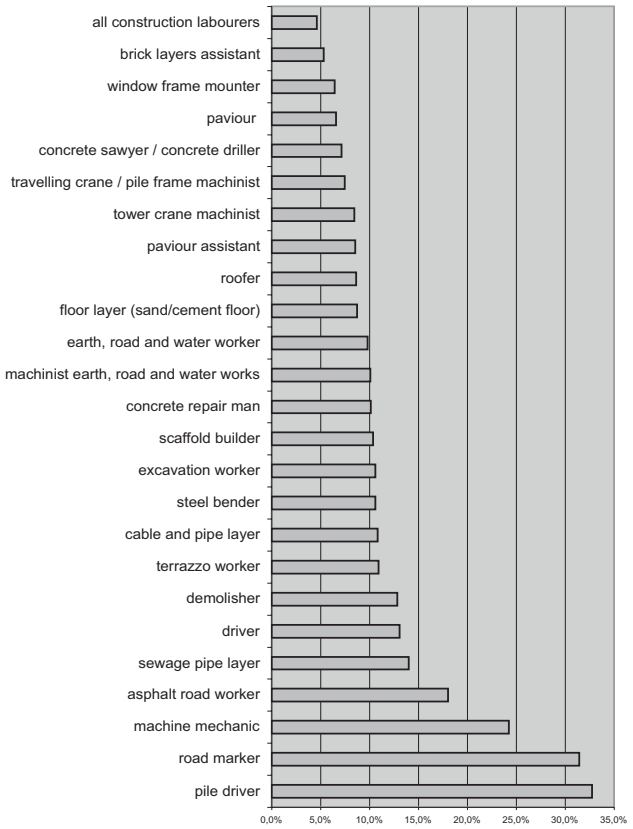

Figure 2: Complaints about

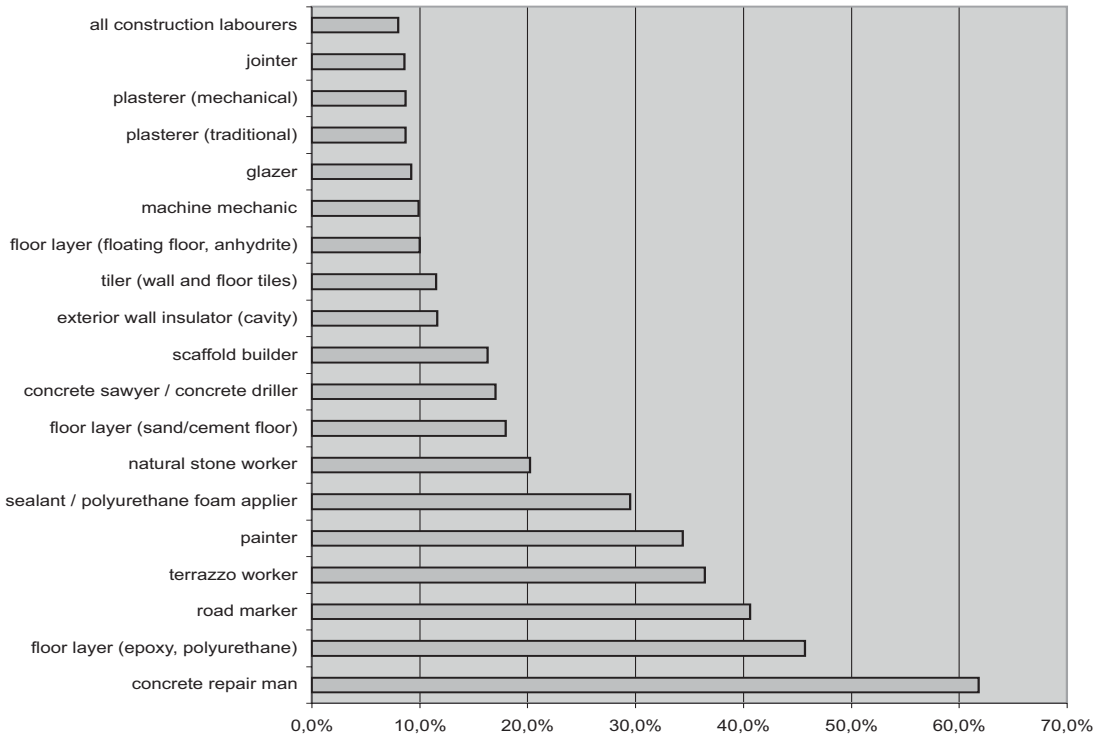

Figure 3: Complaints about chemicals

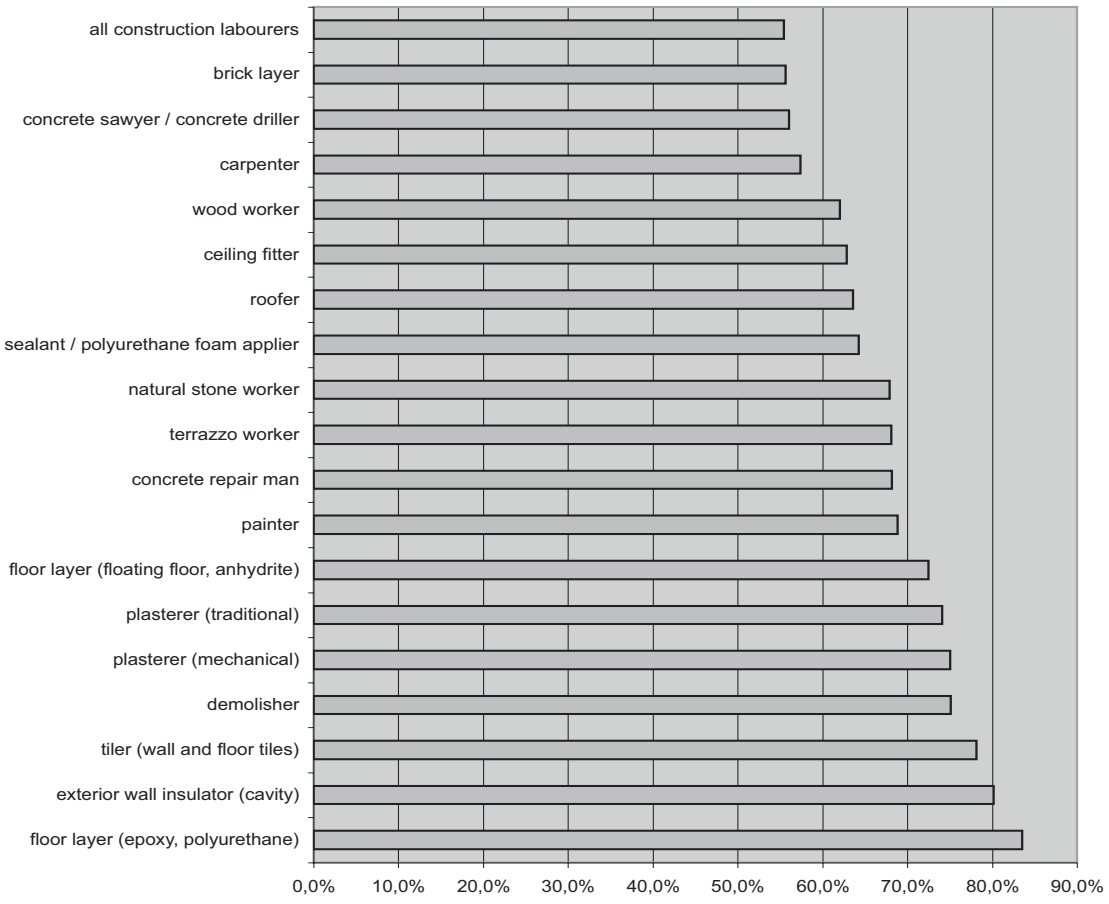

Figure 4: Complaints about dust

Cement

General

Cement is used in the construction industry as a bonding agent.

The Dutch cement industry produces mainly 4 types of cement: Portland cement, slag cement (usually 1/3 Portland cement and 2/3 slag), Portland fly ash cement (usually $^1/_4$ fly ash and $^3/_4$ Portland cement) and mortar (Portland cement, slaked lime, sand and water).

The raw materials for Portland cement are chalk, limestone, clay, shale containing silica and alumina and other materials such as iron, magnesium and acid sulfates.

The raw materials are blended and ground and the pulverized mixture is then heated in a kiln to form fused clinkers.

The cooled clinker may then be ground and mixed with gypsum and other additives which control the setting time and other properties of the mixture (Prodan and Bachofen, 1998; Winder and Carmody, 2002).

The chemical composition of Portland cement is given in Table 2. Portland cement may also contain cobalt and nickel compounds (Frias and de Rojas, 2002).

Health hazards of cement

Cement is one of the main causes of skin disease in the construction industry.

For the health effects of exposure to cement dust during transport, mixing and use see also the paragraph on Dust.

Skin diseases

Cement dermatitis is now thought to be caused by a combination of high alkalinity and abrasiveness of wet cement and contact sensitization by chromates and/or cobalt.

Lime (CaO, calcium oxide) is a major component of cement. In an exothermic reaction with water it forms calcium hydroxide. Wet cement is strongly alkaline (pH 12), has a irritating and caustic effect on the skin and contact may cause chemical burns. Apart from the high alkalinity, relevant factors for the development of cement burns are skin damage due to the abrasive properties of (added) particulates and skin penetration of alkaline cement (DECOS, 2006; Poupon, et al., 2005; Spoo and Elsner, 2001).

The role of hexavalent chromium as a contact sensitizer in cement dermatosis has been known and studied since the 1950s (Denton, et al., 1954; Pirila, 1954). The chromium content (Cr[III] and Cr[VI]) of the raw materials for the

Table 1: exposure to construction products per job

	cement	asphalt/ bitumen	wood dust	wood pre-servatives	mineral wool	asbestos	epoxies	polyure-thanes	concrete release ag.	silica	dust	paints	solvents	diesel exhaust
asphalt road worker		x								x	x		x	x
brick layer	x				x					x	x			
brick layer assistant	x				x					x	x			
cable and pipe layer						x				x	x		x	x
carpenter			x	x	x		x	x	x		x	x	x	
ceiling fitter					x					x	x			
concrete repair man	x						x			x	x		x	
concrete sawyer / concrete driller										x	x			
concrete worker	x								x					
demolisher		x	x	x	x	x				x	x			x
driver														x
earth, road and water worker														x
excavation worker														x
exterior wall insulator (cavity)					x			x		x	x		x	
exterior wall cladder					x					x	x		x	
floor layer (epoxy, polyurethane)							x	x		x	x		x	
floor layer (floating floor)										x	x			
floor layer (sand/cement floor)										x	x			
glazer													x	
jointer	x						x			x	x		x	
machine mechanic													x	x
machinist earth, road and water works											x		x	
natural stone worker	x						x			x	x			x
painter			x	x			x	x			x	x	x	
paviour										x	x			x
paviour assistant										x	x			x
pile driver														x
plasterer (mechanical)										x	x		x	
plasterer (traditional)										x	x			
road marker							x				x		x	x
roofer		x			x					x	x			
sealant / polyurethane foam applier							x	x		x	x		x	
sewage pipe layer	x	x					x	x		x			x	x
terrazzo worker	x						x			x	x		x	
tiler (wall and floor tiles)	x						x			x	x		x	
travelling crane / pile frame machinist													x	
window frame mounter			x		x		x			x	x		x	
wood worker			x	x			x	x					x	

Table 2: The typical composition of the starting materials for portland cement manufacture (Winder and Carmody, 2002).

Ingredient	**Formula**	**Percentage**
Calcium oxide (lime)	CaO	64
Silicium dioxide (silica)	SiO_2	21
Aluminium oxide	Al_2O_3	5.8
Iron oxide	FeO_3	2.9
Magnesium oxide	MgO	2.5
Sulfur dioxide	SO_3	1.7
Hexavalent chromium	Cr[VI]	0.002
Alkali oxides		1.4

production of clinker is usually low. During the processing of the raw materials in the kiln Cr[III] may be oxidized to Cr[VI]. But more important sources for chromates in cement may be vulcanic rock, abrasion of the refractory lining of the kiln, the steel balls in grinding mills and the tools used for grinding the raw materials and the clinker (Prodan and Bachofen, 1998; Winder and Carmody, 2002). The Cr[VI] content in the finished portland cement product may vary from 0.2 to 40 ppm, depending on the origin of the cement (Kersting, et al., 2002; VDZ, 1999).

A strong association between cobalt and chromate allergy has been found in construction workers (Uter, et al., 2004). Allergic contact dermatitis caused by cobalt is commonly regarded as due to cosensitization to primary chromate sensitization (Bock, et al., 2003; Frias and de Rojas, 2002, Goon and Goh, 2005; Uter, et al., 2004).

To reduce the risk of chromate dermatitis, ferrous sulfate is sometimes added to cement. It reduces Cr[VI] to Cr[III] which is not a sensitizer. Studies in Scandinavian countries report a decrease in the prevalence of chromate dermatitis after the addition of ferrous sulfate to cement (Johansen, et al., 2000; Roto, et al., 1996).

In countries were ferrous sulfate was not added to cement during the periods that were studied no significant changes were found in the incidence of chromate allergies (Bock, et al., 2003; Dickel, et al., 2002; Dickel, et al., 2001; Katsarou-Katsari, et al., 2003; Olsavszky, et al., 1998).

A reduction in Cr[VI] concentration in cement may also be obtained by technological changes such as substituting part of the clinker with slag or fly ash and substituting the bricks for kiln lining and by increased use of precast cement products (Goh and Gan, 1996; Winder and Carmody, 2002).

In the European Union the use of cement or cement products containing more than 2 ppm chromium VI is restricted (EC, 2003). This directive has been implemented in the Netherlands in 2004 (Staatsblad, 2004a). The 2 ppm limit only applies to cement in bags. For other forms of cement, there are no restrictions.

Asphalt/ bitumen

General

Asphalt is the residue produced from the non-destructive distillation of crude oil during petroleum refining. Outside the USA, asphalt is more commonly referred to as bitumen, and a mixture of bitumen with mineral matter is referred to as asphalt. In this paper, asphalt is used to refer to the residue both with and without the addition of mineral matter.

Asphalt is a complex mixture of chemical compounds of high molecular weight, predominantly asphaltenes, cyclic hydrocarbons (aromatic or naphthenic) and a lesser quantity of saturated components of low chemical reactivity. The chemical composition depends both on the original crude oil and on the process used during refining. Most asphalt is used in road paving, but it is also used in roofing, waterproofing and insulation and as in ingredient in paints and varnishes (Finklea, 1998).

When asphalts are heated, vapours are released; as these vapours cool, they condense. As such, these vapours are enriched in the more volatile components present in the asphalt and would be expected to be chemically and potentially toxicologically distinct from the parent material. Asphalt fumes are the cloud of small particles created by condensation from the gaseous state after volatilization of asphalt. However, because the components in the vapour do not condense all at once, workers are exposed not only to asphalt fumes but also to vapours through inhalation. The physical nature of the fumes and vapours has not been well characterized (WHO, 2004). Dermal exposure through contact with asphalt fumes that condensed on tools, equipment, skin and clothing can be important (McClean, et al., 2004)

Health hazards of asphalt/bitumen

Acute effects of exposure to asphalt fumes that have been reported include irritation of the eyes and the mucous membranes of the upper respiratory tract (nasal and throat irritation), lower respiratory tract symptoms (coughing, wheezing, shortness of breath), bronchitis, skin irritation and rashes (Burstyn, et al., 2003a; Randem, et al., 2004b; WHO, 2004). Given the presence of confounding co-exposures (i.e., diesel fuel exhaust products, coal tar, fibreglass) and environmental conditions (wind, heat and humidity, ultraviolet radiation), the extent to which asphalt fumes may be associated with these effects on the skin is unclear (WHO, 2004).

Some indications for association of bitumen exposure with mortality for obstructive lung diseases (Burstyn, et al., 2003a) and ischemic heart disease (Burstyn, et al., 2005) have been found.

Evidence for carcinogenicity of asphalt to humans is inconclusive, also because of confounding factors like smoking and exposure to other carcinogens like coal tar or diesel exhaust (Armstrong, et al., 2004; Binet, et al., 2002; Boffetta and Burstyn, 2003; Boffetta, et al., 2003a; Boffetta, et al., 2003b;

Burstyn, 2001; Burstyn, et al., 2003b; Burstyn, et al., 2003c; Kauppinen, et al., 2003; NIOSH, 2000; Randem, et al., 2004a; Shaham, et al., 2003; Swaen, 2004). In 1987, the IARC concluded that there is inadequate evidence for the carcinogenicity of bitumens to humans (IARC, 1985; IARC, 1987).

In a recent evaluation the World Health Organzation concluded as follows:

"Studying the possible health effects attributed to chemical mixtures, including resulting fumes and vapours, is complex. Despite the uncertainties, limitations, and mixed study results, what is clear is that asphalt fume condensates produce malignant skin tumours in mice; and that, when exposed to airborne concentrations of asphalt or asphalt fumes and vapours, workers report symptoms of irritation of the eyes, nose, and throat and, in some, lower airway changes and demonstrate metabolism of the chemical constituents of asphalt fumes and vapours. Taken as a whole, these results suggest that effects do occur in mammalian systems and that the limitations or uncertainties should not preclude taking steps to manage human exposures.

Under various performance specifications, it is likely that asphalt fumes and paints contain carcinogenic substances" (WHO, 2004).

Wood dust

General

Exposure to wood dust in the construction industry may arise from different activities (e.g. sawing, planing, sanding, drilling) (Spee, et al., 2006b). The source of the dust may be different species of hardwood or softwood, plywood, particle board, fiber board or wood chemically treated with glues or preservatives (Kauppinen, et al., 2006; McCann, 1998b). Exposure is often to a mixture of wood dusts.

Most exposure data are from the wood and furniture industries (Kauppinen, et al., 2006). In a recent study in The Netherlands the long-term average exposure to wood dust among carpenters at construction sites was estimated to be 3.3 mg/ m^3 (Spee, et al., 2006b). The current exposure limit for dust from hard wood is 2 mg/m^3. For dust from soft wood, no exposure limit is established (Staatscourant, 2006).

Health hazards of wood dust

Wood dust may cause nasal cancer and have a number of non-malignant respiratory and dermatological effects (DECOS, 2000; Demers, et al., 1997; IARC, 1995; McCann, 1998b).

Cancer

Very high relative risks of sino-nasal cancer, particularly sinonasal adenocarcinoma, have been observed among workers exposed to high levels of dust from hardwoods, such as beech, oak and mahogany. The evidence for softwood dust is less conclusive, and smaller excess risks have been observed (DECOS, 2000; Demers, 1998; Demers, et al., 1997; IARC, 1995; McCann, 1998b). Also, some of the chemicals which may be present in treated wood (formaldehyde, pentachlorophenol and tetrachlorophenol pesticides, creosote) are known carcinogenics (Demers, 1998; Huff, 2001).

Non-malignant respiratory effects and dermatoses

Some woods contain chemicals that are irritants and may cause non-specific irritation of the respiratory tract. Other species may cause conjunctivitis-rhinitis, allergic contact dermatitis, asthma, chronic bronchitis and other pulmonary function abnormalities (Demers, et al., 1997; Estlander, et al., 2001; Hubbard, 2001; McCann, 1998b; Schlunssen, et al., 2004; Schlunssen, et al., 2002), although some of the epidemiological evidence has been criticized (Williams, 2005). Some of the effects may be caused by wood constituents (e.g. alkaloids, quinones, terpenes, cumarins, glycosides, phenols), industrial additions (wood preservatives, pesticides), molds and bacteria (Demers, et al., 1997; McCann, 1998b).

Upper- and lower-respiratory effects have been associated with both softwood and hardwood tree species from both temperate and tropical climates. For example, occupational asthma has been found to be associated with exposure to dust from African maple, African zebra, ash, California redwood, cedar of Lebanon, Central American walnut, Eastern white cedar, ebony, iroko, mahogany, oak, ramin and Western red cedar as well as other tree species (Demers, 1998).

A short list of wood species and their effects on health is given in an Arbouw publication on wood dust (Arbouw, 2002), an extended list may be found in the ILO Encyclopaedia (Warshaw, 1998).

Wood preservatives

General

Wood preservation is the treatment of wood with chemicals to prevent decay from the action of bacteria, fungi or insects. Most wood preservation is done by specialized companies. Methods used are vacuum and pressure methods, spraying, painting, injecting, diffusion, immersion. The use of some wood preservatives has been prohibited or restricted (e.g. arsenic containing preservatives), but construction workers may still be exposed to wood that has been treated with these chemicals.

Health hazards of wood preservatives

Copper preservatives

Copper preservatives are copper salts in combination with other metal salts, such as chromium and arsenic salts (CCA), chromium and boron (CCB) or chromium and fluor (CCF) (VROM, 2006). The use of CCA treated wood has been severely restricted (Staatsblad, 2004b).

Copper compounds

Except for occasional acute incidents of copper poisoning, few effects are noted in normal human populations. 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 (ATSDR, 2004; ILO, 1998b; WHO, 1998b).

Chromium compounds
Chromium compounds may induce skin and mucous membrane irritation or corrosion, allergic skin reactions or skin ulcerations. Chromium (VI) compounds are carcinogenic (ATSDR, 2000; EPA, 1998b; EPA, 1998c; IARC, 1990; ILO, 1998b).
Arsenic compounds
Arsenic compounds may cause vascular disorders, leucocytopenia, anaemia, eczema, mucous membrane lesions and peripheral neuropathy (ATSDR, 2005; ILO, 1998b; WHO, 2001). Arsenic is also considered a carcinogen (IARC, 2004).
Boron compounds
Workers exposed to borax (sodium borate) dust have reported chronic productive cough, and, in those who have experienced long exposures, obstructive abnormalities have been detected, though it is unclear whether these are related to exposure (ATSDR, 1992; ILO, 1998c; WHO, 1998a).
Fluorides
Fluorine and fluorides have a strongly irritating effect on mucous membranes of the eyes and respiratory tract. Chronic exposure may cause bronchitis and alterations in bone density and fragility (ATSDR, 2003).

Coal tar distillates
Creosote and carbolineum are distillation products of coal tar. The major chemicals in coal tar creosote that can cause harmful health effects are polycyclic aromatic hydrocarbons (PAHs), phenol, and cresols. Carbolineum is derived from creosote by incorporation of components with a lower boiling point to obtain a product with a lower viscosity. About 300 chemicals have been identified in coal tar creosote, but as many as 10,000 other chemicals may be present in this mixture (ATSDR, 2002)
The use of carbolineum has been prohibited in The Netherlands since 2001 and the use of creosote is restricted (VROM, 2006).
Reports describing poisoning in workers exposed to coal tar creosote, or in people who accidentally or intentionally ate coal tar creosote prove that these chemicals can be harmful. These reports indicate that brief exposure to creosote may result in a rash or severe irritation of the skin, chemical burns of the surfaces of the eye, convulsions and mental confusion, kidney or liver problems, unconsciousness, or even death. Longer exposure to lower levels by direct contact with the skin or by exposure to the vapours can result in damage to the cornea, and skin damage such as reddening, blistering, or peeling. Creosote induces phototoxicity of the skin, so that exposure to the sun exacerbates its irritant effects. Longer exposures to the vapours of the creosotes can also cause irritation of the respiratory tract (ATSDR, 2002).
Skin cancer and cancer of the scrotum have also resulted from long exposure to low levels of these chemical mixtures, especially through direct contact with the skin during wood treatment or manufacture of coal tar creosote-treated products (ATSDR, 2002). Creosotes have been classified by IARC as probably carcinogenic to humans (Group 2A) (IARC, 1985; IARC, 2006).

Mineral wools

General
Mineral wools (glass wool, rock wool and slag wool) are widely used in the construction industry as thermal and acoustical insulation materials. They belong to a group of synthetic vitreous fibers (SVFs), also known as man-made vitreous fibers (MMVFs), man-made mineral fibers (MMMFs). A categorization scheme for SVFs and a description of different types of vitreous fibers is given in Moore et al. (Moore, et al., 2002). Glass fiber is manufactured from silicon dioxide and various concentrations of stabilizers and modifiers. Most glass wool is produced through use of a rotary process resulting in 3 to 15 µm average diameter discontinuous fibers with variations to 1 µm or less in diameter. The glass wool fibers are bound together, most commonly with phenolic formaldehyde resins, and then put through a heat-curing polymerization process. Other agents, including lubricants and wetting agents, may also be added, depending on the production process. Slag wool and rock wool production involves melting and fibrizing slag from metallic ore and igneous rock, respectively. The production process includes a dish shaped wheel and wheel centrifuge process. It produces 3.5 to 7 µm average diameter discontinuous fibers whose size may range well into the respirable range. Mineral wool can be manufactured with or without binder, depending on end-use applications (Ross and Lockey, 1998).

Health hazards of mineral wools
Most research on health effects has concentrated on possible carcinogenic and genotoxic effects, but irritating effects on eyes, skin and upper respiratory tract have also been reported.
Irritation
Skin, eye, and upper and lower respiratory tract irritation can occur and depends on exposure levels and job duties. Skin irritation has been the most common health effect noted. It is caused by mechanical trauma to the skin from fibers greater than 4 to 5 µm in diameter (Jolanki, et al., 2002; Petersen and Sabroe, 1991; Ross and Lockey, 1998; Stam-Westerveld, et al., 1994). It can be prevented with appropriate environmental control measures including avoiding direct skin contact with the fibers, wearing loose fitting, long-sleeved clothing, and washing work clothing separately. Upper and lower respiratory symptoms can occur in dusty situations, particularly in MMVF product fabrication and end-use applications and in residential settings when MMVFs are not handled, installed or repaired correctly (Albin, et al., 1998; Petersen and Sabroe, 1991; Ross and Lockey, 1998).
Carcinogenicity
SVF products can release respirable fibers during production and use which show structural and morphological similarities with asbestos, a group of naturally occurring fibers which are considered to be proven carcinogens (IARC, 1977). This has given rise to numerous studies and reviews on the carcinogenic potential of SVFs (Berrigan, 2002; Greim, 2004; Maxim, et al., 2003; Moore, et al., 2002; Moore, et al., 2001; Wilson, et al., 1999).

SVFs are now thought to have less potential for adverse health effects than naturally occurring mineral fibers (such as asbestos) because of their amorphous state and the lower biopersistence of at least some categories of SVFs.
In 1987, the International Agency for Research on Cancer (IARC) classified glass wool, rock wool, slag wool and ceramic fibers as possible human carcinogens in group 2B (IARC, 1988b). In 2002 IARC re-evaluated the evidence for carcinogenicity of SVFs because there had been substantial improvements in the quality of the available epidemiological information. In the 2002 re-evaluation, insulation glass wool, continuous glass filament, rock (stone) wool and slag wool were evaluated as *not classifiable as to their carcinogenicity to humans (group 3)* (Baan and Grosse, 2004; Rossiter, 2002).
The European Union uses different criteria than the IARC for the categorization of SVFs (EC, 1997) and differentiates the mineral wools from refractory ceramic fibers and special purpose fibers (Moore, et al., 2002).
Recent studies stress the importance of biopersistence for the assessment of fiber toxicity (Donaldson and Tran, 2002; Donaldson and Tran, 2004; Greim, 2004; Guldberg, et al., 2000; Hesterberg and Hart, 2001; Moolgavkar, et al., 2000; Moolgavkar, et al., 2001; Moore, et al., 2002; Moore, et al., 2001).
Recommendations for further research on SVF toxicology and for the development of occupational exposure limits are formulated in the following reviews: (Greim, 2004; Maxim, et al., 2003; Ziegler-Skylakakis, 2004). The current Dutch occupational exposure limit for mineral wools is 2 fibers per cm^3.
During installation or removal of mineral wool products at a number of Dutch workplaces the exposure level remained below the exposure limit of 2 fibers per cm^3 (Zock, et al., 1999).

Asbestos

General
Asbestos is a generic name for a group of naturally occurring fibrous minerals. Asbestos is highly resistant to chemicals and heat and because of these properties it has been used in a wide variety of industries and products (Harmsma, 2006). Asbestos is subdivided into two groups: the serpentine group and the amphiboles, which differ in crystalline structure, in chemical and surface characteristics and in the physical characteristics of their fibers. Chrysotile (white asbestos), which belongs to the serpentine group, is by far the most commonly used form. The amphiboles amosite (brown asbestos) and crocidolite (blue asbestos) have been used to a much lower extent.

In The Netherlands somewhat over 1.1 million tonnes of asbestos fibers have been used in the manufacturing of asbestos containing products, including debris produced during the production process, see Table 3 (Harmsma, 2006).

More than 50% of asbestos used in the construction industry is bound to cement in asbestos-cement materials for roofing, pipes, tiles, sheeting and siding and other products. About 35% has been used in asbestos cardboard. Other applications are in sprayed asbestos, insulation materials, fire resistant board, floor tiles, bitumen, paints (Harmsma, 2006).
From the 1930s on it was known that asbestos exposure could lead to asbestosis.

Also from that era are some communications of a possible relationship with lung cancer. (IARC, 1977). Conclusive research on the relationship between asbestos and lung cancer came from Doll (Doll, 1955a; Doll, 1955b).
In 1978 the use of crocidolite and in 1993 the use of all other forms of asbestos has been prohibited. Since January 2005 the manufacturing, use, import and export is prohibited in all member states of the European Union (EC 1099a). But the ubiquitous presence of asbestos and asbestos products in for instance buildings, road pavements, pipes and soil will remain a health hazard for many years to come.

Health hazards of asbestos
Exposure to asbestos may lead to a number of asbestos-related diseases which may occur separately or in combination. It should be kept in mind that chrysotile and amphiboles differ in structure, chemical composition and biopersistance and differ in potency for causing lung disease (ILO, 1998a).
Malignant mesothelioma
Malignant mesothelioma is an aggressive, fatal neoplasm originating from mesothelial cells that form the serosal lining of the pleural, peritoneal and pericardial cavities, in decreasing order of frequency (Bielefeldt-Ohmann, et al., 1996; Carbone, et al., 2002). There is a direct relation between

Table 3. Mass balance for asbestos fibres in The Netherlands (in tonnes)

Fibre type	Number in tonnes	Balance in tonnes
Imported asbestos fibres	776.263	
Exported asbestos fibres	6.242	
Balance in The Netherlands		770.021
Asbestos fibres in imported products	608.511	
Asbestos fibres in exported products	252.883	
Balance import/export asbestos products		355.628
Asbestos fibres in The Netherlands		1.125.649

asbestos exposure and the occurrence of mesothelioma: for about 80% of the people who develop mesothelioma exposure to asbestos in the past can be demonstrated. The long incubation period between first exposure to asbestos and onset of disease (20-30 years, although shorter latency periods have also been described) has led to an increasing incidence of mesothelioma worldwide (Gezondheidsraad, 1998; Kazan-Allen, 2005; Robinson, et al., 2005; Stewart, et al., 2004).

Asbestosis

Asbestosis is a pneumoconiosis, a disease of the lungs caused by the inhalation of asbestos fibers. Asbestos fibers cause a fibrogenic reaction and scarring of lung tissue. Fibrosis of the lung tissue leads to loss of elasticity and diminishes the capacity for oxygen uptake (Cugell and Kamp, 2004; Gezondheidsraad, 1999; Henderson, et al., 2004; Niklinski, et al., 2004).

Lung cancer

Whereas malignant mesothelioma is almost always attributable to exposure to asbestos, the relationship between asbestos exposure and lung cancer is less clear. Cigarette smoking is by far the most important cause of lung cancer and most possibly asbestos-related lung cancers occur in smokers (Gezondheidsraad, 2005; Henderson, et al., 2004). The interaction between asbestos exposure and smoking has been extensively studied and epidemiologic studies have established that tobacco smoke and asbestos exposures synergistically interact to enhance lung cancer risk (Berry and Liddell, 2004; Case, 2006; Henderson, et al., 2004; Lee, 2001; Liddell, 2001; Liddell, 2002; Nelson and Kelsey, 2002; Reid, et al., 2006).

Pleural plaques

Pleural plaques are local areas of fibrosis of the parietal pleura, usually bilateral and sometimes calcified. Pleural plaques are considered by some as benign markers of prior exposure, whereas other believe they cause functional impairments and are indicators for future malignancy (Cugell and Kamp, 2004).

Pleural effusion

Benign pleural effusions due to asbestos exposure vary from a completely asymptomatic event to an active, inflammatory pleuritis. The symptoms do not differ from those of other forms of acute pleuritis. Asbestos pleural effusions have no prognostic implications for development of pleural plaques or mesothelioma (Cugell and Kamp, 2004).

Epoxy resins

General

Epoxy resins are formed from polymerisation and crosslinking of base resin and curing agents. Most epoxy resins are made using diglycidyl ethers of bisphenol A and epichlorohydrin, commonly referred to as DGEBA (Diglycidylether of Bisphenol A) as the base resin. Varying the proportions of bisphenol A and epichlorohydrin during manufacturing produces low- and high-molecular weight resins (Tavakoli, 2003).

The epoxy resin system usually consists of the following constituents:

- Base resin;
- Curing agents;
- Reactive diluents;
- Solvents;
- Plasticizers, flexibilizers and toughening agents;
- Fillers including pigments and reinforcing fibers;

In the construction industry epoxy resins are used in a variety of applications such as paints, coatings, floors, sealing, adhesives, binders (ILO, 1998c; Spee, et al., 2006a; Tavakoli, 2003).

The epoxy resins commonly used in construction are of low molecular weight, which is also generally associated with increased volatility and increased risk for inhalation.

Health hazards of epoxy resins

In general, the toxicity of a resin system is a complicated interplay between the individual toxicities of its various component ingredients. After the curing process the reactants will disappear, but after 24 hours some 30% of unreacted ingredients may still be present (M. van den Beld, personal communication).

Base Epoxy Resins

DGEBA epoxy resins are known sensitizers of the skin and those with the highest sensitization potential are those of lower relative molecular weight (average molecular weight of <1000, with a high amount of MW = 340 oligomer). The main sensitizer appears to be epoxy resin oligomer of MW=340 (ILO, 1998c; Rademaker, 2000; Tavakoli, 2003). Chemically, epoxies are characterized by the epoxide-group, which is very reactive towards aminogroups in the curing agents. Unfortunately skin proteins are also amino compounds and that is the reason why epoxies are such potent skin sensitizers (Spee, et al., 2006a).

Of the constituents of DGEBA epichlorohydrin has been shown to cause allergic dermatitis among epoxy resin plant workers. Contact dermatitis due to bisphenol A has also been reported, but outside epoxy resin plants occupational dermatitis due to bisphenol A and epichlorohydrin is rather uncommon (Tavakoli, 2003).

Epichlorohydrin is has been classified as probably carcinogenic to humans (IARC group 2A) (IARC, 1999). Small residues of the monomer epichlorohydrin remain in epoxy resins (usually < 0.1%). The European manufacturers of epoxy resins have agreed upon additional voluntary limit values for the monomer-content (Spee, et al., 2006a).

Curing agents

Common curing agents used in construction are:

- Aliphatic and alicyclic amines such as ethylenediamine, diethylenetriamine, N-amino ethylpiperazine, triethyltetramine and isophorondiamine. These amines are strong bases and are severely irritating to the skin, eyes and respiratory tract. Many are also skin and respiratory tract sensitizers (Tavakoli, 2003).
- Aromatic amines such as 4,4-methylenedianiline (MDA) are not strong irritants, but several are skin sensitizers. Certain aromatic amines may absorb through the skin and

cause damage to organs such as the liver and interfere with the blood's ability to carry oxygen. Some aromatic amine curing agents are known to cause cancer in laboratory animals (IARC, 1989a; Tavakoli, 2003).

Reactive diluents

The viscosity of epoxy resins may be reduced by the addition of reactive diluents, mostly glycidyl ethers. The type and amount of diluent will affect the viscosity and curing characteristics of the epoxy system.

Reactive diluents have been found to cause contact allergy. Contact allergy to reactive diluents without contact allergy to epoxy resins is also possible. Glycidyl ethers are more volatile than DGEBA resins, which may increase the exposure risk (Tavakoli, 2003).

Pigments, solvents, fillers

Pigments added to the epoxy resin system may contain chromates, lead or cadmium.

Fillers may contain asbestos or quartz, see the headings **asbestos** and **silica**.

Polyurethane products

General

Polyurethane or PUR is produced by the reaction between a polyol and a diisocyanate. In the presence of a blowing agent, this reaction will produce a foamed material having excellent thermal insulating properties. Other additives can be used to improve the fire resistance, stability and other properties of the polyurethane products. Polyurethanes are used in paints, synthetic leathers, coatings, adhesives, flooring, insulation, etc. Methylene bis(4-phenylisocyanate) (MDI) and 2,4,-toluene diisocyanate (TDI) are the most widely used isocyanates. Both compounds have about the same toxic effects, but MDI is less volatile and therefore poses a lower risk for inhalation than TDI (EPA, 1998a; WHO, 2000). Unmodified polyurethane resin can be formulated in one- or two-component systems. Two-component systems harden when a diisocyanate curing agent, e.g. an amine, is added to prepolymerized polyurethane resin before application.

PUR foam is usually delivered in one-component pressurized containers in which only MDI is used (EPA, 1998a; WHO, 2000).

Health hazards of PUR

Isocyanates

Isocyanates are irritating to the skin and the mucous membranes, the skin conditions ranging from localized itching to more or less widespread eczema (EPA, 1998a; ILO, 1998c; WHO, 2000). The association between asthma and exposure to diisocyanates has been studied extensively. The isocyanate literature clearly identifies asthma as the principal health concern for polyurethane workers, but there is insufficient information to describe the concentration-response relationship and the mechanism of action (EPA, 1998a; WHO, 2000). There are two types of occupational asthma, irritant-induced and sensitizer-induced asthma, both of which can be attributable to diisocyanates. Irritant-induced asthma has no latent period and begins within 24 hours of a spill or other high exposure to diisocynates (Tarlo and Liss, 2002). In the case of sensitizer-induced occupational asthma there is a latent period of exposure and once sensitized, individuals may react with severe symptoms of asthma after exposure to very low levels of isocyanates (sometimes below 0.02 ppm) if they have become sensitized (Castranova, et al., 2002; Diller, 2002; Gijsbers, et al., 2001; Liu and Wisnewski, 2003; Redlich and Karol, 2002; Tarlo and Liss, 2002).

Even at very low airborne isocyanate concentrations, uptake of isocyanate is demonstrated through biological monitoring. A possible explanation is uptake of isocyanates through the skin (Creely, et al., 2006). Dermal exposure is an important exposure route for isocyanates (Pronk, et al., 2006). In vitro studies suggests that skin absorption may contribute substantially to the uptake of isocyanates (Bello, et al., 2006). In mice, respiratory allergy is developed after skin application of isocyanates (Ban, et al., 2006).

MDI has been classified by IARC as not classifiable as to its carcinogenicity to humans (Group 3), TDI as possibly carcinogenic to humans (Group 2B) (IARC, 1999)

Blowing agents

Formerly, chlorofluorhydrocarbons (CFCs) and hydrochloroflurocarbons (HCFCs) were used as blowing agents in the production of PUR foam. Both types are greenhouse gases and the use has been prohibited. Dimethylether, aliphatic hydrocarbons (pentane, cyclopentane and butane/pentane mixtures) and hydrofluorocarbons (HFCs) are now used as blowing agents (Eck, 2002; EPA, 2006). The aliphatic hydrocarbons used are mildly toxic central nervous system depressants and cause skin or mucous membrane irritation only at rather high exposures, see Table 104.90 in (ILO, 1998c). Long time exposure to dimethylether may lead to chronic toxic encephalopathy (see the heading **organic solvents**). Many HFCs are not highly toxic to human at air exposure levels up to few percent or even over 10% (Tsai, 2005).

Concrete release agents

General

Release agents are used in concreting to contribute easy separating of forms from hardened concrete. The releasing actions of these agents can be classified into two basic categories: physical or chemical actions. Most of the well formulated release agents have both these characteristic properties.

Physical type release agents form a hydrophobic layer between the hydrophilic surfaces of the concrete and the formwork.

Chemical release is based on the saponification reaction between Ca-ions of fresh concrete and fatty acids of release agent. In this reaction a thin layer of insoluble calcium salts is formed in the concrete-form-interface, which contributes to easy release.

Conventional concrete release agents used in the construction industry are based on mineral oils (from petroleum). Mineral oil based release agents are mostly non-biodegradable and emit volatile organic compounds (VOCs) into the atmosphere. Use of vegetable oil based agents is increasing because they are less hazardous to health and environment

(Broekhuizen, 2001; de Brito, et al., 2000; Terwoert, 1999). Formerly, mineral oils used in concrete release were low-grade products with high aromatic and poly-aromatic content. Nowadays the mineral oils are ususally well refined and no longer contain aromatic hydrocarbons, or only in very low concentrations (Terwoert, 1999).

Health hazards of concrete release agents

Although the quality of the oils has improved considerably, application of the release agents is still not without health risk. A large number of release agents are mineral oil based and may contain relatively large amounts of solvents (see the heading **solvents**). In the majority of the cases release agents are atomised, which results in the formation of oil droplets in the air and evaporation of the solvents in the release agents. Furthermore the release agents may contain additives which may result in allergies (Terwoert, 1999).

Respiratory effects

Both mineral oil based and vegetable oil based release agents are usually sprayed on the mould, which results in the formation of oil droplets in the work atmosphere. Inhalation of oil droplets can lead to infections of the airways and lungs. When the droplets enter deep into the lungs it can result in a chemical pneumonia. But also the nose and the upper respiratory tract are susceptible to infections. Exposure to oil mists for concrete workers is usually low.

Effects on the respiratory tract have been reported for occupations with a higher risk of exposure (Bukowski, 2003; Concawe, 1986; Terwoert, 1999).

Effects on the skin

Regular contact of the skin with mineral oils can result in infection of parts of the skin, leading to pimples and carbuncles. Furthermore intense oil-skin contact resulting in damage of the skin may lead to eczema. This effect will be especially important in case of intense and prolonged contact of the oil with the skin. The eczema is usually localised on the hands (Concawe, 1986; Terwoert, 1999).

Incidentally allergic reactions occur as a result of skin exposure to mineral oil-based release agents. The allergic reaction however, is almost always the result of one the additives present in the oil and not from the oil itself. Once the allergic reaction has occurred, every exposure to this particular oil (or to the allergic component in it) will result in an allergic reaction (Concawe, 1986; Terwoert, 1999).

The experience with vegetable oil based release agents is limited, but in case of intense and prolonged skin contact similar effects as for mineral oil are to be expected.

No allergic effects are known for vegetable oil based products. In the printing industry there is considerable experience with the use of these compounds. No sensitizing properties have been discovered so far. Similar to the mineral oil-based products additives are the most likely cause of sensitization. The exact nature of the additives is not known. According to the (Dutch) suppliers the water-based release agents do not contain corrosion inhibitors, biocides or antioxidants. Therefore vegetable based release agents are not expected to cause allergies (Terwoert, 1999).

According to IARC highly-refined mineral oils are not classifiable as to their carcinogenicity to humans (Group 3) (IARC, 1984).

Silica

General

Exposure to dust can occur during almost all activities on construction sites, from excavation for the foundations up until the final sweeping before the completion of the building. Depending on the nature of the building material being used this dust can contain a considerable amount of silica. Crystalline free silica (silicon dioxide, SiO_2) can occur in three phases: quartz, cristobalite and tridymite. These forms are also called "free silica" to distinguish them from the silicates. Quartz is the most important and most prevailing type. The silica content in different rock formations, such as sandstone, granite and slate, varies from 20 to nearly 100% (Parker and Wagner, 1998).

Reports about exposure to respirable quartz in the building industry are relatively scarce, but workplace respirable quartz concentrations often exceed exposure limits (Flanagan, et al., 2006; Flanagan, et al., 2003; Hubbs, et al., 2005; Lumens and Spee, 2001).

In The Netherlands values exceeding the exposure limit of 0.075 mg/m^3 have been measured (IARC, 1988a; Lumens and Spee, 2001; Onos and Spee, 2004; Tjoe Nij, et al., 2003a; Tjoe Nij, et al., 2003b; Tjoe Nij, et al., 2004; Tjoe Nij and Heederik, 2005). Control measures can reduce quartz exposure to acceptable levels (Beamer, et al., 2005; Flynn and Susi, 2003; Golla and Heitbrink, 2004; Tjoe Nij, et al., 2003c).

Health hazards of silica

Occupational exposures to respirable crystalline silica are associated with the development of silicosis, lung cancer, pulmonary tuberculosis, and airways diseases. These exposures may also be related to the development of autoimmune disorders, chronic renal disease, and other adverse health effects (Ding, et al., 2002; NIOSH, 2002; Parker and Wagner, 1998; Peretz, et al., 2006; Steenland, 2005).

Silicosis

Silicosis is a fibrotic disease of the lungs caused by the inhalation, retention and pulmonary reaction to crystalline silica. Chronic, accelerated and acute forms of silicosis are commonly described. These clinical and pathologic expressions of the disease reflect differing exposure intensities, latency periods and natural histories. The chronic or classic form usually follows one or more decades of exposure to respirable dust containing quartz, and this may progress to progressive massive fibrosis (PMF). The accelerated form follows shorter and heavier exposures and progresses more rapidly. The acute form may occur after short-term, intense exposures to high levels of respirable dust with high silica content for periods that may be measured in months rather than years (Linch, 2002; Parker and Wagner, 1998; Tjoe Nij, et al., 2003a; Tjoe Nij and Heederik, 2005).

Silicosis may be complicated by severe mycobacterial or fungal infections. About half of these are caused by

Mycobacterium tuberculosis and result in TB. Epidemiologic studies have firmly established that silicosis is a risk factor for developing TB (NIOSH, 2002; Parker and Wagner, 1998).

Lung cancer

In 1997 the International Agency for Research on Cancer (IARC) classified inhaled crystalline silica as a human carcinogen (group 1) (IARC, 1997). Subsequent reviews of the epidemiologic evidence have called into question the IARC classification of silica as a confirmed human carcinogen, citing concerns about uncontrolled confounding by smoking, radon and other factors, and some inconsistencies in dose-response trends (Hessel, et al., 2000; Soutar, et al., 2000). Nonetheless, there is now relatively broad acceptance internationally that crystalline silica poses a lung cancer risk, albeit not as potent as seen for asbestos (NIOSH, 2002; Peretz, et al., 2006; Steenland, et al., 2001). DECOS concludes that quartz induced lung tumours are preceded by signs of local fibrosis. The best explanation for carcinogenicity is indirect genotoxic action, in which long-term irritation of the target cells is the crucial step. Since the irritation precedes the genotoxicity, the carcinogenicity of quartz is a non stochastic one, implicating that the carcinogenicity of quartz is characterized by a threshold (DECOS, 1998). There seems to be no synergistic interaction between smoking and silica (Yu and Tse, 2006).

COPD

Occupational exposure to respirable crystalline silica is associated with chronic obstructive pulmonary disease (COPD), including bronchitis and emphysema. The results of some epidemiologic studies suggest that these diseases may be less frequent or absent in non-smokers. Exposure to respirable crystalline silica is not associated with asthma (Hnizdo and Vallyathan, 2003; NIOSH, 2002).

Autoimmune and chronic renal diseases

Many case reports have been published about autoimmune diseases or autoimmune-related diseases in workers exposed to crystalline silica or workers with silicosis. In addition, several recent epidemiologic studies reported statistically significant numbers of excess cases or deaths from known autoimmune diseases or immunologic disorders (scleroderma, systemic lupus erythematosus, rheumatoid arthritis, sarcoidosis), chronic renal disease, and subclinical renal changes. The pathogenesis of autoimmune and renal diseases in silica exposed workers is not clear (Ding, et al., 2002; NIOSH, 2002; Steenland, 2005).

Dust

General

"Dust consists of solid particles, ranging in size from below 1 µm up to at least 100 µm, which may be or become airborne, depending on their origin, physical characteristics and ambient conditions" (WHO, 1999). For sampling purposes a number of international organizations have reached agreement on definitions of inhalable, thoracic and respirable fractions (Lippmann, 1998; WHO, 1999). Distinctions are made between (see also figure 5):

1. Those particles that are not aspirated into the nose or mouth and therefore represent no inhalation hazard;
2. The inhalable (also known as inspirable) particulate mass: particles that are inhaled and are hazardous when deposited anywhere within the respiratory tract;
3. The thoracic particulate mass: particles that penetrate the larynx and are hazardous when deposited anywhere within the thorax and;
4. The respirable particulate mass: particles that penetrate through the terminal bronchioles and are hazardous when deposited within the gas-exchange region of the lungs.

Nuisance dust can be defined as dust that, even after years of exposure, has little or no adverse effects on the lungs (although excessive exposure may cause irritation or injuries to the skin or mucous membranes). Portland cement and gypsum are considered nuisance dust, for which a workplace exposure limit of 10 mg per m^3 for total dust applies and 5 mg per m^3 for the respirable fraction (DECOS, 2002).

Figure 5. The inhalable, thoracic and respirable conventions as percentages of total airborne particles. From: Boleij J, Buringh E, Heederik D, Kromhout H (1995) Occupational hygiene of chemical and biological agents, p 51. Elsevier, Amsterdam ISBN 0-444-81997-5

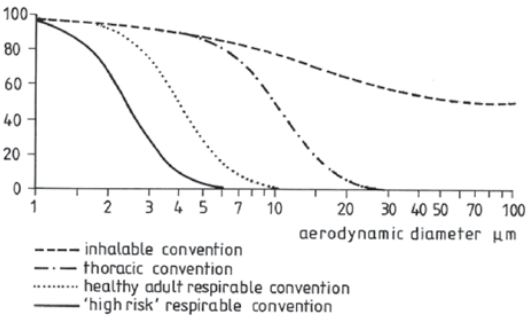

Figure 3.8 The inhalable, thoracic and respirable conventions as percentages of total airborne particles (CEN, 1993)

Health hazards of dust

The harmful effects of dust depend on a number of factors:

- Dust composition: chemical or mineralogical;
- Dust concentration;
- Particle size and shape;
- Exposure time.

Pneumoconioses

One of the definitions of pneumoconiosis is: "Pneumoconiosis is the accumulation of dust in the lungs and the tissue reaction to its presence" (David, 1998).

Very serious forms of pneumoconiosis are silicosis and asbestosis. See also the headings **asbestos** and **silica**.

Cancer

Many dusts are confirmed carcinogens and examples of carcinogenic dusts which may be present at construction sites are: asbestos (see the heading **asbestos**), free crystalline silica (see the heading **silica**), hexavalent chromium (IARC, 1990) and certain wood dusts (IARC, 1995). Fly ash may contain heavy metals among which some genotoxic carcinogens (beryllium,

chromium [VI] en nickel) (Jongen, et al., 2003) and quartz (Borm, 1997).

Systemic poisoning

Some chemical dusts can enter the organism, pass to the bloodstream and have harmful effects on organs, blood or the nervous system (WHO, 1999). Examples are toxic metal dusts (lead, cadmium) (ILO, 1998b).

Allergies

Some dusts may cause allergic reactions. Examples are certain wood dusts (McCann, 1998b) and metals (ILO, 1998b).

Effects on the skin

Some dusts affect the skin and may cause various types of dermatoses. Examples are cement (see the heading **cement**), some metals and wood dusts (ILO, 1998b; McCann, 1998b). Cement and gypsum may cause caustic burns on the skin (Spoo and Elsner, 2001).

Paints and varnishes

General

Paints and coatings include paints, varnishes, lacquers, stains, printing inks and more. Traditional paints consist of a dispersion of pigment particles in a vehicle consisting of a film-former or binder (usually a resin nowadays) and a solvent. In addition, there can be a wide variety of fillers and other additives (e.g. biocides).

A varnish is a solution of oil and natural resin in an organic solvent. Synthetic resins may also be used. Lacquers are coatings in which the film dries or hardens entirely by evaporation of the solvent. Traditional paints were under 70% solids with the remainder being mostly solvents. Air pollution regulations limiting the amount of solvents that can be emitted into the atmosphere have resulted in the development of a wide variety of substitute paints with low or no organic solvents present (McCann, 1998a). Terwoert et al. have discussed the composition of a number of commonly used paints and putties (Terwoert, et al., 2002). Synthetic floorings contain similar resins, fillers and additives to paints and floor layers may be exposed to similar chemicals (Terwoert and Raalte, 2002).

Health hazards of paints and varnishes.

Dermatitis

Construction painters are exposed to a wide variety of chemical substances in paints, plaster and putties and to dusts from sanding which may cause irritant or allergic contact dermatitis (Estlander, et al., 2000; Kaukiainen, et al., 2005a; Terwoert and Raalte, 2002; Terwoert, et al., 2002). There has been some discussion that the shift from solvent based to water based paints has led to an increased risk of dermatitis (Fischer, et al., 1995; Wieslander, et al., 1994b). In a recent study no increased dermatitis risk associated with water based paints was found among construction painters, but the risk for hand dermatitis seems to increase with higher exposure to solvent based paints and glues, especially epoxy and urethane paints and water based putties and plasters (Kaukiainen, et al., 2005a). Fillers, plasters and putties contain ingredients such as limestone, cement, aluminum cement and gypsum and may contain also solvents and epoxy compounds. All of these ingredients are known to affect the skin. Many fillers and plasters are alkaline products and can irritate the skin and even corrode the skin (Kaukiainen, et al., 2005a). Also, plasters and fillers may contain other skin affecting components, like biocides (Bohn, et al., 2000; Gruvberger, et al., 1998; Isaksson, et al., 2004; Jensen, et al., 2003; Reinhard, et al., 2001). The prevalence of skin disorders among painters and construction workers in the Netherlands is discussed in two Arbouw reports (Terwoert and Raalte, 2002; Terwoert, et al., 2002). The conclusion from these reports is that there is no evidence for water based paints to cause more skin disorders than solvent based paints.

Respiratory disorders

Studies on occupational respiratory effects in construction painting are rare. Irritative respiratory and asthma symptoms have been attributed to water based paints, but their adverse health effects are not consistent (Kaukiainen, et al., 2005b; Terwoert, et al., 2002; Wieslander, et al., 1994a; Wieslander, et al., 1997). The results from a recent study indicate a higher risk for respiratory symptoms and chronic bronchitis among construction painters than among carpenters (Kaukiainen, et al., 2005b)

Neurotoxic effects

Construction painters run a higher risk of exposure to organic solvents than other construction workers. The effects of organic solvents on the nervous system is discussed below.

Organic solvents

General

Organic solvents are widely used in industry. In the construction industry they are used in paints, glues, adhesives, cleaning agents, etc. Commonly used solvents include alcohols (methanol, ethanol, isopropanol, glycols), aromatic hydrocarbons (toluene, xylene), solvent mixtures (turpentine, white spirits), chlorinated solvents (methylene chloride), glycolethers/-esters and ketones. Legislation has led to a decline in the use of solvents in a number of industries and a growth in the use of water-based paints (Dick, 2006; EC, 1999).

Health hazards of organic solvents

Solvents are volatile agents and, in general, occupational exposures occur by inhalation of solvent vapour. However, dermal exposure is important in some industries such as painting and industrial degreasing. Dermal uptake may contribute a significant fraction of the total body burden of solvents in workers employed in these sectors (Semple, 2004).

Acute health effects

The acute health effects of organic solvents reflect their central nervous system effects and include headache, dizziness and light-headedness progressing to unconsciousness, seizures, and death (Dick, 2006). Eye, nose and throat irritation may also occur (Chen, et al., 2001b).

Long term health effects

Chronic toxic encephalopathy (CTE)

Studies in Scandinavia in the 1970s and 1980s suggested that long term, high level solvent exposure might be associated with

a syndrome of personality change, memory loss and neurological deficits variously termed chronic toxic encephalopathy (CTE), psycho-organic syndrome or solvent neurotoxicity (Axelson, et al., 1976; Hogstedt, 1994). Since then solvent neurotoxicity has been studied extensively (Chen, et al., 2001a; Daniell, et al., 1999; Dick, et al., 2000; Dick, et al., 2002; Gamble, 2000; Hout, et al., 2006; Nilson, et al., 2003; Nilson, et al., 2002; Ridgway, et al., 2003; Spurgeon, 2001; Triebig and Hallermann, 2001).
Recent, well designed studies suggest that in heavily exposed workers, solvents may have subtle effects on cognitive function (Spurgeon, 2001). The cognitive domains affected by solvent exposures include attention, verbal memory, and visuospatial skills (Daniell, et al., 1999; Nilson, et al., 2002). There is some evidence that solvent neurotoxicity is more common among those with at least 10 years of occupational exposure to solvents. Whether the important determinant of adverse effects is the lifetime (cumulative) exposure, the intensity of exposure or peaks of exposure remains unclear (Dick, 2006). The shift from solvent based paints to water based paints during the last decades has resulted in a decrease in neurotoxic effects (NCvB, 2005; Triebig and Hallermann, 2001).

Ototoxicity

Experimental studies have documented that several solvents (styrene, xylenes, toluene, carbon disulfide, n-hexane) can produce hearing disorders under certain exposure circumstances. Studies in humans have indicated that the effect may be present following exposures that are common in the occupational environment. Synergistic effects between noise and chemicals have been observed in some human and experimental animal studies (Arlien-Soborg and Simonsen, 1998; Dick, 2006; Gagnaire and Langlais, 2005).

Other neurotoxic effects

Some solvents (e.g. n-hexane, methyl n-butyl ketone, carbon disulfide, styrene) are associated with peripheral neuropathy (Dick, 2006). A number of neurodegenerative diseases like Parkinson's disease, essential tremor, Alzheimer's disease, amyotrophic lateral sclerosis and multiple slerosis have been associated with occupational solvent exposure, but results are not unequivocal (Dick, 2006). Taste , sight, smell may also be affected by solvent exposure (IARC, 1989a; ILO, 1998c).

Other effects

Organic solvents differ widely in chemical composition, structure, volatility and in toxic effects on biological systems. Some may be carcinogenic, benzene is known to cause leukaemia, halogenated hydrocarbons may affect the kidneys or liver (IARC, 1989a; ILO, 1998c).
Both paternal and maternal exposure to organic solvents (e.g. ethanol, aromatic solvents, styrene, toluene) has been associated with adverse reproductive and developmental effects (Hooiveld, et al., 2006; Kumar, 2004; Laslo-Baker, et al., 2004; Logman, et al., 2005; Tielemans, et al., 1999; Yarborough and Erdreich, 2005).

Diesel Exhaust

General

Diesel engine emissions (DE) are highly complex mixtures. They consist of a wide range of organic and inorganic compounds distributed among the gaseous and particulate phases. Among the gaseous hydrocarbon components of DE that are individually known to be of toxicologic relevance are the aldehydes (e.g., formaldehyde, acetaldehyde, acrolein), benzene, 1,3-butadiene, and polycyclic aromatic hydrocarbons (PAHs) and nitro-PAHs. The particles present in DE (i.e., diesel particulate matter (DPM)) are composed of a center core of elemental carbon and adsorbed organic compounds, as well as small amounts of sulfate, nitrate, metals, and other trace elements. DE is emitted from 'on-road' diesel engines (vehicles) and from 'nonroad' diesel engines (locomotives, heavy-duty equipment, etc.) (EPA, 2002). The construction industry is one of the industries where some groups of workers run a higher risk of exposure to DE (Singels, et al., 2004).

Health hazards of diesel exhaust

Most attention for the toxic effects of diesel exhaust concentrate on effects on the lungs and respiratory tract, both acute and chronic. Acute exposure to DE can cause acute irritation (e.g., eye, throat, bronchial), neurophysiological symptoms (e.g., lightheadedness, nausea), and respiratory symptoms (cough, phlegm) and asthma-like symptoms. Chronic exposure may lead to inflammation and histopathological changes in the lung (EPA, 2002; Montelius, 2003).
Lung cancer and other cancers have been associated with exposure to diesel exhaust. Diesel exhaust has been classified by IARC as probably carcinogenic to humans (group 2A) (IARC, 1989b). The US Environmental Protection Agency EPA concludes that the human evidence from occupational studies is strongly supportive of a finding that DE is causally associated with lung cancer, though the evidence is less than that needed to definitively conclude that DE is carcinogenic to humans (EPA, 2002). In the Netherlands DE has been classified as carcinogenic (Staatsblad, 1997; SZW, 2006).

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