

Portland cement dust¹⁾

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Supplement 2012

MAK value	–
Peak limitation	–
Absorption through the skin	–
Sensitization (2011)²⁾	–
Carcinogenicity (2011)	Category 3B
Prenatal toxicity	–
Germ cell mutagenicity	–
BAT value	–
Chemical name	Mixture of Ca silicates, aluminates and ferrites (at different volume ratios)
Density at 20°C	2.8–3.2 g/m ³ (IFA 2010)
Colour	Grey powder
Solubility (water)	Poorly soluble
Use	Portland cement is used as a binding agent in the mortar and concrete manufacture.

Numerous epidemiological studies have been published since the appearance of the last documentation in 1993 (documentation “Portland Cement” 1998, a translation of the 1993 German version). They describe effects after exposure to portland cement, making a new assessment is required.

Chemical and physical properties

Portland cement is a mixture of various compounds (see Table 1).

1) Cr(VI) content and quartz level to be assessed separately

2) Only applies to low-chromate cements with a Cr(VI) content below 2 ppm. See chromium(VI) compounds for cements with a higher Cr(VI) content

Table 1 Mean chemical composition (% w/w) of standard cements produced in the Federal Republic of Germany (Locher 1983)

	CaO	SiO ₃	Al ₂ O ₃	TiO ₂	P ₂ O ₅	FeO	MnO	MgO	Na ₂ O	K ₂ O	SO ₃ ¹⁾
PC	64	20	5	0.3	0.1	2.5	0.1	1.5	0.2	0.9	3.0
IPC	58	23	7	0.4	0.1	2.5	0.1	1.5	0.2	0.9	3
BFC	51	26	9	0.5	0.1	1.5	0.5	4.0	0.3	0.8	3.0

PC: portland cement; IPC: iron portland cement;

BFC: blast-furnace slag cement

¹⁾ multimer

The previously valid names and symbols were changed in the revised version of the German standard DIN 1164-10: 2004-08 to take European standards into account. Cement has now been divided into 3 main types: portland cement CEM I (portland cement (PC)); portland slag cement CEM II/A-S, CEM II/B-S (iron portland cement (IPC)) and blast-furnace slag cement CEM III, CEM III/B (blast-furnace slag cement (BFC)) (Geldsetzer 2011).

The initially malleable mixtures of cement and water (cement mortar and cement paste) solidify to become hydrated cement containing about 25% water (related to the cement weight) that is bound chemically and another 10–15% water that is bound adsorptively. Adding calcium sulfates by grinding retards hardening of at least 1 hour. Ettringite is first formed, which after some time converts to monosulfate ($3 \text{ CaO} \cdot \text{Al}_2\text{O}_3 \cdot \text{CaSO}_4 \cdot 12 \text{ H}_2\text{O}$) (Geldsetzer 2011).

Particle form and particle size

The particle size in cement dust varies between 0.05 µm and 5.0 µm (aerodynamic diameter). Particles of 20 µm are sporadically measured (Kalacic 1973).

Exposure at the workplace

Exposure to portland cement dust is expected to occur mainly in the cement and construction industries. In 2008, 7371 workers were employed in the German cement industry according to the Association of the German Cement Industry (<http://www.bdzement.de>), and 705 000 construction workers were employed in the construction industry according to the Association of the German Construction Industry.

1 Toxic Effects and Mode of Action

Portland cement particles are absorbed orally and by inhalation. The respiratory tract is the main target organ in humans after repeated inhalation exposure. Impairment of pulmonary function and clinical signs similar to chronic obstructive pul-

monary disease (COPD) were often diagnosed in the exposed persons. Pneumocoinosis or pleural changes are of subordinate relevance. The results of several epidemiological studies have substantiated the suspected increased risk of cancer of the larynx through portland cement although the studies have deficiencies such as small case numbers and an at times inadequate consideration of the alcohol consumption confounder, socio-economic status and simultaneous exposure to limestone.

Cement dust is corrosive to the eyes and skin, particularly on contact with moisture when calcium hydroxide (pH of 10 to 12) forms from strongly alkaline calcium oxide. Cement dust on the mucosal surface also leads to strongly alkaline reactions and may cause considerable irritation to the mucosa or even ulcerations.

No animal studies have been carried out since the last documentation appeared in 1993 (documentation "Portland Cement" 1998, a translation of the 1993 German version). Due to insufficient data, no conclusions can be drawn on reproductive toxicity.

Studies on the threshold concentration for the induction of an allergic reaction to chromate (see Supplement "Chromium(VI) compounds" 2012, not yet available in English) show that induction of sensitization by portland cement containing a chromate concentration of less than 2 ppm is very unlikely.

2 Mechanism of Action

There are no special studies available for the mechanism of action. However, almost all epidemiological and animal studies (documentation "Portland Cement" 1998, a translation of the 1993 German version) demonstrated irritant and inflammatory reactions, particularly in the upper respiratory tract, after exposure to portland cement. Since this dust consists of 51–64% calcium oxide, corrosive alkalinity plays the decisive role on contact with moisture.

3 Toxicokinetics and Metabolism

There are no data available for toxicokinetics or metabolism.

4 Effects in humans

4.1 Single exposure

There are no data available for single exposure.

4.2 Repeated exposure

Results of occupational health studies of workers in the portland cement industry have been available for several decades.

The 1993 documentation "Portland Cement" (documentation "Portland Cement" 1998, a translation of the 1993 German version) revealed that repeated exposure to portland cement may cause rhinitis, chronic bronchitis and an impairment of pulmonary function. In 1997, a general threshold limit value for dust of 4 mg/m^3 (inhalable fraction) was established on the basis of the DFG chronic bronchitis study, which also included workers exposed to portland cement.

A cross-shift study carried out in a portland cement factory in Saudi Arabia investigated changes in pulmonary function among workers during one shift. Pulmonary function was tested among 149 exposed male workers, who were selected randomly (all working areas were represented). The average age was 38.0 ± 0.8 years. A total of 348 employees from the administration staff of this and other companies (petrochemical and asbestos industries) at an average age of 34.3 ± 0.5 years were used as controls. The control group was selected according to the smoking habits of the exposed persons. Respirable dust was measured gravimetrically in different production areas. There is no information on whether these were person-related or stationary measurements. The 97 measurements of respirable dust revealed high dust concentrations for all activities (geometric mean (GM): $7.1\text{--}20.3 \text{ mg/m}^3$ in 5 working areas). Spirometry was carried out before and after the morning shift. Forced expiratory volume (FEV_1), forced vital capacity (FVC) and maximal mid-expiratory flow rate ($\text{FEF}_{25-75\%}$) were determined. The spirometry data were not adjusted for body height. Significantly lower means for FEV_1 , for FEV_1/FVC (%) (Tiffeneau index) and for $\text{FEF}_{25-75\%}$ were measured among the exposed workers. Regression analysis showed no relationship between changes of the parameter changes and smoking habits. In a multivariate analysis, the post-shift reduction in spirometric parameters was significantly higher in exposed employees than in the controls and was significantly associated with exposure (yes/no) and the pre-shift pulmonary function value (Ali et al. 1998).

Further cross-sectional studies and a retrospective cohort study have been published since the appearance of the 1993 documentation (documentation "Portland Cement" 1998, a translation of the 1993 German version) (Table 2). Some of the studies included pulmonary function tests as well as information on symptoms and objective findings. X-ray findings were evaluated in one cross-sectional study. Another 3 studies among portland cement workers only considered symptoms.

Table 2 Effects in humans after repeated exposure to portland cement

Study population	Country	Exposure (mg/m ³) Record of exposure	Results/comments	Dose-response relationship	References
2736 exposed workers and 755 external controls from 16 of 153 cement plants	United States	total dust GM: 2.9 respirable fraction GM: 0.57 current measurements and duration	impairment of pulmonary function; none PEF (l)	none	Abrons et al. 1988
about 2640 exposed workers and 1589 external controls	United States	see Abrons et al. 1988	X-ray abnormalities of the pleura, but not of the lungs	with the duration of employment	Abrons et al. 1997
304 exposed workers from 4 cement plants	United Arab Emirates	not specified	increased incidences of cough and chronic bronchitis (only prevalence data of symptoms)	not tested	About-Taleb et al. 1995
348 exposed workers and 189 controls of another study (symptoms only) from a cement plant	Jordan	current measurements and duration 3 exposure groups: respirable dust GM: 0.5, 1.6 and 3.9	increased incidences of cough and dyspnoea; no impairment of pulmonary function parameters	not tested	Abudhaise et al. 1997
67 exposed workers and 134 external controls from a cement plant	United Arab Emirates	not specified	increased incidences of cough, phlegm, wheezing, dyspnoea, sinusitis, asthma and chronic bronchitis and reduced pulmonary function parameters	not tested	Al-Neaimi et al. 2001

Table 2 (Continued)

Study population	Country	Exposure (mg/m ³) Record of exposure	Results/comments	Dose-response relationship	References
425 exposed workers from 4 cement plants	Mexico	estimated individual JEM; no measured values		not tested	Alvear-Galindo et al. 1999
150 exposed workers and 355 external controls from a cement plant	Saudi Arabia	97 current dust measurements, GM: 7.1–20.3 in 5 areas	increased incidences of cough, phlegm, dyspnoea, wheezing and bronchial asthma	not tested	Ballal et al. 2004
157 exposed workers and 45 external controls from a cement plant	India	not specified	PEF (↓)	not tested	Chandra-wanshi and Pati 1996
119 exposed workers and 50 external controls (former workers included) from a cement plant	Norway	estimated individual JEM; current measurements: mean: 7.4 mg/m ³ total dust or 0.91 mg/m ³ respirable dust categorized according to low and high	no effect on PEF values	none	Fell et al. 2003
120 workers and 120 external controls from 2 concrete factories	Morocco	not specified	no signs of impaired pulmonary function parameters; no increased COPD spirometry	not tested	Laraqui et al. 2001

Table 2 (Continued)

Study population	Country	Exposure (mg/m ³) Record of exposure	Results/comments	Dose-response relationship	References
280 exposed workers and 73 internal controls from a cement plant	Morocco	not specified	increased bronchitis and dermatitis	+ (but only for 3 exposure groups)	Laraqui Hossini et al. 2002
53 exposed workers, 158 workers from 2 other areas and 211 non-exposed persons (all non-smokers) from a cement plant	Ethiopia	mean respirable dust from 2 areas: high: 43.1 mg/m ³ ; low: 3.2 mg/m ³	increased incidences of cough, phlegm, none dyspnoea and rhinitis and reduced pulmonary function parameters		Mengesha and Bekele 1998
50 exposed workers and 50 external controls	Pakistan	not specified	increased incidences of cough, phlegm, not tested dyspnoea and rhinitis and reduced pulmonary function parameters		Meo et al. 2002
115 exposed workers and 102 internal controls from a cement plant	Tanzania	total dust GM: 10.6 current measurements	reduced pulmonary function parameters	(+) pulmonary function poorer at high exposure	Mwaiselage et al. 2004

Table 2 (Continued)

Study population	Country	Exposure (mg/m ³) Record of exposure	Results/comments	Dose-response relationship	References
32 exposed workers (30 smokers excluded) and 70 external controls from a cement plant	Malaysia	11 years of exposure to fine/coarse/total at 6 sites; GM not specified; max. total dust: 10.18 mg/m ³	reduced pulmonary function parameters	tendency to an increased hospitalization rate after a prolonged exposure period	Noor et al. 2000
546 exposed workers and 857 external controls	Denmark	median total dust: 3.3 mg/m ³ respirable dust 1.5 mg/m ³	increased incidences of cough, phlegm, dyspnoea and rhinitis and reduced pulmonary function parameters	not tested	Vestbo and Rasmussen 1991
412 exposed workers and 179 internal controls (clean jobs) from 4 cement plants	Taiwan	current measurements 3 exposure groups: respirable dust GM: 0.22, 0.55 and 1.24 mg/m ³	only increased incidence of phlegm in the highest exposure group	?	Yang et al. 1993
identical cohort as Yang et al. 1993	Taiwan	current measurements and duration respirable fraction GM: 3.58 mg/m ³	increased incidence of wheezing, dyspnoea and COPD and reduced pulmonary function parameters	not tested	Yang et al. 1996

PEF: peak expiratory flow; JEM: job exposure matrix; GM: geometric mean;

?: questionable; most important studies are in bold type

Retrospective cohort study

A retrospective cohort study recorded breathing difficulties and pulmonary function among 119 Norwegian portland cement workers. The study was carried out in 1998 and 1999. The cohort consisted of retired male portland cement workers who were born between 1918 and 1938 and had worked in a portland cement factory for at least 1 year. Originally, it included 226 workers; 148 of them were still alive. There is no information about the causes of death of the workers who died. The 119 participants were 69 years old on average, and the median exposure period was 21.8 years. A group of 50 workers without any substantial exposure to other chemicals was used as a control group. In both groups, 35% of the persons had died; the ratio of smokers was somewhat higher in the portland cement group (35%) than in the control group (26%). During the study, 20 exposure measurements were carried out yielding a mean total dust concentration of 7.4 mg/m^3 (respirable dust: 0.91 mg/m^3); on the basis of 4 individually estimated JEMs (job exposure matrix), categorization was performed according to a high and a low exposure group (all values were below the Norwegian occupational limit value of 5 mg/m^3 for respirable dust and 10 mg/m^3 for total dust). Crystalline silicon dioxide was detected in 1 of 3 samples at a concentration of 0.06 mg/m^3 . The size of the portland cement particles was $0.05\text{--}5.0 \text{ }\mu\text{m}$ (aerodynamic diameter). The prevalence of respiratory symptoms was somewhat higher among the portland cement workers and positively associated with the cumulative exposure index for 3 of 13 symptoms (cough, dyspnoea at rest and symptoms at work); however, there were no statistically significant differences between the control group and the portland cement plant workers. There were no significant differences for pulmonary function parameters after adjustment for age, body height, smoking and exposure to asbestos. FEV_1 and FVC were measured. The results of the pulmonary function test were similar in the exposed group and in the control group. The spirometry results from the high concentration and low concentration exposure groups were also similar among the portland cement workers. The prevalence of COPD was 14% in the control group and 14.3% for portland cement workers (Fell et al. 2003).

However, the number of examined persons was small. Exposure measurement at the time of the study consisted of only a few measured values. No earlier measurements are available, but the retrospective recording of individual exposure was described in detail and is certainly more conclusive than considering only the employment period. It is the strength of the study that former workers were assessed. Therefore, it is a reliable “negative” study. There is no information about whether the mean concentration refers to the arithmetic or geometric mean. Assuming that exposures were probably higher in the past, respirable dust concentrations of about 1 mg/m^3 seem to be without substantial effects.

Cross-sectional studies

In a cross-sectional study from 2002, the pulmonary function of 115 male workers (96% of recruited persons) from a portland cement plant in Tanzania was compared with 102 internal controls (maintenance and office workers; 95.8%). A mean exposure period of 12 years was reported for cement plant workers. Standardized questionnaires recorded information about smoking and the use of personal protective equipment. At 32%, the proportion of smokers was higher in the portland cement group than that of 25% in the control group (Mwaiselage et al. 2004). Total dust was measured 120 times in 80 workers from 8 working areas, and cumulative exposures were calculated by multiplying the working area means with the employment period. The current dust concentrations varied considerably among the working areas, the geometric means being 38.6 and 21.3 mg/m³ in the two areas with the highest exposure to dust. The annual cumulative dust exposure was 11 mg/m³ · years (range of 0.3–38 · years) in the control group and 69 mg/m³ · years (range of 3–926 · years) for workers. About 30% of the workers in production and maintenance had regularly had worn face masks, but the quality and effectiveness of these masks was not specified. There was no relationship between wearing face masks and the results of pulmonary function testing. Among the portland cement plant workers, the pulmonary function tests revealed a tendency to lower values for FEV₁, FEV₁/FVC and maximal expiratory flow (PEF) and a higher prevalence of pulmonary function impairment at dust concentrations of 3.2 mg/m³ and higher (cumulative: 36 mg/m³ · years and higher). These differences were statistically significant. Similar effects were also obtained among smokers in both groups. The linear regression analysis, which considered body height, age and smoking habits, showed a negative association between cumulative dust exposure and pulmonary function parameters. Among workers exposed to the high concentration of >300 mg/m³ · years, an increased odds ratio (OR) of <0.7 of 9.9 (95% CI: 3.5–27.8) was obtained for FEV₁/FVC. For the group exposed to 100–300 mg/m³ · years, the OR was 1.6 (95% CI: 0.5–5.1) after adjustment for age and smoking habits. Exposed persons showed poorer pulmonary function values than the controls; a dose-response relationship was found for cumulative dust concentrations and spirometric parameters. Cases with distinct obstructive ventilation disorders were extremely often employed in areas with high exposure. Exposures of at least 300 mg/m³ · years were statistically significantly associated with respiratory obstruction. However, only current measured values of the dust concentration were available. The effects are definite and indicate that a limit value of 10 mg/m³ (cumulative: 300 mg/m³ · years) for total dust is too high.

In a cross-sectional study from Morocco, a higher prevalence of respiratory disorders was found among 280 cement plant workers as compared with 73 workers in factories without exposure to cement. At 65% among the workers exposed to cement, it was higher than the 35% in the control group. Cough, phlegm, dyspnoea and rhinitis were the most common symptoms. Among cement workers, 30% suffered from chronic bronchitis as compared with 10% of the controls. The preva-

lence of these symptoms was highest among workers who were smokers or ex-smokers and had been exposed to a high dust concentration over a period of more than 10 years. Pathological spirometry results were obtained in 22% of the workers exposed to cement as compared with 14% of the control group although most (78%) of the deviations were regarded as slight. There was no clear association between the degree of exposure and the results of the pulmonary function analysis. No data on dust measurement are available except for a remark stating that vision was impaired by dust at the workplace and dust deposits were seen on the clothing and hair (Laraqui Hossini et al. 2002).

In a retrospective cross-sectional study, the prevalence of respiratory disorders was determined among workers from 2 concrete factories in Morocco. In addition to cement dust, the workers were exposed to sand and gravel. The study included 120 concrete plant workers who were compared with 120 external controls (civil servants). No dust measurements were available. A higher prevalence of respiratory disorders, rhinitis, conjunctivitis and dermatitis was reported among the concrete factory workers. The ratio of workers with pulmonary function disorders was also higher among concrete workers (Laraqui et al. 2001).

In a cross-sectional study, 50 cement mill workers in Karachi, Pakistan, aged 20 to 60 years and with an exposure of 13 years, were compared with a control group of 50 volunteers of the same structure in terms of age, body height, weight and socio-economic status. Confounders such as smoking were not included in the study. Spirometry and EMG of intercostal muscles were effect parameters. FEV_1 /FCV, PEF and maximal voluntary ventilation (MVV) were consistently lower among workers of the areas exposed to dust, but the $FEF_{25-75\%}$ values were similar to those of the control group (Meo et al. 2002). The study demonstrated that long-term exposure to cement dust may cause disorders of the bronchi. The study is limited since there is no information about the level of dust exposure or about the working conditions in the cement mill. The selection of exposed workers and controls was not described sufficiently, nor were smoking habits recorded. Moreover, the duration of exposure was not associated with effect parameters. Because of the absence of exposure data, this study cannot be used for the assessment of portland cement.

Another cross-sectional study (year of the study not specified) compared pulmonary function and the prevalence of respiratory disorders in a group of workers ($n = 67$; 89%) of Pakistani or Bengal origin from a portland cement factory in the United Arab Emirates with an unexposed cohort of the same structure ($n = 134$; 99%). The ratio of smokers was significantly higher in the group exposed to dust. Exposure was not recorded. The average exposure period was 16 years. As compared with the control group, exposed workers showed a higher prevalence for the following symptoms: cough (30% in the exposed group versus 10% in the control group), phlegm (25% versus 5%), wheezing (8% versus 3%), dyspnoea (21% versus 5%) and sinusitis (27% versus 11%). The prevalence of asthma and chronic bronchitis was higher in the exposed group (6% versus 3% and 13% versus 4%) than in the control group. All pulmonary function parameters (FVC, FEV_1 and FEV_1/FVC)

were considerably reduced in the exposed group and were not associated with smoking habits (Al-Neaimi et al. 2001). No dose-response relationship was established in this study.

In a cross-sectional study (year of the study not specified) from Malaysia, 32 portland cement factory workers at an average age of 35 years who were non-smokers and had worked in the factory between 0.5 and 38 years were compared with an external control cohort ($n = 70$; all non-smokers had an average age of 37 years). The 30 smoking workers were excluded. Current measurements of dust in the packing area of the cement factory yielded a maximum total dust concentration of 10.2 mg/m^3 including $8.0 \text{ mg fine dust/m}^3$. The FEV_1 and $\text{FEF}_{25-75\%}$ were lower in the portland cement worker group than in the control group after adjustment for body height and age. These differences were particularly significant for 30- to 40-year-old exposed persons. After exposure, the cement workers were divided into 3 groups: persons with high (dust exposure at 8.52 mg/m^3 total dust), moderate (2.80 mg/m^3) and low (0.58 mg/m^3) exposure. The decrease in the pulmonary function parameters FEV_1 and $\text{FEF}_{25-75\%}$ correlated with the level of exposure. As compared with the control group, a higher prevalence of the following symptoms was obtained among cement workers: coughing in the morning (25% of cement workers versus 6% of the controls), phlegm in the morning (24% versus 11%) and tightness of the chest (19% versus 6%) (Noor et al. 2000). This study also showed a higher prevalence of chronic respiratory symptoms and an impairment of pulmonary function among cement workers as compared with university students/staff. Although the control cohort was not adjusted for its socio-economic status, an association between the dust exposure level and a decrease in lung function was observed among the cement workers. The number of persons examined was low in this study, and smokers were also excluded. Only a few measured values were recorded for exposure, and only current measurements were described. The internal analysis can hardly be verified since categorization was not explained, nor was the number of persons in the exposure categories described. Therefore, the study cannot be interpreted with regard to a dose-response relationship.

In a cross-sectional study from Ethiopia, 53 cement factory workers, 91 workers of a cotton yarn factory and 67 cigarette factory workers were compared with 211 non-smoking controls. All exposed persons were non-smokers. No information was available about age, sex or duration of employment. The groups were adjusted according to their socio-economic status. Measurement of dust yielded a maximum total dust concentration of $3.2 \pm 3.5 \text{ mg/m}^3$ for kiln workers and of $43.1 \pm 35.3 \text{ mg/m}^3$ for packing workers. The prevalence of respiratory disorders among cement workers was considerably higher than in the control group: 30% of the portland cement workers and 9% of the control group workers suffered from chronic cough, 26% and 9% from chronic bronchitis and 32% and 8% from asthma, respectively. After exposure to cotton yarn and cement, the effects were about the same and more pronounced than after exposure to tobacco dust. FVC was similar in both groups (kiln workers and packing workers in the cement factory). Among packers, FEV_1 was somewhat lower and FEV_1/FVC was considerably lower. PEF (peak ex-

piratory flow) was also lower in packers, but it was not statistically significant. The effects on the lungs and lung function were regarded as adverse (Mengesha and Bekele 1998). A special feature of this study is the exclusive consideration of non-smokers. The study is limited because of the small number of cases in the portland cement factory (pulmonary function was reported for only 28 of the persons categorized according to exposure). Moreover, the packing workers were exposed to very high concentrations; the dust concentrations were apparently only specified as arithmetic means.

A cross-sectional study (year of the survey not specified) included 348 workers (58% of the recruited persons, i.e. 94 of 442, were excluded for different reasons) from a portland cement factory in Jordan. The portland cement workers were men at an average age of 41 years and an exposure period between 3 and 14 years. The cohort was divided into 3 groups according to their exposure level: group 1 with the lowest exposure consisted of administrative staff and transport workers, group 2 consisted of maintenance staff, brick workers and foremen and group 3 of packing workers and cement mill workers. Measurements of dust in exposure groups 1, 2 and 3 yielded respirable dust concentrations with geometric means, presumably 8-hour means, of 0.5 ± 2.1 , 1.6 ± 2.6 and 3.9 ± 4.0 mg/m³. In the last 3 years, 58% of the workers had no respiratory disorders. Among the remaining workers, cough (19%) and dyspnoea (18%) were the most common symptoms. Asthma was diagnosed in about 16% and chronic bronchitis in 15%. The prevalence of asthma was higher in group 3 (27%), but there was no difference between the groups for other respiratory disorders. The prevalence of respiratory disorders was higher among smokers. The pulmonary function parameters FEV₁, FVC, FEF_{25-75%} and FEV₁/FVC were similar in all 3 groups (Abudhaise et al. 1997). In spite of the large number of complaints, no associations were found between symptoms or pulmonary function and exposure. Only values measured at the time of the study were considered. The rate of participation was relatively small and only group 1, which was exposed to the lowest portland cement concentration, can be regarded as a control group, if at all. The discrepancy between a high number of persons with symptoms and the absence of pulmonary function restrictions cannot be explained.

In a study from India the PEF (peak expiratory flow) rate was measured among day workers and shift workers with the aim of determining the influence of shift work on pulmonary function among cement plant workers. For this purpose, 157 day workers and 208 shift workers of a cement plant and 45 external day workers were examined. No exposure data were specified. The PEF values of exposed day workers were not different from the external controls (Chandrawanshi and Pati 1996). The objective of the study was to determine differences between day work and shift work rather than to establish the effect of exposure to dust. Because of the absence of exposure data, this study is not used for the assessment of portland cement.

In a cross-sectional study (year of the study not specified) from Taiwan, pulmonary function and respiratory symptoms of 412 workers at an average age of 44 years and an average exposure period of 17 years in four cement plants were compared

with 179 workers without exposure to cement from the same plant. The total cohort excluded 123 persons, 70 of these because of an insufficient employment period (<5 years). A total of 147 measurements were carried out in areas with exposure (respirable dust; GM: 3.58 mg/m³). The prevalence of cough (19% versus 12%) and of increased phlegm (18% versus 13%) was higher in the exposed group, but the prevalence of wheezing, dyspnoea and chronic bronchitis was similar in both groups. After adjustment for smoking, age and body height, the pulmonary function parameters FVC, FEV₁, FEV₁/FVC and forced expiratory flows at 50% and 75% of the vital capacity were significantly lower in the exposed group than in the control group (Yang et al. 1996). This study thus revealed clearly adverse effects among the persons exposed in regard to symptoms and pulmonary function. This study also only recorded current measured values for the dust concentration. All persons apparently participated in the study; only a small number of persons that were not employed for a sufficiently long period had to be excluded.

Another survey studied the pulmonary function of cement workers employed in four cement plants in Taiwan. It was the same cohort as in the study by Yang et al. (1996), but the workers were internally categorized according to 3 exposure groups (geometric means: 0.22, 0.55 and 1.24 mg/m³). The duration of employment as a sign of cumulative exposure was not considered. The average age and the employment period were similar in the 3 groups although groups 2 and 3 included more smokers. The prevalence of cough, which was higher in group 3 (17% as compared with 11% for groups 1 and 2), was the only difference in respiratory symptoms among the groups; this difference was statistically significant after adjustment for smoking. No comparison with the control group was made. The prevalence of other symptoms, chronic bronchitis and the results of the pulmonary function analysis were similar in all groups. This study showed a significantly increased prevalence of cough among the workers exposed to dust at a concentration of 1.24 mg/m³ (respirable fraction). Pulmonary function revealed no significant differences (Yang et al. 1993). However, categories were only based on current measured values rather than on cumulative exposure.

A cross-sectional study (year of the survey not specified) in a Mexican portland cement factory recorded symptoms by means of a questionnaire. The study included 425 workers (94% of the workforce) at an average age of 39 years. No controls were included. All had worked in the factory for their whole working life. Individual cumulative exposure was calculated from the specific employment period and by assigning 1 to 6 exposure levels. Dust was not measured. Most of the workers had been exposed to dust exposure levels of 4, 5 or 6 most of the time. Very few workers below 25 reported respiratory symptoms. However, 8% of the 25- to 44-year-old workers and 16% of the 45-year-old workers suffered from bronchitis; 72% and 83%, respectively, had dyspnoea and 24% and 32%, respectively, had wheezing. An occupational disease was diagnosed among 32% and 52% of the workers, respectively; the criteria of diagnosis were not described. The prevalence of respiratory symptoms was highest in the high exposure group after adjustment for age. There were definite associations between exposure and respiratory symptoms

(Alvear-Galindo et al. 1999). Since no control group was used and dust was not measured, this study cannot be used to derive a quantitative association between exposure to portland cement and pulmonary function symptoms.

In a cross-sectional study (year of the study not specified) from the United Arab Emirates the respiratory symptoms of cement workers were investigated. A sample of 304 workers (total workforce unknown) was selected randomly from 4 cement factories. They accounted for about 50% of the workers of each factory and were 38 years old on average; 88% were from India. No controls were included. Chronic cough (19%) and chronic bronchitis (12%) were reported most frequently. Blocked nose, itching eyes, dermatitis, headache and fatigue were described as further symptoms (Abou-Taleb et al. 1995). Because of the absence of exposure data, this study is not used for a quantitative assessment of portland cement.

In a cross-sectional study from the United States, 2640 workers from 16 portland cement factories were X-rayed. These factories were selected at random from the 153 cement factories in the United States. The participants' average age was 41 years, and 95% of them were male. The controls were recruited from 10 factories without exposure to cement, but there was exposure to solvent vapours, for example. Persons with particularly high exposure who worked as welders, grinders or spray painters for the entire day were excluded. Person-related measurements of dust (211 respirable dust samples and 1011 total dust samples) for orientation yielded a maximum respirable dust concentration of 0.57 mg/m^3 (range: 0.01–46.22) and a maximum total dust concentration of 2.90 mg/m^3 (range: 0.01–78.61) (presumably an 8-hour mean). Crystalline silicon dioxide was determined in about 15% of the individual samples of the respirable fraction, and the limit value of 0.05 mg/m^3 valid at that time was exceeded in about 10% of these cases. No asbestos fibres were detected in the samples. The X-rays showed small deviations between the two groups. Round and irregular small opacities (category 1 or larger) were observed in 1% of the portland cement workers and in about 0.1% of the control group. Three portland cement workers had small opacities of category 2, while opacities of category 1 were found in all other cases. The prevalence of pleural abnormalities was about 1.6% in exposed workers and 0.2% in the control group. These differences were statistically significant even after adjustment for age and smoking. An analysis that considered the duration of employment and cement exposure showed an association between exposure to cement and pleural changes, but not for the diffuse lung opacities (Abrons et al. 1997). This study showed an association between employment in the cement industry and a slight increase of X-ray abnormalities that was particularly pronounced in smokers. However, only very few cement workers had findings that indicated a severe pulmonary disease. The cross-sectional design and the exclusive availability of current measured values are substantial weak points of this study. The selection of the controls and the fact that a few highly exposed controls were excluded is discussable. The study showed slight effects for pleural changes, but not for intrapulmonary abnormalities.

In a cross-sectional study, 150 selected workers of an Arab portland cement factory were compared with 355 external control workers. Respirable dust was mea-

sured 97 times in 5 working areas. The current dust concentrations varied among the working areas at geometric means between 7.1 and 20.3 mg/m³. Cumulative exposures were calculated. The following symptoms were effect parameters: cough, increased phlegm, wheezing, shortness of breath and bronchial asthma. Smoking was taken into account. The rate of respiratory symptoms was increased in exposed persons, and persons with symptoms had higher cumulative exposures (Ballal et al. 2004). According to the available measured values, exposure was high when compared with the limit value of 5 mg portland cement/m³ valid in Germany at that time for the inhalable dust fraction.

A study in cement plants in 24 European countries recorded exposure characteristics at the workplace, respiratory symptoms and pulmonary function parameters since 2007. Elevated odds ratios were recorded for symptoms and impairment of the respiratory tract, but not for chronic bronchitis among persons with high exposure as compared with those with low exposure. FEV₁ showed a dose-related reduction (Nordby et al. 2011).

Summary

The 1993 documentation "Portland Cement" (documentation "Portland Cement" 1998, a translation of the 1993 German version) indicated that a fibrogenic connective tissue reaction was possibly caused by cement dust. Most of the studies published since then were cross-sectional studies that were carried out in portland cement plants outside Europe. The only study from Europe was a retrospective cohort study with a relatively small number of Norwegian cement workers (Fell et al. 2003). This study demonstrated that prolonged exposure to cement dust at a concentration of about 1 mg/m³ (respirable fraction) is not associated with decreased pulmonary function or increased prevalence of COPD.

All available studies have some shortcomings. Because of the cross-sectional design, workers who had left the plants due to health problems were not recorded. Current and regularly obtained historical dust measurements at the workplace are often missing. Nevertheless, all these studies have described an increased prevalence of obstructive pulmonary diseases, chronic bronchitis and reduced pulmonary function among portland cement workers. Cough, increased phlegm and dyspnoea were the most common symptoms. The lowest dust concentrations associated with reduced pulmonary function were 3 mg/m³ for total dust (Mwaiselage et al. 2004) and 0.58–4 mg/m³ for respirable dust (Noor et al. 2000; Yang et al. 1996). The lowest concentration that was significantly correlated with an increased prevalence of respiratory disorders was 1.2 mg/m³ respirable dust (Yang et al. 1993).

The currently available data do not allow the derivation of a limit value. There is no doubt that high exposure to portland cement by inhalation may cause clinical signs similar to COPD. Pneumoconiosis or pleural changes are of subordinate relevance. All studies in the low exposure range only included current measured values.

Therefore, valid conclusions about limit values can be drawn only on the assumption that earlier exposures were certainly not lower (Abrons et al. 1988).

4.3 Effects on skin and mucous membranes

Portland cement dust causes corrosions and ulcerations of the skin on contact with moisture when strongly alkaline calcium hydroxide (pH: 10–12) forms. Because of its alkaline effect, portland cement may also be corrosive to the eyes. Cement dust that deposits on the mucosal surface also leads to strongly alkaline reactions and may cause considerable irritation to the mucosa – up to ulcerations (ECB 2000).

4.4 Allergenic effects

Unlike low-chromate cement, cement containing chromium has long been known to induce cement dermatitis, an allergic reaction of the skin to chromate (see Supplement “Chromium(VI) compounds” 2012, not yet available in English; EU 2003; NIOH 2003). Cements produced in the former GDR according to the wet process and gypsum sulfuric acid process contained less than 0.4 ppm chromate and only rarely induced hand eczema (Reifenstein et al. 1986). However, in cements produced in the dry process, small amounts of chromate may form from chromium compounds contained in the raw materials or from the abrasion of the cement mills when cement clinker is manufactured in the kiln. Adding iron(II) sulfate reduces water-soluble hexavalent chromium to trivalent chromium, which remains bound in the alkaline milieu of cement as poorly soluble chromium hydroxide/chromium oxide (Fregert et al. 1979; Geier and Struppek 1995). In Germany, where an industry-wide regulation mandated the addition of iron sulfate to packed cement as of the year 2000 and its addition in other areas from 2002, there had been no substantial decrease in the incidence of sensitization by 2000 among surveyed persons that were employed and examined in the construction industry (Uter et al. 2004). Since 2005, low-chromate cement of the type that is made ready-for-use by adding water may only be put on the German market if it contains less than 2 mg soluble chromium(VI) 1 kg dry weight of cement. If reducing agents are used to decrease the chromium content, suitable storage conditions and a maximum storage period have to be observed (Bundesregierung 2004; EU 2003). After a decline in the prevalence of chromate sensitization was observed in the Scandinavian countries very soon after the introduction of low-chromate cement (Avnstorp 1989 a, b, 1991, 1992; Zachariae et al. 1996), meanwhile, a similar decrease in newly registered cases of cement or chromate eczema has been reported in Germany (Kluger 2007). A recent review of the data of the Information Network of German Departments of Dermatology from 1994 to 2008 also showed a decrease in sensitization to chromate. From 1994 to 1996, 1997 to 1999 and 2000 to 2002, the reaction rates among

patients from the construction industry with exposure to cement and occupationally induced skin disease (bricklayers, concrete workers, composition floor layers, tilers and plasterers) were 43.1%, 41.7% and 37.1%, respectively, but declined to 26.9% from 2003 to 2005 and 29.0% from 2006 to 2008. When compared with workers who started work before 1994, the reaction rate among workers who did not begin until after 1999 was even more clearly reduced (13 of 65 tested versus 40.4% of 687 tested persons). Since the number of workers who were tested during the 3-year periods dropped concurrently, the absolute number of construction workers registered with chromate allergy more than halved during the study period (Geier et al. 2011). Individual protective measures were apparently not, or not the only, cause of this decline since the rate of sensitization to epoxy resin in the group whose employment began after 1999 almost tripled as compared with the group of workers who started work before 1994. Moreover, no increased reaction rates were established for “typical” rubber allergens, which may be an indication of increased skin protection through the use of rubber gloves.

An earlier Finish study had already demonstrated that the reduced incidence of sensitization to chromate after low-chromate cement was introduced in 1987 was very probably not due primarily to improved individual protective measures since the frequency of irritant skin diseases was almost constant (Roto et al. 1996).

4.5 Reproductive and developmental toxicity

A study from Taiwan (Kaohsiung) showed an increased prevalence of premature deliveries among mothers living within 0–2 km of a portland cement plant as compared with mothers living within 2–4 km (Yang et al. 2003). After adjustment for various potential confounders, the odds ratio for premature delivery was 1.3 (95% CI: 1.09–1.54) for mothers living within 0–2 km versus mothers living within 2–4 km. This suggests an association between air pollution caused by portland cement plants and an increased incidence of premature births.

However, in spite of the high number of volunteers ($n = 17\,116$), the results of this study have been assessed critically. First, the premature birth rates were relatively low in both groups of mothers (5.71% and 4.45%) and second, standardization according to age was not very precise; only 3 groups of age were differentiated (≤ 20 years, 21–34 years and ≥ 35 years). However, age-related differences are expected to occur in the 21–34 year group. Adjustment according to socio-economic status was also relatively crude (married or not; less than 12 years school/training or 12 and more years). Living close to a portland cement plant might also be associated with a lower socio-economic status. This would have to be considered in more detail. An attempt was made to minimize the influence of previous pregnancies by including only women in the study who were facing their first viable birth. However, previous spontaneous abortions, abortions or stillbirths were not taken into account although they have a considerable influence on the premature birth

rate (Voigt et al. 2009). Finally, other potential confounders were ignored (maternal body mass index and tobacco consumption; both substantially affect the premature birth rate). The premature birth risk after portland cement exposure cannot be assessed because of the methodological weaknesses of this study.

4.6 Genotoxicity

In a study from India, the frequencies of sister chromatid exchanges (SCEs) were measured in the lymphocytes of workers from an Indian portland cement plant (Fatima et al. 1995). The cohort of exposed persons consisted of 59 non-smokers aged 24–54 years, and the exposure period was 1–17 years. The mean SCE rate of 8.98 per cell among exposed workers was significantly higher than that of 3.5 in the control group. The SCE rates were significantly increased in relation to the employment period. No adjustment for age was made. The relevance of these findings is unclear.

4.7 Carcinogenicity

Based on earlier study findings (Brown et al. 1982; Cauvin et al. 1990; Flanders and Rothman 1982; Flanders et al. 1984; Olsen and Sabroe 1984; Peterson and Zwerling 1998; Rothman et al. 1980; Zaganiski et al. 1986), an association between exposure to portland cement dust and laryngeal cancer cannot be ruled out (documentation “Portland Cement” 1998, a translation of the 1993 German version; HSE 2004). To clarify this suspicion, the studies that appeared after 1993 will be described in detail here. The Health Safety Executive, U.K., published a summary of the studies on the carcinogenicity of portland cement (HSE 1994, 2004). Table 3 and Table 4 show the results of the studies on the carcinogenicity of portland cement.

A retrospective cohort study investigated cancer mortality among 2400 workers at 2 Swedish cement factories (Jakobsson et al. 1993). The cohort consisted of male workers who had worked at the factories for at least 1 year since January 1952. Death certificates provided information on the cause of any death prior to the end of 1986. About 90% of the cancer diagnoses were confirmed by histopathological and cytological examinations. The analysis focused on cancers occurring more than 15 years after exposure began. Very limited exposure data were available. The report stated that new cement ovens and automatic process controls were introduced at both plants during the 1960s and ventilation was improved during the 1970s, suggesting that dust exposure during the latter years of the study period was considerably lower than before. Dust measurements made at factory A from the middle of the 1970s onwards were lower than 10 mg/m³ for total dust (presumably as an 8-hour mean). At factory B, total dust concentrations were less than 20 mg/m³ during the early 1970s and less than 10 mg/m³ in later years. Concentrations greater than

Table 3 Cohort studies on the carcinogenicity of portland cement among exposed workers

Cohort	Country/observation period	Exposure	Cause of death tumour localization	Deaths obsvd./exp.	Risk	95% CI	Remarks	References
2400 male workers in 2 cement factories	Sweden 1952–1986	52 111 person years	all causes of death	495/527	0.94 ^a	0.86–1.03		Jakobsson et al. 1993
			all tumours	97/116	0.83 ^a	0.68–1.02		
			right-sided colon tumours	13/5.75 12/4.39	2.52 ^a 2.73 ^a	1.34–4.32 1.41–4.77	total >15 exposure years	
					4.31 ^a	1.73–8.87	>25 exposure years	
1526 male cement factory workers	Sweden 1958–1987	21 341 person years	right-sided colon tumours	12/4.59	2.61 ^a	1.35–4.57		Jakobsson et al. 1994
546 male cement factory workers	Denmark 1974–1985		all tumours	162/158	1.03 ^b	0.88–1.2	total	Vestbo et al. 1991
			respiratory tract tumours		0.5 ^b	0.1–1.5	1–20 exposure years	
					1.0 ^b	0.4–2.6	≥21 exposure years	

Table 3 (Continued)

Cohort	Country/observation period	Exposure	Cause of death tumour localization	Deaths obsvd./exp.	Risk	95% CI	Remarks	References
2498 cement factory workers (70% men)	Lithuania 1978–2000	43 490 person years	all deaths from malignant neoplasms	102	1.3 ^c 1.2 ^a	1.0–1.51 1.0–1.4	no adjustment for smoking habits	Smallyte et al. 2004
			lungs	40	1.4 ^c 1.5 ^a	1.0–1.9 1.1–2.1		
1172 masons	Iceland 1955–1993	not specified	lungs	25/14.81 23/14.1	1.69 ^a 1.63 ^a	1.09–2.49 1.03–2.45	total ≥10 exposure years	Rafnsson et al. 1997
				16/9	1.77 ^a	1.01–2.88	≥30 exposure years	
307 799 construction workers	Sweden 1971–2001		pharynx	65 52 13	1.9 ^d 1.9 ^d 1.9 ^d	1.2–3.1 1.1–3.2 0.7–5.0	“ever” moderate high	Purdue et al. 2006
260 052 construction workers	Sweden 1991–2000	73 241 person years	oesophageal adenocarcinomas	5	3.8 ^e	1.5–9.6	high	Jansson et al. 2005

^a standardized incidence rate (SIR); ^b observed/expected; ^c standardized mortality rate (SMR); ^d relative risk (RR) compared with 4445 never-exposed patients;
^e incidence rate ratio (IRR)

Table 4 Case-control studies on the cancer risk after exposure to portland cement

Cases	Controls	With cement exposure cases/controls	Tumour localization	Exposure to cement	Relative risk [95% CI]	References
164 patients with squamous cell carcinoma of larynx	656	20.2/10.5%	supraglottal carcinoma	not specified	1.88 [0.8–4.39]	Maier et al. 1992
105 patients with squamous cell carcinoma of oropharynx and hypopharynx	420	34.3/10.1%	squamous cell carcinoma in oropharynx and hypopharynx	not specified	2.2 [0.9–5.2]	Maier et al. 1994
209 patients with squamous cell carcinoma in oral cavity, pharynx and larynx	110	24.8/2.7%	supraglottal carcinoma	not specified	12.9 [3.9–43.0]	Maier et al. 2002
257 patients with squamous cell carcinoma of larynx	769	6.8/2.4% 8.1/2.7%	laryngeal carcinoma	moderate high	2.22 [1.02–4.84] 1.87 [0.88–4.01]	Dietz et al. 2004
270 patients with confirmed lung cancer	383	44/48 62/56 106/104	squamous cell carcinoma of lungs	1–20 years ≥21 years ever exposed	1.6 [0.9–2.7] 1.6 [0.9–2.5] 1.6 [1.1–2.3]	De Stefani et al. 1996

100 mg/m³ were found for some cleaning and maintenance tasks, but the use of respiratory protection was required while performing these operations. Data from factory B were based on personal sampling, but the sampling time was not provided. The quartz content of the dusts was below 5%, and no cases of silicosis were reported at these plants. Overall mortality (495 observed deaths versus 527 expected) was similar to the national rate in Sweden. Overall tumour morbidity (162 observed cases versus 160 expected) did not differ from the national rate either. However, the analysis of the individual tumour sites revealed a significantly increased risk for tumours of the right side of the colon (standard incidence rate (SIR): 2.7; 95% CI: 1.4–4.77; 12 cases). The risk was increased among those exposed to cement dust for more than 25 years (SIR: 4.3; 95% CI: 1.7–8.9; 7 cases). However, the risk of right-sided colon tumours was not significantly increased at factory A. All colon cancer cases had been confirmed by histology or cytology. The left-sided colon tumour rate was not increased. The authors discussed the possibility of bias due to the use of a control group of farmers who are known to have very low rates of colon cancer.

In a later study, the risk of right-sided colon tumours among workers of 2 Swedish cement factories was compared with that for workers without exposure to cement and for fishermen (Jakobsson et al. 1994). All workers were employed for a minimum of 1 year, and only cancers occurring more than 15 years after the beginning of employment were considered. Cancer mortality and morbidity was determined for the period of 1958 to 1987 (slightly later for fishermen), and SMR and SIR were calculated with reference to the general male population of Sweden. The cohort of cement workers consisted of 1526 workers with a median birth year of 1927. The asbestos cement cohort included 981 workers from a building materials factory; the median birth year was 1913. The third cohort consisted of 3965 workers from a variety of industries where some chemical exposure was likely. The cohort of fishermen was 8092 in number, with a median birth year of 1924. Overall cancer mortality for cement workers was similar to that of the general population (SMR: 1.02; 95% CI: 0.92–1.25) and to that of “other” industrial workers. The cancer mortality for asbestos cement workers was slightly higher, and for fishermen slightly lower. Morbidity from right-sided colon tumours was significantly increased among cement workers (SIR: 2.61; 95% CI: 1.35–4.57), but the risk of left-sided tumours was not elevated. The results for asbestos cement workers were similar. Morbidity for colon cancers among “other” industrial workers and fishermen was similar to that of the general population. This study showed an increased risk of right-sided colon tumours among workers with long-term exposure to cement dust compared with “other” industrial workers and the general population.

The relation between employment in a Danish portland cement factory and cancer morbidity was examined in a cohort study from Denmark (Vestbo et al. 1991). The cohort of cement workers consisted of 546 men who were between 46 and 69 years old and had at some time before 1974 worked in the factory for 1 year or more; it was compared with a control group of the same structure (n = 856). No dust measurements were available from the time before 1974. Person-related dust

measurements carried out after 1974 showed that 9% of the measured values were above 5 mg/m³. Cement workers were not found to have an increased risk for any types of tumour as compared with the general population. However, the risk of respiratory tract tumours was higher among cement workers than in the general male population. After all workers with exposure to asbestos (5%) had been excluded and the group was adjusted for age and smoking, the relative risk for respiratory tract tumours was 0.5 (95% CI: 0.1–1.5) for men who had been employed at the cement factory for 1–20 years and 1.0 (95% CI: 0.4–2.6) for men employed for ≥20 years; both values were not statistically significant. The results of this study did not provide any evidence of an increased risk of cancer morbidity due to exposure to cement dust.

A cohort study from Lithuania examined mortality and cancer incidence among workers of a portland cement plant (Smailyte et al. 2004). The study population comprised workers who were exposed to cement and employed in the factory for at least 12 months between 1956 and 2000 and were available for the follow-up period of 1978 to 2000. Of the 2498 participants, 70% were men. Some information about the annual mean total dust concentrations was available from the time after 1975. They were calculated from static sampling measurements (sampling time was not stated) conducted 4 times a year in the different work areas. The dust concentrations were highest in the packing (ranging from 15.0–19.1 mg/m³) and calcining (8.2–19.2 mg/m³) departments. Overall mortality was not increased among male cement workers (376 observed deaths; SMR: 1.0; 95% CI: 0.9–1.1), but deaths from malignant neoplasms were elevated (102 observed; SMR: 1.3; 95% CI: 1.0–1.5), mainly due to lung cancer (SMR: 1.4; 95% CI: 1.0–1.9), but not significantly. Mortality of obstructive respiratory disease was not increased. Among females, mortality was similar to that of the general Lithuanian population. Cancer mortality for male cement workers was not significantly elevated (SIR: 1.2; 95% CI: 1.0–1.4) except for lung cancer (SIR: 1.5; 95% CI: 1.1–2.1). All cement workers had also been involved with the production of asbestos cement at another plant (unclear). When these were excluded from the analysis, the SIR for lung cancer remained elevated (SIR: 1.4; 95% CI: 1.0–2.0), although not significantly. A correlation existed between the duration of employment and an increased lung cancer risk. There was no relationship between lung cancer and cumulative dust exposure although the male cement workers were exposed to the highest annual dust concentrations of 55–130 mg/m³ (SIR: 2.0; 95% CI: 1.2–3.4) and more than 130 mg/m³ (SIR: 1.8; 95% CI: 1.1–3.0). The authors described an increased risk of bladder cancer, but this increase was not significant (8 cases; SIR: 1.8; 95% CI: 0.9–3.5) and there was no correlation with duration of employment or cumulative dust exposure. There was a trend towards an increased incidence of stomach cancer associated with cumulative dust exposure; the SIR was 1.1 for an annual exposure to 55–130 mg/m³ (4 cases; 95% CI: 0.4–2.8) and the SMR was 1.5 for exposure to more than 130 mg/m³ (6 cases; 95% CI: 0.6–3.0). The study is limited mainly because no data on tobacco consumption were available. It was stated that about half of the male population of Lithuania are smokers, and it is likely that more blue-collar workers smoke than

white-collar workers. Therefore, smoking would have to be included in the study as an important confounder. If the analysis had taken smoking into account, the statistically significant differences observed for lung cancer among men in this study may have been eliminated. For this reason, conclusions on an association between exposure to cement and the incidence of lung cancer cannot be drawn from this study.

A retrospective cohort mortality study was carried out in all 1172 Icelandic masons who were born after 1880 and still alive in 1955. Death certificates were evaluated to analyze cancer mortality. Most of the cancer diagnoses were verified histopathologically. Masons alive at the time of the study were questioned on their smoking habits and duration of employment. No quantitative exposure data were available (Rafnsson et al. 1997). The study showed an increased risk of lung cancer compared with the general population of Iceland (SIR: 1.69; 95% CI: 1.09–2.49). The SIR was increased when the analysis allowed for a latency period of 30 years since completing vocational training (SIR: 1.77; 95% CI: 1.01–2.88). The percentage of non-smokers among masons was estimated to be lower than in the general population, but this cannot explain the increased risk. There were no increased risks for the cement masons at any other tumour site. Possible exposure to silicon dioxide was assumed, but no cases of silicosis were identified.

In a cohort study among 307 799 Swedish construction workers (all men), Purdue et al. (2006) found 510 squamous cell carcinomas (period: 1971–2001) in the region of the head and neck (171 in the oral cavity, 112 in the pharynx and 227 in the larynx). Exposure to a number of hazardous substances including cement dust was assessed using a semi-quantitative job-exposure matrix. There was a significantly increased relative risk for pharyngeal carcinoma after former occupational exposure to cement dust (exposure category: ever; 20 cases of 65; RR: 1.9 (95% CI: 1.2–3.1)) and also after moderate exposure to cement dust (16 cases of 52; RR = 1.9 (95% CI: 1.1–3.2)), but not after high exposure to cement dust (4 cases of 13; RR = 1.9 (95% CI: (0.7–5.0)) as compared with patients never exposed to cement (445 cases). No significant relationships with exposure to cement dust were identified with reference to laryngeal carcinoma, oral cavity carcinoma or the summary consideration of all examined cancer types. These results suggest that cement dust may cause pharyngeal carcinoma. The strengths of this study are the cohort size, consideration of the tobacco consumption confounder and the use of a job-exposure matrix developed for this cohort. However, the absence of an obvious dose-response relationship (significant RR for moderate, but not for high exposure to cement; possibly due to the small number of highly exposed workers), the absence of information about the exposure period and the absence of adjustment for the risk factor of alcohol consumption have to be assessed critically.

A prospective cohort study examined 260 052 Swedish construction workers from 1971 to 2000 (Jansson et al. 2005). IRRs (incidence rate ratios) were calculated for 3 different types of cancer for various occupational exposures including exposure to cement dust using a multivariate Cox regression model adjusted for attained age, time of entering the cohort, smoking habits and body weight. A positive asso-

ciation was found between high exposure to cement dust and the risk of oesophageal adenocarcinoma (IRR: 3.8; 95% CI: 1.5–9.6) as compared with controls. Moderate exposure to cement dust revealed no significantly increased risk for oesophageal adenocarcinoma. Cement dust exposure did not lead to a significantly increased risk of cardia adenocarcinoma or oesophageal squamous cell carcinoma as compared with the control. The study also showed positive associations between high exposure to asbestos and oesophageal adenocarcinoma and between high exposure to asphalt fumes or wood dust and cardia adenocarcinoma. The large number of cases, the long follow-up period spanning many years and the consideration of some confounders are the strengths of this study. A limitation of the study is that risks may also occur by chance because of the large number of individual statistical tests (12 specific and 2 combined occupational exposures with reference to 3 types of cancer) and 2 significances. It should also be noted critically that the alcohol consumption of the study population was not taken into account.

A multisite case-control study from Uruguay from 1996 with 270 confirmed lung cancer patients and 383 control persons without tumours at the time of the study and comparability for most confounders analyzed occupational risk factors for lung cancer (De Stefani et al. 1996). There was an increased cancer risk for workers with exposure to cement (OR =1.6; 95% CI: 1.1–2.3). Squamous cell carcinoma was the main form of lung cancer among construction workers.

A cohort study among 9118 workers examined the association between employment in a cement factory in France and total mortality for all causes of death and cancer as a specific cause of death. There were 430 (4.7%) deaths during the follow-up period; malignant neoplasm was the cause of 48% of these deaths. The SMR (standardized mortality ratio) was 0.68 (95% CI: 0.61–0.74) and that for cancer mortality was 0.80 (95% CI: 0.69–0.92). According to the authors, this study does not support previous observations that cement workers are at a higher risk of mortality after exposure to cement dust (Dab et al. 2011).

German case-control studies for squamous cell carcinomas of the oral cavity, pharynx and larynx

Earlier case-control studies (Maier et al. 1990, 1991), which were summarized in the 1993 documentation “Portland Cement” (see documentation “Portland Cement” 1998, a translation of the 1993 German version), reported an elevated risk of squamous cell carcinomas of the oral cavity, pharynx and larynx associated with occupational exposure to portland cement. However, the available results were not sufficient to confirm an association between inhaled cement dust and the development of laryngeal cancer. Further studies on this subject have been published in the last few years.

A case-control study among 164 patients of the Heidelberg University ENT clinic with squamous cell carcinoma of the larynx and 656 control persons investigated a possible association between exposure to portland cement and a risk of laryngeal cancer (Maier et al. 1992). About 92% of the tumour patients were unskilled or

skilled workers. Only 8% of the persons questioned reported a higher level of education, as opposed to at least 31.1% of the control. After statistical correction for possible alcohol and tobacco effects, an increased relative risk of developing laryngeal cancer was detected among workers with long-term exposure to cement dust. The risk associated with cement dust primarily referred to supraglottal carcinomas (odds ratio (OR) =1.88; 95% CI: 0.8–4.39). This value was not statistically significantly increased. However, this might be due to the small number of cases used.

Another case-control study among 105 patients with squamous cell carcinoma of the oropharynx and hypopharynx and 420 control persons investigated the influence of occupational factors on the risk of developing pharyngeal cancer (Maier et al. 1994). The study showed that 34.3% of the tumour patients worked in the construction industry as compared with 10.1% of the controls. Construction workers accounted for 26.7% of the tumour patients and 7.1% of the control persons. The odds ratio of developing pharyngeal cancer that was associated with construction work was estimated to be 2.5 (95% CI: 1.1–5.5). Statistical correction for the influences of alcohol and tobacco consumption resulted in an increased relative risk of pharyngeal cancer after exposure to cement (RR =2.2; 95% CI: 0.9–5.2) that was statistically not significant. The authors attributed the fact that the level of significance of $p < 0.05$ was not reached to the difficulty of assigning adequate controls to the cases when adjusting for alcohol consumption in the risk analysis due to the high average alcohol consumption in the tumour patient cohort (86.7 g/day versus 22.2 g/day in the control group).

In another case-control study (Maier and Tisch 1997; Maier et al. 1999), a statistically significantly increased relative risk of pharyngeal carcinoma (RR =2.5; CI: 1.1–5.9) and laryngeal carcinoma (RR =2.3; CI: 1.5–3.6) was identified among construction workers even after statistical correction of possible effects from alcohol and tobacco consumption. While 23.3% of the tumour patients were employed as construction workers, this was only the case for 8.2% of the control persons. A random sample analysis of AOK Heidelberg's patients showed that 21.2% of the men who had developed a malignant tumour of the upper aerodigestive tract were employed in the construction industry.

In a case-control study from 2002 assessing 209 male patients with confirmed squamous cell carcinomas and 110 male control persons who were without a cancer diagnosis at the time of the study and were comparable in terms of age, tobacco and alcohol consumption, occupational risk factors were analyzed for squamous cell carcinomas in the oral cavity, larynx and pharynx (Maier et al. 2002). The level of education and further training was significantly lower in the tumour patient group ($p < 0.001$). For example, 17.2% of the tumour patients had no vocational qualification as compared with 7.3% in the control group. The percentage of blue-collar workers, especially construction workers, was higher among the tumour patients (20.9% vs. 7.3%; $p = 0.002$). There was an increased relative cancer risk for workers with exposure to asbestos and cement (OR =12.9; $p < 0.001$).

In a case-control study (known as the “Rhine-Neckar larynx study”), the occupational risk factors associated with squamous cell carcinoma of the larynx were investigated (Dietz et al. 2004). Patients ($n = 257$) with histologically confirmed diagnoses of laryngeal cancer from the ENT clinics of Heidelberg, Mannheim, Ludwigshafen, Darmstadt and Heilbronn, who had been treated between May 1998 and December 2000, were compared with a control cohort of 769 persons of the same structure. Detailed interviews were held to obtain information on exposure. Specific questions were asked about exposure to cement, for example questions on the type of mortar or plaster material (on the basis of cement or lime) used, the handling of mortar or unslaked and slaked lime, daily direct or indirect exposure to cement dust and methods of processing cement products on-site. The time of exposure to cement was calculated for every work process and summarized in 3 estimation terms: “no exposure”, “moderate exposure” and “high exposure”. In the case of varying levels of simultaneous exposure within a group, the maximum exposure level was used for the period in question. Although the calculations in the publication make reference to cement dust exposure, exposure to lime and cement (before about 1965) and exposure to portland cement (after about 1965) were not clearly differentiated.

Alcohol consumption was calculated from the data provided in the interview – i. e. daily, weekly and monthly consumption during the 10-year period leading up to the interview – for all alcoholic beverages taking into account their alcohol content and then summarized in the following categories: <25 g, 20–50 g, 50–75 g and >75 g. As a surrogate for socio-economic status, 3 levels of schooling were used: 9 years and fewer equivalent to general school education, 10 years equivalent to intermediate secondary school) and more than 10 years equivalent to university (or applied sciences technical schools) entrance qualification). Smoking was found to be a significant risk factor for laryngeal cancer. The OR for heavy smokers (>40 pack years) was 32.5 (95% CI: 15.1–71.0) after adjustment for alcohol consumption. High alcohol consumption was also a risk factor, shown by an OR of 2.4 (95% CI: 1.5–9.7) for those consuming more than 75 g alcohol per day after adjustment for tobacco consumption. An analysis of the risk for tumours of the upper respiratory tract and exposure to portland cement showed that 23.3% of the patients were exposed to cement as compared with 14.4% of the control group, corresponding to an OR of 1.18 (95% CI: 0.77–1.18) after adjustment for alcohol and tobacco consumption and socio-economic status. A second analysis evaluated the responses to job-specific questions and found definite exposure to cement in only 14.8% of the cases as compared with 5.1% of the cases from the control group. After adjustment for alcohol and tobacco consumption and socio-economic status, an OR of 2.04 was established for laryngeal cancer (95% CI: 1.16–3.56), but no association between duration of exposure and the time of tumour onset. Among cement workers with high exposure to cement of more than 3000 hours, the relative risk was 1.87 (95% CI: 0.88–4.01), while in the low exposure group with less than 3000 cement working hours, the OR was 2.22 (95% CI: 1.02–4.48) after adjustment. The participation rate among the controls was 64%. This may have been a potential cause of differ-

ences in social status between cases and controls since a higher social status is generally associated with a higher willingness to participate. Information on exposure was obtained by interviews and expressed in working hours with lime/cement. Only portland cement was used from 1965 onwards, while co-exposures to cement dust and lime had been typical in the period prior to 1965 and cannot be separated retrospectively. According to the authors, none of the workers classified as exposed to cement had been exclusively exposed to lime. No dust concentration measurements were available. Increases of the relative risk for laryngeal cancer were observed after working with cement (before 1965 with additional exposure to lime), but these were considerably lower and no longer significant after adjustment for tobacco and alcohol consumption and socio-economic status. A monotonically increasing relationship was not evident when the findings were differentiated by low, moderate and high duration of exposure. Information on asbestos exposure additionally obtained by questionnaires yielded no risk factor and was therefore not included in the adjustment. Exposure varied with time due to diverse compositions of the lime and cement mixture. Heavy metals in cement, for example chromium VI, and the assumed alkaline reaction (pH values at 12.5–13) are also being discussed as possible causes of laryngeal cancer. The absence of dust measurement data and of monotony in the working hours-response relationship and an inadequate separation from exposure to lime limit the study in terms of the conclusions that can be drawn on the carcinogenicity of portland cement. According to the authors, the study provided evidence of a statistically significantly increased risk of laryngeal cancer after exposure to cement (in some cases with lime) irrespective of other factors such as asbestos or smoking. The authors recommended clarifying the carcinogenic potential of portland cement in more detail.

5 Animal Experiments and in vitro Studies

No recent animal studies are available on long-term exposure.

In vitro studies

In vitro studies were carried out using various portland cements. The particle size was smaller than 5 µm. Toxic effects were investigated in the NR8383 rat alveolar macrophage cell line. Titanium dioxide and quartz were used as negative and positive controls, respectively. Cytotoxicity was determined by means of 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrasodium bromide (MTT) and LDH assays, oxidative stress was established by measuring total glutathione depletion, and ESR (electron spin resonance) was applied to determine ROS formation. The release of tumour necrosis factor-α (TNFα), interleukin-1β (IL-1β) and macrophage inflammatory protein-2 (MIP-2) cytokines was determined by the enzyme-linked immunosorbent assay (ELISA). No toxic effects in macrophages or any decrease of glutathione was observed in comparison to quartz, nor was any formation of reactive

oxygen species detected. The samples were found to activate only macrophages for TNF α -production, and this was associated with their CaO content. The study showed that the effect of portland cement is more similar to titanium dioxide than to quartz DQ 12 (van Berlo et al. 2009).

In a study with 9 different cement samples (particle size smaller than 10 μm), no signs of cytotoxicity or inflammatory markers were observed in human primary epithelial cells or non-small-cell lung carcinoma cells A549, but the release of IL-8 was suppressed (Bauer et al. 2010).

6 Manifesto (MAK value, classification)

Portland cement dust is corrosive to the skin and eyes.

Carcinogenicity. Epidemiological studies on portland cement provided sporadic evidence of a carcinogenic potential in the larynx as the target organ. Some studies showed an increased risk of laryngeal cancer for the occupational group of construction workers who had been exposed to portland cement (with earlier co-exposure to lime), but there was no dose- or exposure-response relationship. No animal studies are available on the carcinogenic effects of portland cement. Due to the epidemiological findings, a suspected carcinogenic potential in humans cannot be ruled out for portland cement. Therefore, portland cement has been classified in Carcinogen Category 3B.

MAK value. Several epidemiological studies described irritant and inflammatory reactions after exposure to portland cement dust, mainly in the upper respiratory tract. Since the toxicological profile is determined by irritation, etc., chemical irritation in the upper respiratory tract should be avoided. The results available from studies with humans show that the general threshold limit value for dust of 4 mg/m³ (I fraction) does not provide protection against irritation caused by portland cement. The available evidence describes adverse effects at high portland cement concentrations and indicates the presence of a dose-response relationship. However, no MAK value can be derived from the available studies. Therefore, a scientifically based MAK value cannot be established. The previous MAK value has been withdrawn.

Germ cell mutagenicity. On the basis of the available data, genotoxicity is not suspected. Portland cement is therefore not classified into any of the germ cell mutagen categories.

Prenatal toxicity. There are no valid studies available for developmental toxicity. Since no MAK value has been established, portland cement has not been classified in any of the pregnancy risk groups.

Sensitization. Several studies demonstrated a decrease in the incidence of chromate sensitization among construction workers exposed to cement after the introduction of low-chromate cement. It is not possible to conclusively assess whether this measure helps to completely avoid sensitizations since the findings were possi-

bly influenced by other exposures and further factors such as various percentages of old sensitizations and different cohort characteristics, etc., and there was only a relatively small number of cases. Studies on the threshold concentration for the induction of an allergic reaction to chromate (see Supplement “Chromium(VI) compounds” 2012, not yet available in English) showed that sensitization is very unlikely to be induced by a chromate concentration of less than 2 ppm in portland cement dust. Therefore, low-chromate cement formulations are currently not designated with “Sh”. However, it cannot be ruled out that low-chromate portland cements may also induce allergic reactions in the case of an existing chromate sensitization.

Absorption through the skin. No data are currently available on possible absorption of portland cement through the skin. It is not possible to estimate the toxicological relevance of this route of exposure for systemic absorption of portland cement or its components. Portland cement has not been designated with “H” for the time being.

7 References

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