The MAK-Collection for Occupational Health and Safety

GMS PUBLISSO ZB MED-Publikationsportal Lebenswissenschaften

Portland cement dust

MAK Value Documentation, supplement – Translation of the German version from 2020

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Keywords

Portland cement; lung; lung function; irritation; carcinogenicity

Abstract

The German Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area has re-evaluated Portland cement dust [65997-15-1]. The critical effect of Portland cement dust is an irritant and inflammatory reaction, especially of the upper respiratory tract. In 2012, Portland cement dust was classified in Carcinogen Category 3 B because there was some evidence from epidemiological studies that Portland cement dust can cause pharyngeal tumours in construction workers who are additionally exposed to lime. More recent epidemiological studies could not rule out this suspicion. Therefore, Portland cement dust remains in Carcinogen Category 3 B. A maximum concentration at the work place (MAK value) could not be derived.

Citation Note:
Hartwig A, MAK Commission. Portland cement dust. MAK Value Documentation, supplement – Translation of the German version from 2020. MAK Collect Occup Health Saf. 2022 Sep;7(3):Doc051. https://doi.org/10.34865/mb6599715e7_3ad

Manuscript completed:
25 Oct 2018

Publication date:
30 Sep 2022

MAK value	–
Peak limitation	–
Absorption through the skin	–
Sensitization (2011)	–[a)]
Carcinogenicity (2012)	**Category 3 B**
Prenatal toxicity	–
Germ cell mutagenicity	–
BAT value	–
CAS number	65997-15-1
Density at 20 °C	2.8 to 3.2 g/cm^3 (Hartwig 2015)
Solubility	poorly soluble (Hartwig 2015)

[a)] Applies only to low-chromate cements with a chromium(VI) content of less than 2 ppm (2 mg/kg). For cements with a higher chromium(VI) content, see the documentation on chromium(VI) compounds.

Note: The quartz content and chromate content are to be evaluated separately.

Since the last supplement (Hartwig 2015), epidemiological studies describing effects after exposure to Portland cement dust have been published which make a re-evaluation necessary.

General

Today, cement is produced in modern cement plants in a continuous process using the dry method. In this process, the raw materials limestone (as a source of calcium oxide), clay (for silica and alumina), sand (for silica) and iron ore (iron(III) oxide) are ground together, dried and burned at 1400 °C. During calcination, calcium oxide, CO_2 and the 5 clinker compounds are produced: tricalcium silicate (alite; in short C_3S (3 $CaO{\cdot}SiO_2$)), dicalcium silicate (belite, in short C_2S (2 $CaO{\cdot}SiO_2$)), tricalcium aluminate (in short C_3A (3 $CaO{\cdot}Al_2O_3$)) and tetracalcium aluminate ferrite (in short C_4AF or $C_4(A,F)$ (4 $CaO{\cdot}Al_2O_3{\cdot}Fe_2O_3$)) and calcium aluminate ferrite (in short $C_2(A,F)$ (2 $CaO{\cdot}Al_2O_3{\cdot}Fe_2O_3$)). These calcium silicates are cooled to < 200 °C. After the addition of gypsum or anhydrite, which delays the curing rate, the granules are finely ground. Portland cement has a chemical composition of 58% to 66% calcium oxide (CaO), 18% to 26% silicon dioxide (SiO_2), 4% to 10% aluminium oxide (Al_2O_3) and 2% to 5% iron oxide (Fe_2O_3) (InformationsZentrum Beton 2017).

During processing, the hardening of Portland cement is triggered by the addition of water, whereby insoluble, stable calcium silicate hydrates are formed during hydration. Other hydration products include calcium hydroxide, calcium aluminate hydrates, calcium ferrite hydrates, hydrates containing sulfate and related compounds. Due to the formation of the alkaline calcium hydroxide from the calcium silicate, the cement paste (mixture of mixing water and Portland cement powder) has a pH of 12.5. The hardening process takes place via the initial stiffening of the cement paste (about 3 hours) to the final setting and hardening of the cement (> 28 days).

In order to attain the burning temperature of the cement clinker, hard coal and lignite and, to a lesser extent, heavy fuel oil were used in the past. Since 1990, petroleum coke as well as light and heavy fuel oil have been used. In addition to fossil fuels, alternative fuels such as used tyres, waste oil, animal meal, waste wood, solvents, fuller's earth and sewage sludge are also used (VDZ 2018).

1 Toxic Effects and Mode of Action

Portland cement dust can be absorbed by inhalation and orally. Known effects in humans are impairments in lung function. Pneumoconiosis or pleural changes are of minor importance. There is a suspected increased risk of laryngeal cancer (Hartwig 2015).

Portland cement dust is corrosive to the eyes and skin. On contact with the mucous membrane surface in the larynx and respiratory tract, the highly alkaline calcium hydroxide may be formed due to the high calcium oxide content, leading to mucous membrane irritation, severe burns, ulceration and necrosis (cement burn).

2 Mechanism of Action

See Hartwig (2015).

3 Toxicokinetics and Metabolism

There are no new data available.

4 Effects in Humans

The literature cited in this section contains only information on dust concentrations. In the production of cement (see below), a complex mixture of numerous hazardous substances has been detected in the emitted clean gas from the rotary kilns. This means that the health hazards of cement production are not fully taken into account by determining merely the level of cement dust in the air inhaled.

In one study, the exposure to dust in 9 Portland cement factories in Korea was evaluated in the period from 1995 to 2009. For this purpose, 2370 personal air samples were analysed. The level of exposure to total dust in all production areas was, on average, 2.29 ± 2.70 mg/m^3. The highest level of exposure to total dust was determined in 1995 at 11.26 ± 7.67 mg/m^3. Work areas with higher exposure levels throughout this period include: cement milling (3.28 ± 3.67 mg/m^3), packaging (2.93 ± 2.48 mg/m^3), loading (2.73 ± 2.68 mg/m^3) and crushing (2.56 ± 2.79 mg/m^3) (Koh et al. 2015).

In addition to cement dust, the following compounds were determined in 2017 in emissions from cement plants in Germany (clean gas from rotary kilns), for which the provisions of the Federal Immission Control Act (*Bundes-Immissionsschutzgesetz*) apply: nitrogen oxides (up to 500 mg/m^3), sulfur dioxide (up to 3250 mg/m^3), mercury, ammonia (up to 1080 mg/m^3), carbon monoxide, gaseous inorganic chlorine and fluorine compounds, polycyclic aromatic hydrocarbons, benzene, toluene, ethylbenzene, xylene, polychlorinated biphenyls and, mainly in very low concentrations, polychlorinated dibenzo-*p*-dioxins and dibenzofurans (VDZ 2018).

4.1 Single exposures

There are no data available.

4.2 Repeated exposure

A cross-sectional study of 227 male workers from a cement factory in the United Arab Emirates investigated the association between exposure to cement dust and lung disease. There were 149 exposed workers engaged in production at the crushing machines (n = 18), raw mills (n = 28), kilns (n = 27), cement mills (n = 33) and in packaging (n = 43). The control group consisted of 78 workers employed in administration, finance and other departments at the factory who were not exposed. Personal air samples were taken for randomly selected workers in production to determine their exposure to total dust. Demographic data, the work history, the use of respirators (N95), smoking habits and

respiratory symptoms (coughing, sputum, chronic bronchitis, dyspnoea, the degree of dyspnoea, diagnosed asthma, the occurrence of wheezing/whistling sounds of the chest) were determined by means of a questionnaire. The majority of the workers (89.9% exposed; 93.4% not exposed) were Indians. The exposed workers were less educated and significantly younger on average (38.2 years old) than the workers not exposed (41.6 years old). Thirty percent of the exposed workers had been employed in the factory for at least 10 years and 52% of the workers not exposed had been employed for at least 5 years. There was no significant difference between the 2 groups in terms of smoking. Only 79.2% of the production workers wore the mandatory respirators occasionally to frequently. The mean concentrations of total dust determined in the person-related samples were 8.9 mg/m^3 in the entire production area (crushers 4.2 mg/m^3, kilns 5.3 mg/m^3, cement mills 7.0 mg/m^3, raw mills 12.8 mg/m^3, packaging area 15.2 mg/m^3). The mean cumulative dust levels were between 23.5 mg/m^3 × year at the crushing machines and 145.1 mg/m^3 × year in packaging. The calculation of "raw" (unadjusted) prevalence odds ratios (OR) yielded a positive exposure–risk relationship for the complaints and diseases coughing, dyspnoea and chronic bronchitis. After adjustment for smoking (as a dichotomous variable), statistically significant increased ORs were found for the association between exposure and coughing and sputum, as well as increased prevalences for wheezing, dyspnoea, the degree of dyspnoea, chronic bronchitis and asthma (Ahmed and Abdullah 2012). These effects were observed at high dust concentrations at the workplace, which, however, cannot be used to derive limit values.

In a cross-sectional study, 100 apparently healthy individuals at a cement factory in Pakistan (workers: 50 non-smokers aged 36.86 ± 1.50 years, controls: 50 non-smokers aged 37.80 ± 1.66 years) were investigated using spirometry. Lung function served as an effect parameter. Exposure was recorded only as the exposure duration. In workers employed for over 10 years, a significant reduction in forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) compared with the corresponding control group was found (Meo et al. 2013). Due to the insufficient description of the selection of the collective and a lack of exposure data, this study is not suitable for deriving a limit value for Portland cement dust.

Another study investigated the effects of exposure to cement dust on residents living near Indian cement factories (group 1: about 2 km away, group 2 as a reference: about 20 km away). Air quality was analysed from March to December 2011. The mean "respirable" dust concentration in group 1 was 664 µg/m^3 and 87.8 µg/m^3 in group 2. The values for the "non-respirable" dust fraction were 543 µg/m^3 and 80 µg/m^3. Health effects on residents (n = 2000; 1000 male, casual labourers and farmers; 1000 middle class females) were recorded by questionnaires. In the neighbouring region, the subjects exhibited the following symptoms much more frequently than the reference group: allergic reactions affecting breathing (96%), chronic bronchitis (57%), asthma (49%), emphysema (9%), pneumonia (21%), tuberculosis (19%), shortness of breath (96%), coughing (96%), wheezing (96%), chest pain (49%), irregular heart beat (51%), swelling in legs and feet (43%), high blood pressure (85%), eye irritation (97%), skin allergies (95%) and other symptoms. The serum concentrations of nitric oxide and reactive oxygen species were determined in 21 affected and 21 unaffected persons. The values for both parameters were higher in the region with greater exposure than in the reference region. The reported exposure data indicate high exposures to particulate matter, SO_2 and NO_2 (Mehraj et al. 2013). This study cannot be used to derive a limit value for Portland cement dust due to the heterogeneity of the exposure.

In a cross-sectional study of residents of a rural region in Korea living within 1 km (more exposed group: n = 318, 59% males and 41% females) and more than 5 km (less exposed group: n = 129, 65% men and 35% women) from a cement factory, the effects on the pulmonary system were investigated. The number of subjects who had valid pre- and post-bronchodilator test results decreased with increasing age, to 47% for male, over 70-year-old, more exposed residents. The mean FVC values in the post-bronchodilator tests were significantly lower within all age groups (up to 59 years, 60 to 69 years, > 70 years) in the more exposed group. There were no significant differences in FEV1 values among women in these age groups. Obstructive ventilatory impairment (FEV1/FVC < 0.7) occurred in 9.7% of subjects in the more exposed group (OR = 1.60; 95% CI (confidence interval): 0.70–3.65) and in 8.5% in the less exposed group, while restrictive ventilatory impairments (FEV1/FVC ≥ 0.7, FVC% predicted < 80%) were identified for 21.6% of subjects in the more exposed group (OR = 2.55; 95% CI: 1.37–4.76) and for 12.4% in the less exposed group. For obstructive ventilatory impairments, smoking habits played a statistically significant role (ex-smokers /smokers : OR = 3.10; 95% CI: 1.10–8.66). The proportion of persons with ventilatory impairment was higher (OR = 2.63; 95% CI: 1.5–4.61) in the more

exposed group (31.3%) than in the less exposed group (20.9%); this difference was statistically significant. In addition, men (OR = 3.30; 95% CI: 1.68–6.48; compared with women), elderly persons (60–69 years old OR = 2.92; 95% CI: 1.53–5.56; over 70 years old OR = 7.03; 95% CI: 3.71–13.32; compared with < 60 years old) and ex-smokers /smokers (OR = 1.44; 95% CI: 0.72–2.84) were more frequently affected. Within the study, the particle concentration in the air was determined on a total of 21 days (every 7 days) from June to October. The mean concentrations of the particulate fraction PM_{10} were 45.5 µg/m³ (95% CI: 37.8–53.3) in the more exposed group and 38.5 µg/m³ (95% CI: 32.3–44.7) in the less exposed group, whereas for the $PM_{2.5}$ fraction they were 25.5 µg/m³ (95% CI: 18.7–32.3) and 19.3 µg/m³ (95% CI: 14.1–24.6). More detailed information on the exposure levels and on the substances to which the residents were exposed is not available (Kim et al. 2015). This study shows effects, but due to obvious limitations in the quality of the lung function measurements and the heterogeneity of the exposure, they cannot be taken into consideration for the setting of a limit value.

A summary of 26 epidemiological studies was published in the form of a systematic review by Fell and Nordby (2017). The authors concluded that numerous studies have limitations which do not allow a clear recommendation for a limit value to be made. However, dose-dependent effects on lung function could be identified after exposure to cement dust in concentrations of more than 2.2 mg/m³ for respirable dust and 4.5 mg/m³ for total dust.

4.3 Carcinogenicity

A meta-analysis of the association between exposure to Portland cement dust and cancer included 14 cohort studies and 12 case-control studies. Among the studies considered are also the cohort studies by Koh et al. (2011, 2013) and Giordano et al. (2012) described below. In several places, the methodology of a systematic review was not adequately implemented: for example, the comparison of titles and abstracts as well as full texts was evidently not carried out, and there is no quality assessment of the studies included. Aggregate effect size estimates were calculated for different effect estimates (OR, standardized mortality ratio (SMR), standardized incidence ratio (SIR)), which reduces the effectiveness of the meta-analysis. As a result, the authors found an increased SIR for lung cancer that was borderline statistically significant (SIR = 1.32; 95% CI: 1.00–1.76). The SMR was not increased (SMR = 0.93; 95% CI: 0.62–1.39). In the case of laryngeal cancer, there was an OR of 1.12 (95% CI: 0.76–1.65) that was not statistically significant and a SIR of 1.03 (95% CI: 0.46–2.33) that was not statistically significant. A statistically significant association with Portland cement dust exposure was shown by the SIR for colon cancer (SIR = 1.38; 95% CI: 1.02–1.88). There was no statistically significant increase in the SMR (SMR = 1.05; 95% CI: 0.79–1.40). Risk estimates around 1 are obtained for cancer in all instances. The authors point out the heterogeneity of the studies in several evaluations (Cohen et al. 2014). Overall, the review, which has methodological shortcomings, does not find a clearly increased cancer risk following exposure to cement dust. However, the increased risks found in some analyses should be subjected to further investigation in future studies.

4.3.1 Case–control studies

There are no new data available.

4.3.2 Cohort studies

In a retrospective cohort study of male workers in Korean Portland cement factories, the association between exposure to Portland cement dust and the incidence of cancer was investigated. For this purpose, a cancer mortality cohort (n = 5146), consisting of workers employed for at least 1 day in the period from 1992 to 2007 in 1 of 4 Portland cement factories, and a cancer incidence cohort (n = 5596), consisting of workers employed for at least 1 day in the period from 1988 to 2005 in 1 of 6 Portland cement factories, were used. Data for the Korean male population from the same periods were used as a reference. Information regarding the work activity and duration of employment was obtained from the companies, although no employment history was reported. During the quarry process, the workers were exposed mainly to limestone dust, and during the kiln-to-packing process, mainly to cement dust, often containing chromium(VI). The workers were divided into the exposure groups quarry, production (which includes working on all production processes up to packaging), maintenance and laboratory (chemical analysis of the cements and by-products). The proportion of workers in production was high in both cohorts at around 40%. The SMR for all causes of mortality

was 0.83 (95% CI: 0.68–1.01), which was lower than that of the general population, while the SIR for all cancers was the same (SIR = 1.01; 95% CI: 0.87–1.18). There was no statistically significant increase in mortality from respiratory cancers (SMR = 1.25; 95% CI: 0.66–2.13). There was a statistically significant increase in the incidence of stomach cancer in the production group (SIR = 1.56; 95% CI: 1.02–2.26) and a not statistically significant increase in the maintenance group (SIR = 1.46; 95% CI: 0.67–2.76). In the latter group, the slightly increased incidence of cancer of the liver, bile duct and gall bladder was likewise not statistically significant (SIR = 1.68; 95% CI: 0.77–3.18). Data for the smoking habits of the workers or exposure data were not available in the study (Koh et al. 2011). Therefore, the results are difficult to interpret.

In a retrospective cohort study, the data from 1324 male workers (19–72 years old, mean age 39.9 ± 17.5 years) from 2 Portland cement factories in North Choongchung, Korea, who were employed from 1997 to 2005, were included. Two of the 6 cement factories of the aforementioned cohort study by Koh et al. (2011) were selected on account of the occupational histories and exposure data that were available in these factories. Here, investigations were conducted twice a year by an external company; the data from the period 2004 to 2008 in both factories were used to determine the cumulative dust exposure. For this purpose, personal air monitoring was carried out. The workers were divided into the exposure groups quarry, raw mill, kilns/cement mill/packing, maintenance and laboratory. Office workers in the factories served as the reference group. A total of 52 cancer cases were found with a total number of person-years of 11 243. The dust exposure in the kiln/cement mill/packing area was highest with a median total dust concentration of 2.36 ± 2.17 mg/m^3 (factory A) and 2.33 ± 2.19 mg total dust/m^3 (factory B). The median cumulative dust exposure was determined, and 11.61 mg/m^3 × year was used as the cut-off criterion for classification as high or low exposure. The incidences for all cancers and for rectal carcinomas were increased in a statistically significant manner (SIR = 1.35; 95% CI: 1.01–1.78 and SIR = 3.05; 95% CI: 1.32–6.02, respectively), but in the individual groups (low exposure: SIR = 1.23; 95% CI: 0.69–2.03 and SIR = 3.66; 95% CI: 0.75–10.69; high-exposure: SIR = 1.41; 95% CI: 0.99–1.95 and SIR = 2.78; 95% CI: 0.90–6.48) the incidences were not statistically significant. There was also an increased incidence of stomach cancer (statistically significant; SIR = 2.18; 95% CI: 1.19–3.65) and cancers of the respiratory tract (not statistically significant; SIR = 1.50; 95% CI: 0.55–3.26) in the group of highly exposed workers. The authors state, as shortcomings of the study, that no information was available for the smoking habits of the workers and for other confounders such as Helicobacter pylori infection. In addition, the classification of the exposure groups was based on the cumulative dust exposure over the preceding 5 years and thus does not reflect the course of exposure over time (Koh et al. 2013, 2015). The study is limited by a relatively small number of cases. The result for stomach cancer is based on 14 cases. Further studies are needed to clarify these results.

A cohort study was conducted to investigate the effects of exposure to Portland cement dust on 748 male workers employed in a cement factory in Rome between 1940 and 2006. Portland cement production included all process steps from extraction of the raw material to cement retail. The workers were divided into the following groups: high exposure, unskilled workers (87%, n = 652) directly involved in cement production for at least 10 years; low exposure, technician engineers (3%, n = 19); administrative workers and professional engineers (10%, n = 77). Population data from the Lazio region were used as a reference group. For 39 workers, previous exposure to asbestos could not be excluded. 280 workers from the study period had already died; in 9 cases (3%) the cause of death could not be clarified. The main causes of death included diseases of the cardiovascular system (43%), respiratory system (12%), digestive system (6%), cancer (24%), endocrine, metabolic and nutrition-related diseases (4%), external injuries (4%) and other causes (4%). Cohort all-cause mortality (SMR = 0.87; 95% CI: 0.77–0.98), mortality from all cancers (SMR = 0.64; 95% CI: 0.48–0.82), mortality from cancers of the lips, oral cavity and pharynx (SMR = 0.59; 95% CI: 0.39–0.92) and larynx/trachea/bronchus/lungs (SMR = 0.56; 95% CI: 0.32–0.89) were significantly lower compared with the incidences in the reference group. The authors suggest this may be the result of a possible healthy worker effect. However, the SMRs for malignant neoplasms of the lymphoid/haematopoietic system (SMR = 1.42; n = 9) and for respiratory diseases (SMR = 1.41; n = 29) were increased among the cement factory workers. Of the 9 workers who died of malignant tumours of the lymphoid/haematopoietic system, 8 workers had been highly exposed to Portland cement dust. One worker had previously been employed in a cement/asbestos production factory. Data for smoking habits were not available (Giordano et al. 2012). This study does not contain information on exposure levels.

5 Animal Experiments and in vitro Studies

There are no new data available.

6 Manifesto (MAK value/classification)

Critical effects of Portland cement dust are irritant and inflammatory reactions, especially in the upper respiratory tract.

MAK value. A NOAEC (no observed adverse effect concentration) for Portland cement dust cannot be derived from the available epidemiological studies. It is therefore not possible to establish a MAK value.

Carcinogenicity. Animal studies of the carcinogenic effects of Portland cement dust are not available. From epidemiological studies, there is evidence of an increased risk of laryngeal cancer (Hartwig 2015) in the occupational group of construction workers who were exposed to Portland cement dust and also to lime(stone). The fact that the workers were probably also exposed to other carcinogens must be taken into account. The mechanism of action of tumour formation is not known. Since a suspected carcinogenic effect of Portland cement dust cannot be excluded, classification in Carcinogen Category 3 B has been retained.

Germ cell mutagenicity. As no new studies are available, Portland cement dust has not been classified in one of the categories for germ cell mutagens.

Notes

Competing interests

The established rules and measures of the Commission to avoid conflicts of interest (www.dfg.de/mak/conflicts_interest) ensure that the content and conclusions of the publication are strictly science-based.

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