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Effect of Cement Dust Components on Human Health

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Abstract

The process of cement manufacture is consistently associated with the generation of particulate matter, leading to the potential occupational exposure of factory personnel to cement dust. While it is advisable to utilize protective equipment, data from developing nations suggest that industrial establishments often fail to implement adequate safety precautions. The presence of hazardous elements such as aluminum, silica, and chromium in cement dust encountered in occupational settings has the potential to induce inflammatory alterations in vital organs, including the liver, skin, and lungs. Chronic exposure to aluminum has been observed to induce lipid peroxidation, which has been associated with the development of kidney failure and toxic alterations in the neurological system. Both Portland cement dust as a whole and its individual constituents are categorized as chemical risks. Individuals are subject to exposure to cement dust during several stages of its life cycle, including production, transportation, and construction sites.

Keywords: Cement dust, aluminum, silica, chromium

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1. Introduction

The global incidence of morbidity and mortality associated with occupational diseases is on the rise, despite the fact that these conditions are avoidable. According to research published by the International Labor Organization in 2013, it was observed that an annual total exceeding 2.3 million individuals succumb to occupational ailments [1]. The usage of Portland cement as a construction material in building practices dates back to approximately 25 BC, as documented by Vitruvius Pollio. Portland cement is comprised of calcium oxide, silicon dioxide, aluminum trioxide, ferric oxide, and chromates. The user's text is already academic in nature. The cement particles exhibit an aerodynamic diameter that spans from 0.05 to 10µm. These particulates mostly gain entry into the human body through the processes of inhalation and ingestion [2].

The process of cement manufacture is consistently associated with the generation of particulate matter, leading to the potential occupational exposure of factory personnel to cement dust. While it is advisable to utilize protective equipment, data from developing nations suggest that industrial establishments often fail to implement adequate safety precautions. Research has demonstrated that the extent of pulmonary function impairment is contingent upon the duration of exposure. Nevertheless, there exist stories that challenge this assertion. The findings of these papers indicate that there is no evidence to support the notion that exposure to cement dust leads to an increase in the occurrence of respiratory disorders or a higher incidence of respiratory symptoms among employees [3].

2. Effect of Cement Dust Exposure on Human Health

Workers in this field run the danger of being exposed to physical, chemical, and mechanical elements that could be harmful to their health. Workers may be exposed to cement by inhalation, skin, or ocular contact. The length, intensity, and sensitivity of the exposure determine the risk of harm associated with cement workers. The skin, eyes, and respiratory tract mucous membranes might get irritated by cement dust. It builds up in the respiratory system, which results in a response that raises the pH levels, irritating the mucous membranes that are exposed [4]. Because the majority of occupational diseases among cement workers develop over a longer period of time, it is challenging to gauge the severity of work-related disorders. Most employees in the cement industry have chromium allergies, which can cause anything from minor skin rashes to serious skin ulcers. Additionally, chronic sinusitis, bronchitis, and cough are more common in those workers. Portland cement is thought to be a potential contributor to occupational lung illnesses because cement particles are of a size that can be inhaled [5].

Employees at a Portland cement manufacturing plant commonly report acute ocular irritation and a heightened incidence of respiratory symptoms. Additionally, these workers exhibit diminished forced vital capacity (FVC),

forced expiratory volume in one second (FEV1), and FEV1/FVC ratio. Furthermore, there is an observed correlation between these health effects and an elevated daily mortality rate among the affected individuals [6].

The inhalation of cement dust has been associated with the development of pneumoconiosis, as well as an increased risk of lung, stomach, and colon cancer. Additionally, it is worth noting that this particulate matter has the potential to infiltrate the systemic circulation, thereby exerting its influence on many organs such as the heart, liver, bone, spleen, muscles, and hair. Furthermore, it has the capacity to disrupt the microstructure and physiological functioning of these organs. There is a limited body of research examining the biomarkers associated with the inflammatory response among individuals employed in the cement industry, and the findings from these studies lack clarity [7].

Research has demonstrated that prolonged exposure to cement dust is associated with a decrease in lung function, a notable rise in Total Leukocyte Count, the occurrence of chest pain, coughing, and ocular complications. There is an observed elevation in the levels of erythrocyte sedimentation rate (ESR), aspartate transaminase (AST), alanine transaminase (ALT), serum creatinine, and urea [8]. Workers in cement industry who at high risk of lung inflammation must do routine chest X ray and laboratory investigation for early diagnosis of any lung impairment [9].

3. Pathogenesis of Lung Diseases Caused Due to Cement Dust Exposure

3.1. Inflammatory Response of the body against cement dust

Within the human body, inflammation serves as a vital defense mechanism, wherein cells and molecules of the immune system collaborate to establish a network aimed at eliminating harmful substances and safeguarding the overall well-being of the organism. Nevertheless, the presence of chronic and prolonged inflammatory processes can lead to the deterioration of tissues as a consequence of an impaired equilibrium between immunocytes and molecules. The inflammatory response entails the initiation of a signaling cascade, which subsequently regulates the production of inflammatory mediators within the tissue's local cells and facilitates the recruitment of inflammatory cells from the bloodstream [10]. The presence of hazardous elements such as aluminum, silica, and chromium in cement dust encountered in occupational settings has the potential to induce inflammatory alterations in vital organs, including the liver, skin, and lungs. Chronic exposure to aluminum has been observed to induce lipid peroxidation, which has been associated with the development of kidney failure and toxic alterations in the neurological system [11].

Aluminum has been found to exhibit inhibitory effects on enzymes such as alkaline phosphatase, phosphodiesterase, and hexokinase. The inhalation of hexavalent chromium may lead to the development of asthma, nasal congestion, and itching in the nasopharyngeal region. Direct contact with the skin can result in burns, while ingestion of hexavalent chromium can cause harm to the liver, kidneys, and gastrointestinal system. Numerous studies have investigated the systemic impacts of cement dust and its hazardous components [12].

4. Effect of Cement Dust Component on Lungs

Chronic bronchitis, frequently accompanied by emphysema (chronic obstructive pulmonary disease or COPD), has been identified as the predominant respiratory ailment resulting from exposure to cement. Subsequently, silicosis and mixed dust fibrosis have been documented as subsequent respiratory diseases in terms of prevalence [12]. Microelements such as chromium found in cements are categorized as carcinogenic substances. Extensive research has demonstrated a robust correlation between exposure to cement dust and an increased risk of developing laryngeal carcinoma. Concrete workers exhibited an elevated relative risk for the development of lip, stomach, lung, and prostate cancers. Prolonged industrial exposure to cement is associated with elevated levels of DNA damage and suppression of repair mechanisms. The results of this study indicate the need for ongoing biomonitoring of occupational cohorts to evaluate their overall health state [10].

4.1. Silicosis

Silicosis is a type of pulmonary ailment that arises as a consequence of occupational exposure to particulate matter composed of crystalline silica. The condition is characterized by the presence of inflammation and scarring, which manifest as nodular lesions primarily located in the upper lobes of the lungs. This condition can be classified as a form of pneumoconiosis. Silicosis, specifically the acute variant, is distinguished by symptoms such as dyspnea, coughing, pyrexia, and cyanosis, which manifests as a bluish discoloration of the skin. Misdiagnosis of the condition in question can frequently occur, as it is easily mistaken for pulmonary edema, pneumonia, or tuberculosis [13].

4.1.1. Etiology

There are two distinct types of silicon dioxide that occur naturally: amorphous and crystalline. Silicon dioxide in its crystalline state is a prevalent naturally occurring mineral, frequently encountered in many substances including sandstone, quartz, and granite. Although the inhalation of the amorphous form does not seem to result in clinically relevant difficulties, the inhalation of the crystalline molecule has the potential to induce lung illness [13]. The presence of silica in the airways leads to the generation of reactive oxygen species upon interaction with the surfaces of the alveoli and endobronchial passages. Phagocytosis is the process by which macrophages internalize smaller particles, leading to the subsequent generation of more free radicals. The activation of macrophages and the presence of silica particles lead to oxidative damage, which then triggers the production of inflammatory cytokines, enhances cell signaling, and induces death in both parenchymal cells and macrophages. As the disease advances, there is a manifestation of fibroblast infiltration in a nodular form [14].

4.1.2. Epidemiology

Based on data provided by the Occupational Safety and Health Administration (OSHA), it has been determined that a considerable number of workers, over two million, are consistently subjected to various forms of respirable silicon dioxide. Enhanced consciousness and implementation of safety protocols within occupational settings have resulted in a significant decline of this particular ailment over the past few decades. Nonetheless, it is important to acknowledge that

existing preventive measures are not flawless, and instances of silicosis still emerge [15].

4.1.3. Pathophysiology

The inhalation of crystalline silicon dioxide leads to the formation of mineral deposits in the terminal bronchioles and alveoli. The activation of alveolar macrophages and the direct harmful effects on the surrounding lung parenchyma are consequences of the presence of foreign particles. The production of inflammatory cytokines, such as IL-1 and TNF-alpha, the creation of free radicals, and the amplification of cell-signaling pathways are consequences of cellular damage. Various cytokines contribute to the stimulation of fibrosis [16]. The involvement of tissue mast cells has also been documented. Moreover, there exists data indicating that silica hampers the capacity of macrophages to impede the proliferation of mycobacteria. This phenomenon serves as an explanation for the frequently seen correlation between silicosis and tuberculosis. In the acute manifestation of silicosis, there is a potential occurrence of direct toxicity affecting alveolar type 2 cells and macrophages. The involvement of immunologic components in the pathogenesis of silicosis has been hypothesized for a considerable period, however empirical evidence to substantiate this claim remains elusive [17].

4.2. Chromium

Chromium has corrosive properties, cytotoxicity, and carcinogenicity in humans, hence leading to the development of acute and chronic lung tissue toxicity [18].

4.2.1. Health Hazards of Chromium

The respiratory tract is the primary organ affected by the toxic effects of hexavalent chromium (Cr (VI)), which can occur through both acute (short-term) and chronic (long-term) inhalation. Acute exposure to Cr (VI) may result in symptoms such as dyspnea, coughing, and wheezing. Prolonged exposure to some substances can result in the development of ulcerations and perforations in the nasal septum, chronic bronchitis, reduced pulmonary function, pneumonia, and several other respiratory consequences. Based on empirical and epidemiological evidence, the International Agency for Research on Cancer (IARC) has designated Cr (VI) as a Group 1 carcinogen, indicating its recognition as a confirmed human carcinogen [19].

4.2.2. Pathophysiology

The exact mechanism underlying the harmful effects of Cr (VI) remains incompletely elucidated; nonetheless, multiple investigations have provided evidence that Cr (VI) compounds elicit oxidative stress, DNA damage, apoptotic cell death, and perturbations in gene expression. The presence of redox-active enzymes and small molecules leads to a decrease in Cr (VI) levels. This reduction process results in the formation of intermediate unstable states, namely Cr(V) or Cr (IV). These states have the potential to facilitate the production of free radicals, including hydroxyl, thiyl, ascorbate, and carbon-based radicals. These radicals have the ability to cause damage to macromolecular targets, such as DNA. It is noteworthy that Cr (III) exhibits reactivity towards DNA and proteins; nonetheless, it demonstrates an inability to traverse cellular membranes [20].

In contrast, Cr (VI) species have a distinct behavior as they do not exhibit reactivity towards nucleophilic targets. However, they possess the ability to readily traverse cell membranes via anion channels. Upon entry into the cellular environment, hexavalent chromium (Cr (VI)) undergoes a fast reduction process, resulting in the formation of trivalent chromium (Cr (III)). Subsequently, Cr (III) engages in interactions with cellular proteins and DNA [21]. The implementation of enhanced work environments, protocols, and hygiene practices has effectively mitigated occupational contact with Cr (VI) compounds, resulting in a decrease in conventional pulmonary complications like tracheobronchitis or pneumonia. Nevertheless, prolonged exposure to Cr (VI) still poses a risk for the development of airway disorders, encompassing airway irritation, sensitization, and lung cancer. Hence, it is imperative to employ tests of high sensitivity in order to assess the initial biochemical alterations that take place in the respiratory system subsequent to exposure to Cr (VI) [22].

4.3. Aluminum

While aluminum is generally not regarded as a highly dangerous material, there have been documented associations between its exposure and the development of pneumoconiosis under specific circumstances. Multiple research projects have demonstrated that the inhalation of aluminum (Al) dust and fumes has the potential to adversely affect pulmonary function. However, it is important to note that conflicting findings have also been reported in certain investigations. It has been proposed that all employees may be susceptible to respiratory disorders and deterioration of ventilatory function testing, ultimately leading to the development of chronic obstructive pulmonary disease (COPD). The precise etiology of pulmonary damage resulting from aluminum exposure remains uncertain [23]. Aluminum has the potential to enhance oxidative and inflammatory stress, which can result in the disruption of lung epithelial function. Chronic inflammation of the lungs seems to play a role in the development of chronic obstructive pulmonary disease (COPD), and indicators of this mechanism show potential as predicting factors in COPD. The potential utilization of serum A1AT enzyme as a genetic marker for the early detection of disease and prognosis assessment in individuals with chronic obstructive pulmonary disease (COPD) has been suggested. The clinical significance of alpha-1 antitrypsin (A1AT) becomes evident in persons with hereditary A1AT deficiency, as they demonstrate a heightened vulnerability to chronic inflammatory disorders such as chronic obstructive pulmonary disease (COPD), liver illnesses, and sporadically systemic vasculitis [24]. The potential of Al to enhance oxidative and inflammatory stress may result in a higher susceptibility to asthma and COPD in patients with A1AT deficiency. The serum concentration of C-reactive protein (CRP), a widely employed biochemical marker, exhibits a positive association with the extent of pulmonary inflammation in individuals with stable chronic obstructive pulmonary disease (COPD). The correlation between increased levels of C-reactive protein (CRP) and decreased lung function indices was identified in population-based surveys. Hence, there exists the potential for utilizing CRP as a predictive tool for assessing the likelihood of future chronic obstructive pulmonary disease (COPD) occurrences [25].

5. Conclusion

Inhalation of cement dust and its components affect human health badly mainly lung function.

References

[1] F.R. Martins-Melo, M.J. Carneiro, A.N. Ramos, J. Heukelbach, A.L.P. Ribeiro, and G.L. Werneck. (2018). The burden of neglected tropical diseases in Brazil, 1990-2016: A subnational analysis from the Global Burden of Disease Study 2016. PLoS Neglected Tropical Diseases. 12(6): e0006559.

[2] A.F. Sosa Gallardo & J. L. Provis. (2022). Early-age characterization of Portland cement by impedance spectroscopy. Advances in Cement Research. 34(12): 542-559.

[3] N. Mohamad, K. Muthusamy, R. Embong, A. Kusbiantoro, and M.H. Hashim. (2022). Environmental impact of cement production and Solutions: A review. Materials Today: Proceedings. 48: 741-746.

[4] T. Kowalik, D. Logoń, M. Maj, J. Rybak, A.Ubysz, and A. Wojtowicz. (2019). Chemical hazards in construction industry. In E3S Web of Conferences. EDP Sciences. 97: 03032.

[5] N.H. Kamaludin, J. Jalaludin, S.B.M. Tamrin, A.M. Akim, T. Martiana, N. Widajati. (2020). Exposure to silica, arsenic, and chromium (VI) in cement workers: a probability health risk assessment. Aerosol and Air Quality Research. 20: 2347-2370.

[6] A. Hartwig, M. Arand, and M.A.K. Commission. (2022). Portland cement dust. MAK Value Documentation, supplement–Translation of the German version from 2020. The MAK Collection for Occupational Health and Safety. 7(3): Doc051.

[7] J.O. Jaber and S.D. Probert. (1999). Environmental-impact assessment for the proposed oil-shale integrated tri-generation plant. Applied Energy. 62(3):169-209.

[8] C.O. Okonkwo, C.E..Ugwu, A.C. Anakor, C.C. Dike, and E. Nwobodo. (2015). The effects of cement dust on hematological parameters of cement workers in Asaba, Delta State, Nigeria. Journal of Environmental Science, Toxicology, and Food Technology. 9(1): 5-8.

[9] A.M. Hussein, I. Galal, M.M. Mohammed, H.K. Abd Elaal, and K. Aly. (2020). Is there a correlation between pulmonary inflammation index with covid-19 disease severity and outcome? SVU International Journal of Medical Sciences. 5(2): 35-.50

[10] L.T. Obaji-Ogar, A.C. Nsonwu-Anyanwu, and F.A. Odum, (2020). Oxidative DNA Damage and Pro-inflammatory Response in Chronic Exposure to Cement Dust. Asia Pacific Journal of Medical Toxicology. 9(1).

[11] M. Wang, Y. Yu, K. Dai, Z. Ma, Y. Liu, J. Wang, and C. Liu. (2016). Improved osteogenesis and angiogenesis of magnesium-doped calcium phosphate cement via macrophage-immunomodulation. Biomaterials Science 4(11): 1574-1583.

[12] O. Erhabor, B.I. Kebbe, I.I. Zama, N. Abdullahi, Y. Marafa, A.N. Okwesili. (2013). Effect of occupational exposure of cement dust on some haematological parameters of workers in a cement company in Sokoto, Nigeria. International Journal of Health Care and Medical Science. 1(7): 21-25.

[13] C. Nishida, K. Yatera. (2022). The impact of ambient environmental and occupational pollution on respiratory diseases. International Journal of Environmental Research and Public Health. 19(5): 2788.

[14] M.S. Mapa, R.S.H. Koralege. (2023). Silicosis and Asbestosis. Medical Geology: Enroute to One Health. 14: 83-94.

[15] M.P. Rupani. (2023). Challenges and opportunities for silicosis prevention and control: need for a national health program on silicosis in India. Journal of Occupational Medicine and Toxicology. 18(1): 1-12.

[16] S.V. Cherian, A. Kumar, P.M. de Groot, M.T. Truong, and C.A. Moran. (2023). Pneumoconiosis. In The Thorax: Medical, Radiological, and Pathological Assessment. Cham: Springer International Publishing. 693-710.

[17] J. Sui, Y. Ding, M. Chen, G. Wang, C. Ding, S. Liu, and H. Zhang. (2023). BMP4 Peptide and Enoxacin-Loaded Mesoporous Silica Nanoparticles Direct Cell Fate and Boost Bone Regeneration During Infection. Materials & Design.7: 112066.

[18] P.B. Tchounwou, C.G. Yedjou, A.K. Patlolla, and D.J. Sutton. (2012). Heavy metal toxicity and the environment. Molecular, Clinical, and Environmental Toxicology. 3: 133-164.

[19] K. Yatera, Y. Morimoto, S. Ueno, S. Noguchi, T. Kawaguchi, F. Tanaka, and T. Higashi. (2018). Cancer risks of hexavalent chromium in the respiratory tract. Journal of UOEH. 40(2): 157-172.

[20] Y. Li, Y. Zhang, F. Su, Y. Wang, L. Peng, and D. Liu. (2022). Adsorption behavior of microplastics on the heavy metal Cr (VI) before and after ageing. Chemosphere.1(302): 134865.

[21] F.M. Mitloehner and M.S. Calvo. (2008). Worker health and safety in concentrated animal feeding operations. Journal of Agricultural Safety and Health. 14(2): 163-187.

[22] K.P. Vlahovich, A. Sood. (2021). A 2019 update on occupational lung diseases: A narrative review. Pulmonary Therapy. 7: 75-87.

[23] S. Onutai, S. Jiemsirilers, P. Thavorniti, and T. Kobayashi. (2015). Aluminium hydroxide waste based geopolymer composed of fly ash for sustainable cement materials. Construction and Building Materials. 101: 298-308.

[24] E.B. da Costa, E.D. Rodríguez, S.A. Bernal, J.L. Provis, L.A. Gobbo, A.P. Kirchheim. (2016). Production and hydration of calcium sulfoaluminate-belite cements derived from aluminum anodizing sludge. Construction and Building Materials. 122:373-383.

[25] H. Kinoshita, P. Swift, C. Utton, B. Carro-Mateo, G. Marchand, N. Collier, and N. Milestone. (2013). Corrosion of aluminum metal in OPC-and CAC-based cement matrices. Cement and Concrete Research. 1(50): 11-18.