Research Square

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Alteration of Hematological indices among cement industry workers due to reductive variation of Cardio-respiratory parameters

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Research Article

Keywords: Cement Dust Exposure, Occupational Health, Pulmonary Function Test, Hematological Parameters, Cement Industry Workers, Respiratory Impairment

Posted Date: November 4th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11290355/v1

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Additional Declarations: No competing interests reported.

Abstract

Cement dust exposure is a major occupational health concern among cement industry workers and may contribute to respiratory and systemic health disorders. This cross-sectional study assessed the effects of chronic cement dust exposure on pulmonary and hematological parameters among cement industry workers aged 20–55 years. A total of 90 participants were enrolled, including 45 exposed workers and 45 age-matched controls. Data were collected through structured questionnaires, clinical assessment, pulmonary function testing, and laboratory investigations. Pulmonary function analysis revealed reduced Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second (FEV_1) among exposed workers, although the differences were not statistically significant. Peak Expiratory Flow Rate (PEFR) was significantly decreased ($p = 0.0089$), indicating impaired respiratory function. Hematological findings demonstrated significant alterations in packed cell volume (PCV), RBC count, platelet count, monocyte count, MCH, and MCHC. The findings suggest that long-term occupational exposure to cement dust may adversely affect respiratory function and hematological status. Regular health surveillance, use of personal protective equipment, and workplace dust control measures are recommended to minimize occupational health risks.

INTRODUCTION

Chronic Respiratory diseases are the most common non- communicable disease worldwide, mainly due to the widespread nature of harmful environmental, occupational and lifestyle respiratory exposures [1].Chronic respiratory diseases (CRDs) are long-term and often progressive disorders that affect the airways, lung tissue, or both. These diseases interfere with normal breathing by limiting airflow, damaging lung structures, or causing long-term inflammation [2].

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by a history of respiratory symptoms such as wheeze, shortness of breath, chest tightness, and cough that vary over time and in intensity, together with variable expiratory airflow limitation [3]. Chronic Bronchitis is defined by the presence of chronic productive cough and is involves in bronchial inflammation, leading to mucosal thickening, goblet cell hyperplasia and produce excess mucus that narrows the airway lumen [4]. Restrictive lung disease damages the lung parenchyma that causes inflammation and fibrosis of the interstitium that reduces lung compliance and restrict the ability of the lungs to expand fully [5]. Pulmonary fibrosis is the classic restrictive lung disease characterized by progressive lung scarring which thickens the alveolar walls, impairing the diffusion of oxygen from the air into the blood stream and is the most common form of idiopathic pulmonary fibrosis (IPF) for which the cause is unknown [6]. Pulmonary hypertension is defined by an increase mean pulmonary arterial pressure due to vascular obstruction or remodelling, which increases the resistance to blood flow through the lungs. This increases the right ventricle after load and potentially leading to the right- sided heart failure [7]. Pulmonary hypertension can be an idiopathic condition including congenital heart disease, connective tissue disease, and chronic lung disease like COPD and pulmonary fibrosis [3]. Airflow obstruction is measured by spirometry, as it is most reliable widely used lung function test. In

COPD, airflow obstruction is caused by the mixture of small airways disease and parenchymal destruction; the contributions vary from person to person [8].

According to the global burden there is an increased number of CRD cases from 381.2 million in 1990 to 451.8 million in 2019, further rise to 569.2 million by 2023 while the death related to these disease increased from 3.0 million in 1990 to 4.3 million to 2019 and has reached to 4.2 million in 2023 [9, 10]. The increasing burden of CRDs risks factor associated with tobacco smoking, occupational dust exposure, gases, fumes, ambient PM pollution and household air pollution from solid fumes [11]. In 2021, smoking remained the primary cause to global DALY (disability-adjusted life year) burden, accounting for 341.7 per 100,000.Household air pollution from solid fuels 181.2 per 100,000. Occupational exposure to particulate matter, gases and fumes contributed the least to the global DALY burden, with a rate of 148.7 per 100,000 [9]

India faces a particularly acute challenge regarding occupational respiratory disease. The country's rapid industrialization over recent decades, while driving economic growth, has simultaneously expanded worker exposure to hazardous airborne contaminants across numerous industrial sectors [12]. The prevalence of occupational lung disease in India is estimated to affect millions of workers, yet remains significantly underreported due to limited surveillance systems, inadequate occupational health infrastructure, poor awareness among workers and employers, and gaps in regulatory enforcement [13].India is currently a home to an estimated 33 million patient with asthma and 55 million patients with chronic obstructive pulmonary disease, accounting for over 10% of the global burden for these illnesses [14]. As per the global burden of COPD study, India ranks second number globally. Air pollution (house hold and outdoor) contributes to over 50% of the national COPD, while tobacco smoking contributes to 20% – 25% [15].

Prolonged inhalation to cement dust cause changes in systemic circulation and spread to the different organs of the body effecting the tissue like heart, liver, spleen, etc. ultimately leading to the alteration in microstructure and physiological performance Exposure to cement dust causes haematological and cytogenic damage among workers [16]. The human hematopoietic system is sensitive to environmental factors due to rapid synthesis and destruction of cells with heavy metabolic demand [17]. Hematological parameters and differential white blood cells are sensitive and can provide important diagnostic and prognostic tool regarding the toxicity associated with cement dust exposure [18].

The present research focuses on combining hematological, and inflammatory markers to better understand the patterns and progression of chronic respiratory diseases. This study aims to provide a clearer and more detailed understanding of the basic mechanisms of the disease and their importance in patient care.

METHODOLOGY

Study site: Cement industry (East Jaintia Hills), Meghalaya, India

Sample size:

Exposed group

45(sample size is obtained from the references of the exposure study group in a small and clustered of population, for the current study additionally 5% number of sample included against the standard sample size) [19]

Control group

45(sample size is obtained from the references of the exposure study group in a small and clustered of population, for the current study additionally 5% number of sample included against the standard sample size) [19]

Inclusion Criteria

Workers exposed to cement dust and have been employed in a cement factory for at least one year, within the age of 20 to 55 years.

Exclusion Criteria

Workers with exposure duration of less than six months to cement factory and having severe respiratory disorders.

Statistics analysis

Data were analysed by using the IBM 2023 Statistical Package for Social Sciences (SPSS 2023). Continuous variables were expressed as mean ± standard deviation (SD). Comparative analysis of pulmonary function and haematological parameters among cement industry workers and control group population were performed using independent t- test for normally distributed variables. A *p*- value of < 0.05 was considered statistically significant for all parameters.

RESULTS

The present study aim to evaluate the prevalence of Chronic Respiratory complications and its impact on hematological Parameters including Hemoglobin estimation, Packed Cell Volume, RBC Count, WBC Count, Platelet Count, Red Cell Indices and Differential Leucocyte Count among cement industry workers and to compare with control group population. A total of 90 samples was collected out of which 45 samples was collected from cement industry workers and 45 samples from control group population. All the samples were processed in Pathology laboratory and CIF laboratory of Assam down town University.

Table 1
Respiratory Complications among the Cement industry workers

Severity	FVC	FEV1	FEV1/FVC	PEFR
Mild	77.53 ± 8.40	93.32 ± 9.36	115.3 ± 8.62	78.84 ± 14.32
Moderate	122.00 ± 11.6	84.07 ± 6.87	66.28 ± 8.40	27.00 ± 3.71

Table 1 represents the spirometry parameters among cement industry workers categorized into the severity of respiratory impairment. Workers in the mild category showed moderately reduced respiratory function with FVC (77.53 ± 8.40), FEV1 (93.32 ± 9.36), FEV1/FVC (115.3 ± 8.62) and PEFR (78.84 ± 14.32). In the moderate category, FVC (122.00 ± 11.6), FEV1 (84.07 ± 6.87), FEV1/FVC ratio (66.28 ± 8.40) and PEFR showed a markedly reduction to 27.00 ± 3.71, indicating greater airflow limitation and impaired lung function.

Table 2
Distribution of Respiratory Complications among the Control group population

Severity	FVC	FEV1	FEV1/FVC	PEFR
Mild	87.44 ± 11.62	97.49 ± 13.75	110.50 ± 9.19	87.18 ± 12.17

Table 2 shows the spirometry parameters of the control group population. The pulmonary function test shows mild in FVC (87.44 ± 11.62), FEV1 (97.49 ± 13.75), FEV1/FVC ratio (110.50 ± 9.19) and PEFR (87.18 ± 12.17).

Table 3
Alteration of Hematological Parameters among the Cement industry workers

Sl No	Parameter	Mean ± SD	*p* value
1	Hemoglobin	15.50 ± 2.06	$p > 0.05$
2	PCV	45.60 ± 6.15	$p < 0.05$
3	RBC count	5.55 ± 0.78	$p < 0.05$
4	WBC count	7308.89 ± 2018.76	$p > 0.05$
5	Platelet count	2.42 ± 0.63	$p < 0.05$
6	Neutrophil	59.39 ± 8.49	$p > 0.05$
7	Lymphocyte	31.12 ± 8.03	$p > 0.05$
8	Eosinophil	4.71 ± 2.82	$p > 0.05$
9	Monocyte	4.75 ± 1.00	$p < 0.05$
10	Basophil	0.00 ± 0.00	Nan
11	MCV	82.55 ± 9.13	$p > 0.05$
12	MCH	24.54 ± 4.09	$p < 0.05$
13	MCHC	29.59 ± 2.32	$p < 0.05$
14	AEC	0.32 ± 0.16	$p > 0.05$

Table 3 analysis of erythrocyte-related parameters revealed significant increases in packed cell volume (PCV) and red blood cell (RBC) count by 12.36% and 11.35% respectively. Although hemoglobin levels decreased by 1.76%, the change was not statistically significant.

Assessment of leukocyte parameters showed no significant change in total white blood cell (WBC) count, which increased by 3.79% Neutrophil percentage decreased by 5.43% while lymphocyte and eosinophil percentages increased by 3.66% and 26.89% respectively; however, these changes were not statistically significant. In contrast, monocyte percentage exhibited a significant increase of 37.12% Basophil values remained unchanged, and therefore statistical analysis was not applicable.

A significant increase of 18.82% was observed in platelet count Among the red cell indices, mean corpuscular volume (MCV) showed a slight, non-significant increase of 1.29%.Conversely, mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) decreased significantly by 11.18% and 12.92% respectively. Absolute eosinophil count (AEC) increased by 26.79%, although this change did not attain statistical significance.

Table 4
Alteration of Hematological Parameters among the Control group population

Sl No	Parameter	Mean ± SD	*p* value
1	Hemoglobin	13.74 ± 2.02	$p > 0.05$
2	PCV	40.58 ± 6.23	$p < 0.05$
3	RBC count	4.98 ± 0.67	$p < 0.05$
4	WBC count	7042.22 ± 1656.46	$p > 0.05$
5	Platelet count	2.03 ± 0.63	$p < 0.05$
6	Neutrophil	62.80 ± 8.19	$p > 0.05$
7	Lymphocyte	30.02 ± 8.20	$p > 0.05$
8	Eosinophil	3.71 ± 2.90	$p > 0.05$
9	Monocyte	3.47 ± 1.22	$p < 0.05$
10	Basophil	0.00 ± 0.00	Nan
11	MCV	81.50 ± 7.94	$p > 0.05$
12	MCH	27.63 ± 3.81	$p < 0.05$
13	MCHC	33.98 ± 2.56	$p < 0.05$
14	AEC	0.25 ± 0.20	$p > 0.05$

Table 4 the hematological parameters showed varying degrees of change following the intervention. Hemoglobin levels showed a slight increase of 1.79% but this change was not statistically significant.A significant reduction was observed in PCV (− 11.00%) and RBC count (− 10.20%). Similarly, platelet count decreased significantly by (15.84%).Although WBC count decreased by 3.65%, the change was not statistically significant. Among the differential leukocyte counts, neutrophils showed a non-significant increase of 5.74%, while lymphocytes decreased by 3.53% and eosinophils decreased by 21.19% both non-significantly.A significant decrease was observed in monocyte count (− 27.07%). Basophil count remained zero throughout the study; therefore, statistical analysis could not be performed.Regarding red cell indices, MCV showed a slight non-significant decrease of 1.27%.In contrast, MCH increased significantly by 12.59% and MCHC increased significantly by 14.84%.The absolute eosinophil count (AEC) decreased by 21.13%, but this reduction was not statistically significant.

Overall, significant changes were observed in PCV, RBC count, platelet count, monocyte count, MCH, and MCHC, whereas the remaining hematological parameters did not show statistically significant alterations

Figure 1 the respiratory parameters of the cement industry workers were compared with the control group using as independent t- test. The mean of FVC and FEV1 in lower in the cement industry workers (80.84 ± 22.07 and 91.22 ± 22.13) as compared to the control group population (87.44 ± 11.62 and 97.49 ± 13.75), the difference is not statistically significant (p = 0.0804 and 0.1109).

Similarly the mean of FEV1/FVC ratio was slightly higher in cement industry workers (112.06 ± 11.75) compared to control group population (110.50 ± 9.19), the difference was not statistically significant (p = 0.4845). PEFR was significantly reduce in cement industry workers (77.69 ± 20.31) when compared to control group population (87.18 ± 12.17), and the statistically significant (p = 0.0089)

Figure 2 the different blood cells of the cement industry workers were compared with the control group by using independent t-test. The mean RBC count is higher (5.55 ± 0.78) than the control group population (4.98 ± 0.67), and the difference was statistically significant with *p*value (0.0004). Similarly the WBC count is higher in cement industry workers (7308.89 ± 2018.76) compared to the control group population (7042.22 ± 1656.46), showing a non-significant difference in *p*value (0.4952).

Platelet count is also higher in cement industry workers (2.42 ± 0.63) as compared to control group population (2.03 ± 0.63) and the difference is statistically significant, *p*value (0.0051).

Figure 3 the differential leucocyte count of cement industry workers were compared with the control grouppopulation by using independent t-test. The mean of neutrophil count was lower in cement industry workers (59.39 ± 8.49) when compared to control group population (62.80 ± 8.19), the difference was not statistically significant (p = 0.0557).

Similarly the mean of lymphocyte count and Eosinophil count and was higher in cement industry workers (31.12 ± 8.03) and (4.71 ± 2.82) when compared with control group population (30.02 ± 8.20) and (3.71 ± 2.90), the difference was not statistically significant with p value (0.5221 and 0.1017). The monocyte count was significantly elevated in the cement industry workers (4.75 ± 1.00) when compared to the control group population (3.47 ± 1.22) with p value (< 0.005).

Figure 4 the hematological parameters of cement industry workers were compared to the control group population using independent t- test. The mean hemoglobin concentration was slightly lower in cement industry workers (13.50 ± 2.06) as compared to control group population (13.74 ± 2.02), the difference was not statistically significant (p = 0.5742). The packed cell volume (PCV) was higher in cement industry workers (45.60 ± 6.15) compared to control group population (40.58 ± 6.23), and shows highly significant (p = 0.0002).

The MCV is slightly higher in cement industry workers (82.55 ± 9.13) as compared to control group population (81.50 ± 7.94), the difference was not statistically significant (p = 0.5615). The MCH and MCHC was lower in cement industry workers (24.54 ± 4.09 and 29.59 ± 2.32) when compared with control group population (27.63 ± 3.81 and 33.98 ± 2.56) the difference was highly significant (p = 0.0004 and < 0.005).

DISCUSSION

Cement manufacturing remains one of the most hazardous industrial occupations globally. Prolonged exposure to these airborne particulates has been associated with the development of CRD and systemic inflammation [19] The present study, conducted among cement industry workers of East Jaintia Hills, Meghalaya, aged between 20 to 55 years, total 90 samples were collected out of which 45 samples are collected from each group, aimed to characterized the prevalence of CRD and evaluate its impact on pulmonary function and haematological parameters.

Spirometric assessment in the present study revealed that cement workers exhibited significantly reduce PEFR compared to the control population while FVC and FEV1 were lower in workers [20, 21].The FEV1/FVC ratio was elevated in cement workers, suggesting possible restrictive impairment patterns rather than purely obstructive disease, the significantly reduced PEFR, along with lower FVC and FEV1, among cement workers may be associated with prolonged occupational exposure to cement dust, respirable crystalline silica, calcium oxide, and other fine particulate matter generated during cement production, handling, grinding, packing, and transportation [22, 23]. Because these particles are small enough to reach the conducting and lower respiratory airways, repeated inhalation can cause mechanical irritation of the airway epithelium, chronic inflammation, oxidative stress, increased mucus secretion, mucosal swelling, and gradual structural changes in the bronchi and bronchioles [24, 25]. These inflammatory changes can increase airway resistance and reduce the ability of the lungs to move air rapidly, which may particularly explain the significant reduction in PEFR, an important indicator of large- and medium-airway airflow [26]. Continuous exposure may also produce thickening of the airway walls, narrowing of smaller airways, reduced lung compliance, and early fibrotic changes in the lung tissue, thereby contributing to the reduction in FEV1 and FVC [27, 28]. The lower FEV1 indicates that the volume of air expelled during the first second of forced expiration is reduced, while the lower FVC suggests a reduction in the total volume of air that can be forcibly exhaled [29]. The elevated FEV1/FVC ratio in the workers may occur because FVC has decreased proportionally more than FEV1, which can be suggestive of a restrictive ventilatory tendency or reduced lung volume, rather than the classical obstructive pattern in which the FEV1/FVC ratio is reduced; however, true restriction should be confirmed by measuring total lung capacity [29]. In addition, chronic cement-dust exposure can activate inflammatory cells such as neutrophils and macrophages, leading to the release of inflammatory mediators and reactive oxygen species, which may cause epithelial injury and progressive remodeling of respiratory tissues [30, 31]. If exposure continues for a prolonged period, these changes may gradually result in persistent cough, phlegm production, wheezing, chest discomfort, exertional breathlessness, reduced exercise capacity, impaired oxygen transfer, and progressive occupational respiratory impairment [32]. In severe or long-term exposure, persistent inflammation and fibrosis may contribute to conditions such as chronic bronchitis, pneumoconiosis or silica-related lung disease, and other chronic respiratory disorders [33]. Therefore, the reduced PEFR and lower FVC and FEV1 observed in cement workers may represent early or developing pulmonary functional impairment resulting from repeated occupational dust exposure, emphasizing the importance of effective dust suppression, adequate ventilation, use of properly fitted respiratory protective equipment, reduction of exposure duration, and

periodic spirometric monitoring to detect and prevent further deterioration of lung function [23, 34]. Within the study group, severe respiratory cases demonstrated markedly reduced pulmonary function parameters compared to mild and moderate.

The significant hematological alterations observed among cement industry workers compared with the control group may be attributed primarily to prolonged occupational exposure to cement dust, respirable particulate matter, silica-containing particles, and other alkaline components, which can induce chronic respiratory irritation, systemic inflammation, oxidative stress, and physiological adaptation [19, 25] The significantly increased RBC count and PCV may reflect a compensatory response to repeated exposure to airborne dust and possible mild or intermittent impairment of pulmonary gas exchange; when tissue oxygen availability is reduced, the kidneys may increase erythropoietin production, stimulating erythropoiesis and consequently increasing RBC mass and PCV [35]. Dehydration, heat exposure, and physical workload in cement-processing environments may also contribute to hemoconcentration and increased PCV. Despite the increased RBC count, the reduction in MCH and MCHC indicates that individual red cells contain less hemoglobin and have a lower hemoglobin concentration, which may suggest hypochromic changes and altered iron availability/utilization, possibly related to chronic inflammation, nutritional factors, or occupational exposure-related oxidative stress [19, 35]. Thus, the combination of increased RBC and PCV with reduced MCH and MCHC may represent a compensatory erythropoietic response occurring alongside altered red-cell hemoglobinization. The significantly elevated platelet may be related to chronic low-grade inflammation caused by inhaled cement dust, because inflammatory cytokines can stimulate megakaryocyte activity and platelet production; increased platelet numbers may also contribute to a pro-thrombotic tendency if the inflammatory stimulus persists [36]. Although the total WBC count and differential leukocyte count (DLC) did not differ significantly between the groups, the significantly increased monocyte percentage is important because monocytes participate in the body's response to inhaled particulate matter and chronic tissue irritation [19, 31]. After entering tissues, monocytes can differentiate into macrophages and release inflammatory mediators and reactive oxygen species, contributing to persistent pulmonary inflammation and tissue injury [24, 30]. The absence of a significant change in total WBC or most DLC parameters may indicate that the exposure is producing a chronic, low-grade inflammatory response rather than an acute systemic infection or severe inflammatory reaction [31, 36]. If occupational exposure continues for many years, persistent oxidative stress and inflammation may contribute to progressive respiratory dysfunction, endothelial activation, altered blood rheology, and increased cardiovascular and thrombotic risk [37, 38].

Overall, the observed increase in RBC, PCV, platelets, and monocyte percentage, together with reduced MCH and MCHC, may reflect a combination of compensatory erythropoietic adaptation, chronic inflammatory stimulation, oxidative stress, and altered hemoglobinization associated with prolonged occupational cement-dust exposure [35] in a large cross-sectional study of cement dust exposed workers, who reported compensatory erythrocytosis with hypochromic red cell morphology. similarly noted acute effects of cement dust on haematological parameters, particularly reductions in MCH and MCHC[16].

CONCLUSION

The present study investigated the prevalence of chronic respiratory disease and its systemic impact on haematological parameters among cement industry workers of East Jaintia Hills, Meghalaya. The findings yield several important conclusions with implications for occupational health policy and future research.

The present study concludes that a significant reduction in PEFR among cement industry workers compared to the control group. Reduce in PEFR suggest that there is an impaired respiratory capacity and airflow limitation. In addition to respiratory changes, workers showed an alteration in blood parameters, indicating that cement dust exposure may affect the multiple organ system, while changes in haematological parameters reflect inflammatory and physiological responses to chronic occupational exposure among the cement industry workers.

The finding emphasizes the need to strengthen occupational safety practices in cement industries including dust suppression methods, proper work place ventilation, and regular use of personal protective equipments that can help to reduce dust inhalation and limit long- term health effects.

DECLARATIONS

Conflict of Interest

All authors have given the consent that there is no conflict of interest for the current research.

Funding Sources

No funding sources available for the current study.

Author Contribution

S.N. AND K.S. WROTE THE MAIN MANUSCRIPTS.P. AND S.N. PREPARED THE TABLES AND FIGURESJ.D. AND S.K.N. REVIEWED THE MANUSCRIPT

Acknowledgement

Authors are acknowledged the supportive arrangements of Assam down town University, Panikhaiti, Guwahati, Assam, India, for the completion for the current research in the AY 2025-2026.

REFERENCES

1. Labaki WW, Han MK (2020) Chronic respiratory diseases: a global view. Lancet Respir Med 8:531–533. https://doi.org/10.1016/S2213-2600(20)30157-0
2. da Cunha IN, Autry E, Bennara K, van Boven J, Fearn A, Garcia-Cardenas V, Kahlon A, Moranta F, Nduva Sammy R, Paulino E, Williams S, Zheng Kang L (2022) Chronic respiratory diseases: a handbook for pharmacists. International Pharmaceutical Federation (FIP), 106 pp. https://www.fip.org/file/5230
3. Saladi PA, Kamala GR, Ch H, Charishma S, Hansika BLS, Vijaya Durga M, Naveena P (2025) Pathophysiology and risk factors for chronic respiratory diseases: review article. J Pharma Insights Res 3:182–189. https://doi.org/10.69613/xpgxaq57
4. Zhang J, Wurzel DF, Perret JL, Lodge CJ, Walters EH, Dharmage SC (2024) Chronic bronchitis in children and adults: definitions, pathophysiology, prevalence, risk factors, and consequences. J Clin Med 13:2413. https://doi.org/10.3390/jcm13082413
5. Guler SA, Corte TJ (2021) Interstitial lung disease in 2020: a history of progress. Clin Chest Med 42:229–239. https://doi.org/10.1016/j.ccm.2021.03.001
6. Luppi F, Kalluri M, Faverio P, Kreuter M, Ferrara G (2021) Idiopathic pulmonary fibrosis beyond the lung: understanding disease mechanisms to improve diagnosis and management. Respir Res 22:109. https://doi.org/10.1186/s12931-021-01711-1
7. Naeije R, Richter MJ, Rubin LJ (2022) The physiological basis of pulmonary arterial hypertension. Eur Respir J 59:2102334. https://doi.org/10.1183/13993003.02334-2021
8. Eschenbacher WL (2016) Defining airflow obstruction. Chronic Obstr Pulm Dis 3:515–518. https://doi.org/10.15326/jcopdf.3.2.2015.0166
9. Cao Z, He L, Luo Y, Tong X, Zhao J, Huang K, Chen Q, Jiao L, Liu Y, Geldsetzer P, Yang T, Wang C, Bärnighausen TW, Chen S (2025) Burden of chronic respiratory diseases and their attributable risk factors in 204 countries and territories, 1990–2021: results from the Global Burden of Disease Study 2021. Chin Med J Pulm Crit Care Med 3:100–110. https://doi.org/10.1016/j.pccm.2025.05.005
10. Oh J, Kim S, Yim Y, Kim MS, GBD 2023 Global Chronic Respiratory Disease and Covid Collaborators, Hay SI, Shin JI, Yon DK (2026) Global, regional, and national burden of chronic respiratory diseases and impact of the COVID-19 pandemic, 1990–2023: a Global Burden of Disease study. Nat Med 32:197–223. https://doi.org/10.1038/s41591-025-04077-9
11. Shupler M, Godwin W, Frostad J, Gustafson P, Arku RE, Brauer M (2018) Global estimation of exposure to fine particulate matter (PM2.5) from household air pollution. Environ Int 120:354–363. https://doi.org/10.1016/j.envint.2018.08.026
12. Chakraborty D, Sahu S (2025) Prevalence of respiratory issues and intervention strategies among different informal sectors in India: a systematic review. Discov Public Health 22:359. https://doi.org/10.1186/s12982-025-00724-1
13. Mishra J, Acharya S, Taksande AB, Prasad R, Munjewar PK, Wanjari MB (2023) Occupational risks and chronic obstructive pulmonary disease in the Indian subcontinent: a critical review. Cureus 15:e41149. https://doi.org/10.7759/cureus.41149

14. Garud A, Biswas D, Moitra S, Moitra S (2024) Health promotion in the management of respiratory diseases: an Indian perspective. Lancet Respir Med 12:e77. https://doi.org/10.1016/S2213-2600(24)00332-1
15. Doke PP (2023) Chronic respiratory diseases: a rapidly emerging public health menace. Indian J Public Health 67:192–196. https://doi.org/10.4103/ijph.ijph_726_23
16. Farheen A, Hazari MA, Khatoon F, Sultana F, Qudsiya SM (2017) Hematological parameters are acutely effected by cement dust exposure in construction workers: hematological changes due to cement dust. Ann Med Physiol 1:31–35. https://doi.org/10.23921/amp.2017v1i1.263312
17. Scharf P, Broering MF, Oliveira da Rocha GH, Farsky SH (2020) Cellular and molecular mechanisms of environmental pollutants on hematopoiesis. Int J Mol Sci 21:6996. https://doi.org/10.3390/ijms21196996
18. Krishna L, Pandarikkal JR, Sreedharan R, Sampson U, Annamala PT, Unni KM (2019) Assessment of respiratory morbidity among loading and unloading workers exposed to cement dust. Int J Res Med Sci 7:2422–2427. https://doi.org/10.18203/2320-6012.ijrms20192541
19. Ahmad R, Akhter QS, Haque M (2021) Occupational cement dust exposure and inflammatory nemesis: Bangladesh relevance. J Inflamm Res 14:2425–2444. https://doi.org/10.2147/JIR.S312960
20. Dewangan PK, Verma N, Shrivastava N, Prasad MA (2021) Pulmonary function parameters and its determinants among cement factory workers in Chhattisgarh: a cross-sectional study. Indian J Public Health 65:226–230. https://doi.org/10.4103/ijph.IJPH_41_21
21. Shanshal SA, Al-Qazaz HK (2021) Consequences of cement dust exposure on pulmonary function in cement factory workers. Am J Ind Med 64:192–197. https://doi.org/10.1002/ajim.23211
22. Vinnikov D, Abenova A, Raushanova A, Rapisarda V (2023) Occupational exposure to fine particulate matter in the reinforced concrete production and its association with respiratory symptoms and lung function. BMC Public Health 23:1813. https://doi.org/10.1186/s12889-023-16753-x
23. Cetintepe SP, Demirbas OB, Dinke B, Ilhan MN (2025) Is exposure to cement dust and heavy metals associated with reduced pulmonary function? A cross-sectional study among cement factory workers in Türkiye. BMJ Open 15:e102214. https://doi.org/10.1136/bmjopen-2025-102214
24. Vanka KS, Shukla S, Gomez HM, James C, Palanisami T, Williams K, Chambers DC, Britton WJ, Ilic D, Hansbro PM, Horvat JC (2022) Understanding the pathogenesis of occupational coal and silica dust-associated lung disease. Eur Respir Rev 31:210250. https://doi.org/10.1183/16000617.0250-2021
25. Darabi F, Keshavarzi M, Khanjani N, Yousefinejad S, Zamanian Z (2023) Effect of antioxidants on oxidative damage, and hematological and psychological parameters in cement-exposed workers. Hyg Environ Health Adv 6:100060. https://doi.org/10.1016/j.heha.2023.100060
26. Dushyant K, Walia GK, Devasenapathy N (2023) Lung function and respiratory morbidity among informal workers exposed to cement dust: a comparative cross-sectional study. Ann Glob Health 89:47. https://doi.org/10.5334/aogh.4089

27. Sohrabi Y, Sabet S, Yousefinejad S, Rahimian F, Aryaie M, Soleimani E, Jafari S (2022) Pulmonary function and respiratory symptoms in workers exposed to respirable silica dust: a historical cohort study. Heliyon 8:e11642. https://doi.org/10.1016/j.heliyon.2022.e11642
28. Chen CH, Tsai PJ, Chang WW, Chen CY, Chen CY, Yates D, Guo YL (2024) Dose-response relationship between lung function and chest imaging response to silica exposures in artificial stone manufacturing workers. Environ Health 23:25. https://doi.org/10.1186/s12940-024-01067-1
29. Stanojevic S, Kaminsky DA, Miller MR, Thompson B, Aliverti A, Barjaktarevic I, Cooper BG, Culver B, Derom E, Hall GL, Hallstrand TS, Leuppi JD, MacIntyre N, McCormack M, Rosenfeld M, Swenson ER (2022) ERS/ATS technical standard on interpretive strategies for routine lung function tests. Eur Respir J 60:2101499. https://doi.org/10.1183/13993003.01499-2021
30. Blanco-Pérez JJ, Blanco-Dorado S, Rodríguez-García J, Gonzalez-Bello ME, Salgado-Barreira A, Caldera-Díaz AC, Pallarés-Sanmartín A, Fernandez-Villar A, González-Barcala FJ (2021) Serum levels of inflammatory mediators as prognostic biomarker in silica exposed workers. Sci Rep 11:13348. https://doi.org/10.1038/s41598-021-92587-0
31. Rezaei A, Ghafari ME, Sohrabi Y, Aliasghari F, Yousefinejad S, Soleimani E, Jafari S (2024) Systemic inflammation indices as hematological biomarkers of inflammatory response in non-silicotic workers exposed to respirable silica dust. Toxicol Lett 395:26–39. https://doi.org/10.1016/j.toxlet.2024.03.005
32. Beigoli S, Amin F, Kazemi Rad H, Rezaee R, Boskabady MH (2024) Occupational respiratory disorders in Iran: a review of prevalence and inducers. Front Med (Lausanne) 11:1310040. https://doi.org/10.3389/fmed.2024.1310040
33. Nandi SS, Lambe UP, Sarkar K, Sawant S, Deshpande J (2021) A rapid point of care CC16 kit for screening of occupational silica dust exposed workers for early detection of silicosis/silico-tuberculosis. Sci Rep 11:23485. https://doi.org/10.1038/s41598-021-02392-y
34. Wardyn PM, Edme JL, de Broucker V, Cherot-Kornobis N, Ringeval D, Amouyel P, Sobaszek A, Dauchet L, Hulo S (2023) The impact of occupational exposure to crystalline silica dust on respiratory function (airway obstruction and FEF25-75) in the French general population. Environ Res 222:115382. https://doi.org/10.1016/j.envres.2023.115382
35. Ahmad R, Haq MA, Sinha S, Mehta M, Kumar S, Haque M, Akhter QS (2024) Impact of occupational cement dust exposure on hematological health parameters: a cross-sectional study. Cureus 16:e72673. https://doi.org/10.7759/cureus.72673
36. García-Núñez A, Jiménez-Gómez G, Hidalgo-Molina A, Córdoba-Doña JA, León-Jiménez A, Campos-Caro A (2022) Inflammatory indices obtained from routine blood tests show an inflammatory state associated with disease progression in engineered stone silicosis patients. Sci Rep 12:8211. https://doi.org/10.1038/s41598-022-11926-x
37. Kowalczyk P, Krych S, Kramkowski K, Jęczmyk A, Hrapkowicz T (2024) Effect of oxidative stress on mitochondrial damage and repair in heart disease and ischemic events. Int J Mol Sci 25:12467. https://doi.org/10.3390/ijms252212467

38. Donia T, Khamis A (2021) Management of oxidative stress and inflammation in cardiovascular diseases: mechanisms and challenges. Environ Sci Pollut Res 28:34121–34153. https://doi.org/10.1007/s11356-021-14109-9.

Figures

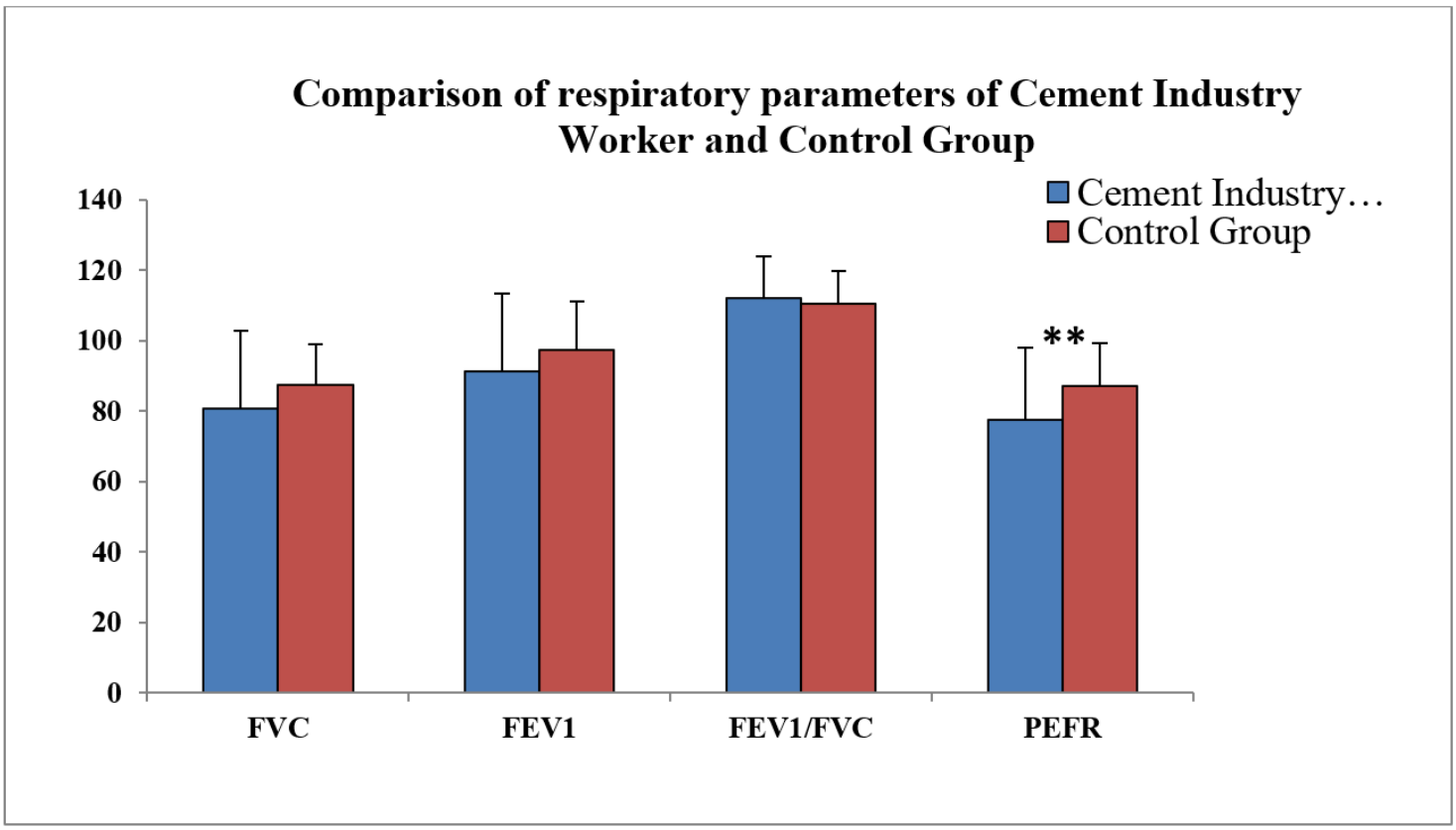


Figure 1

Comparison of respiratory parameters of cement industry workers and control group population

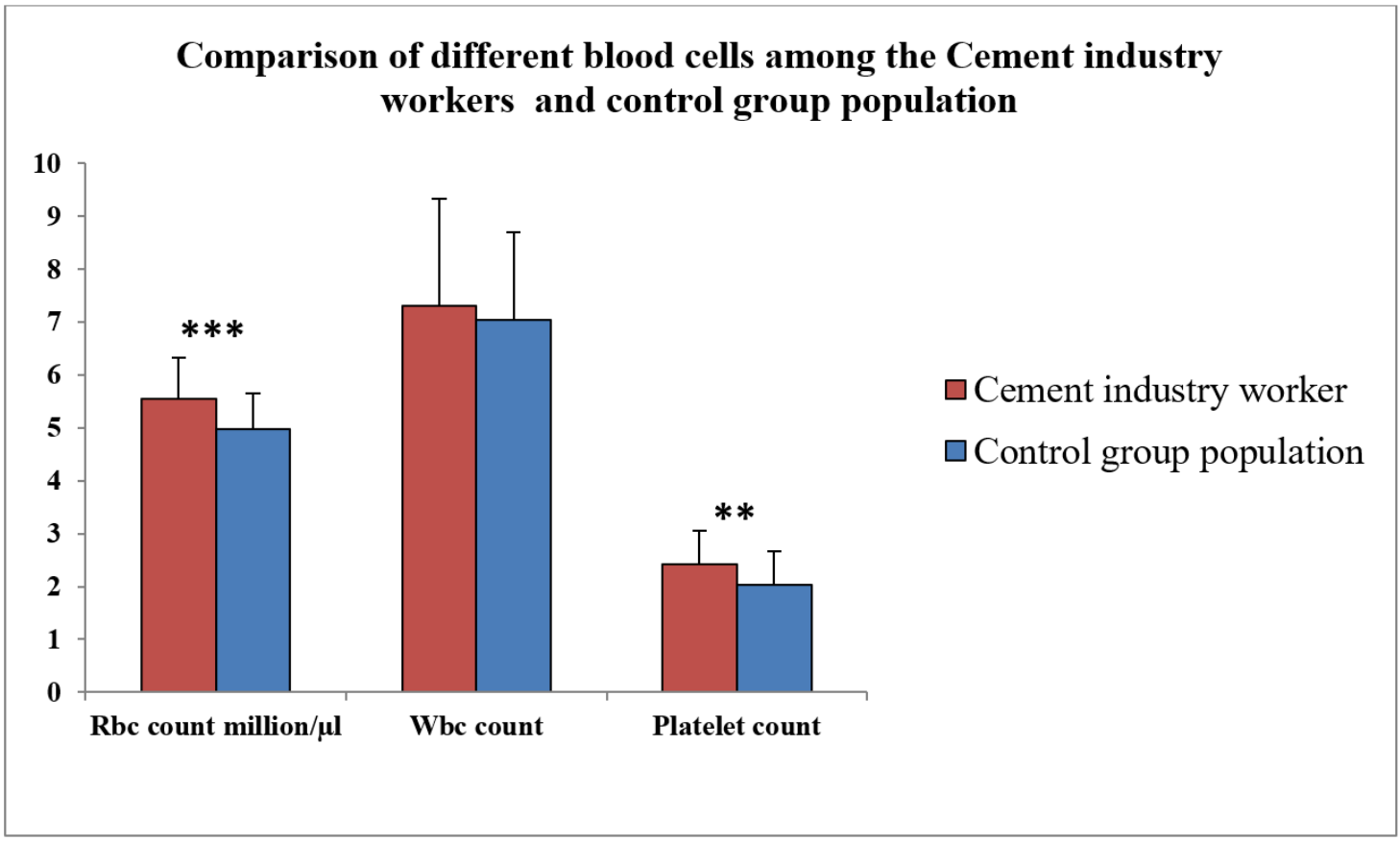


Figure 2

Comparison of Different blood cells among the Cement industry workers and control group population

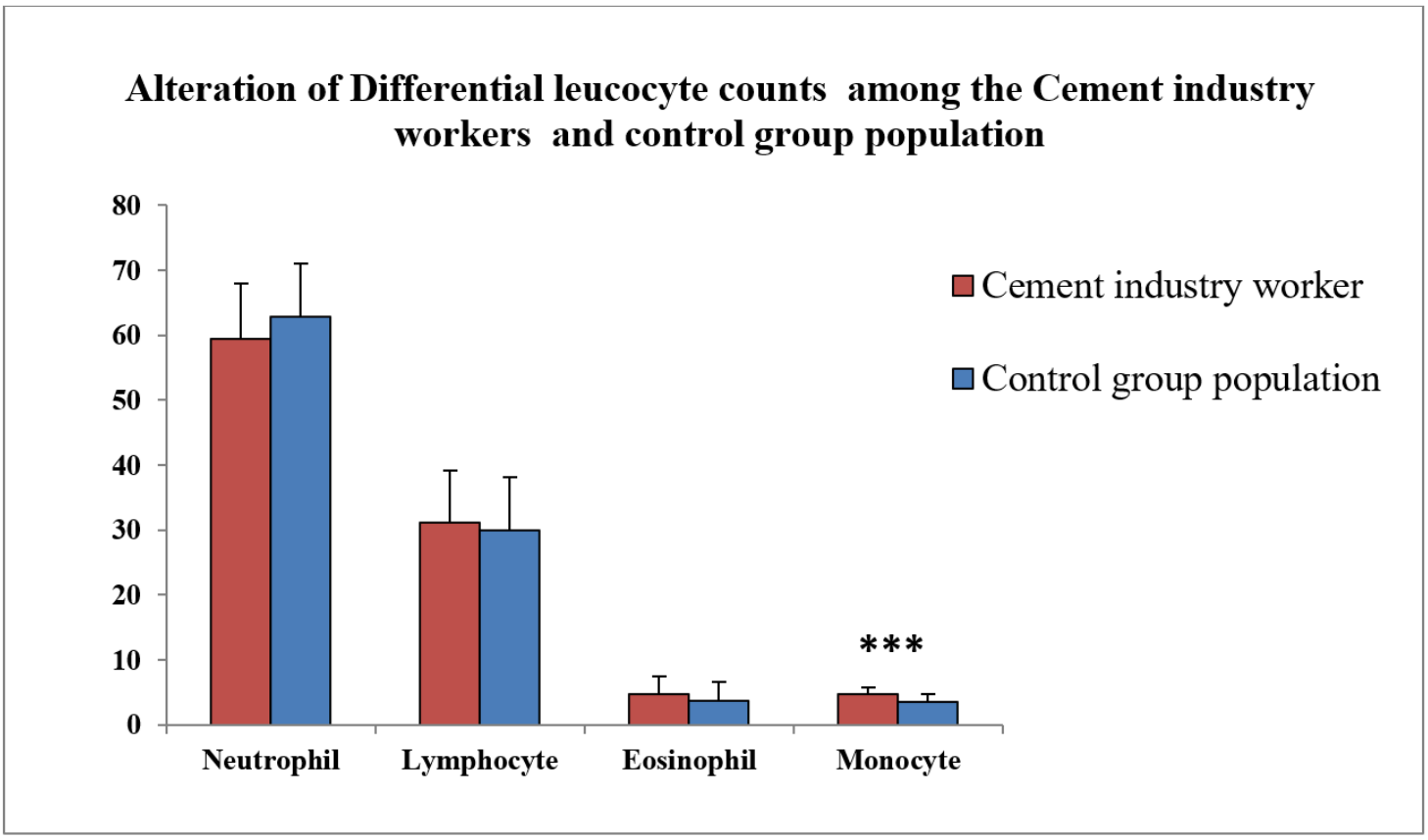


Figure 3

Alteration of Differential Leucocyte Counts among the Cement industry workers and control group population

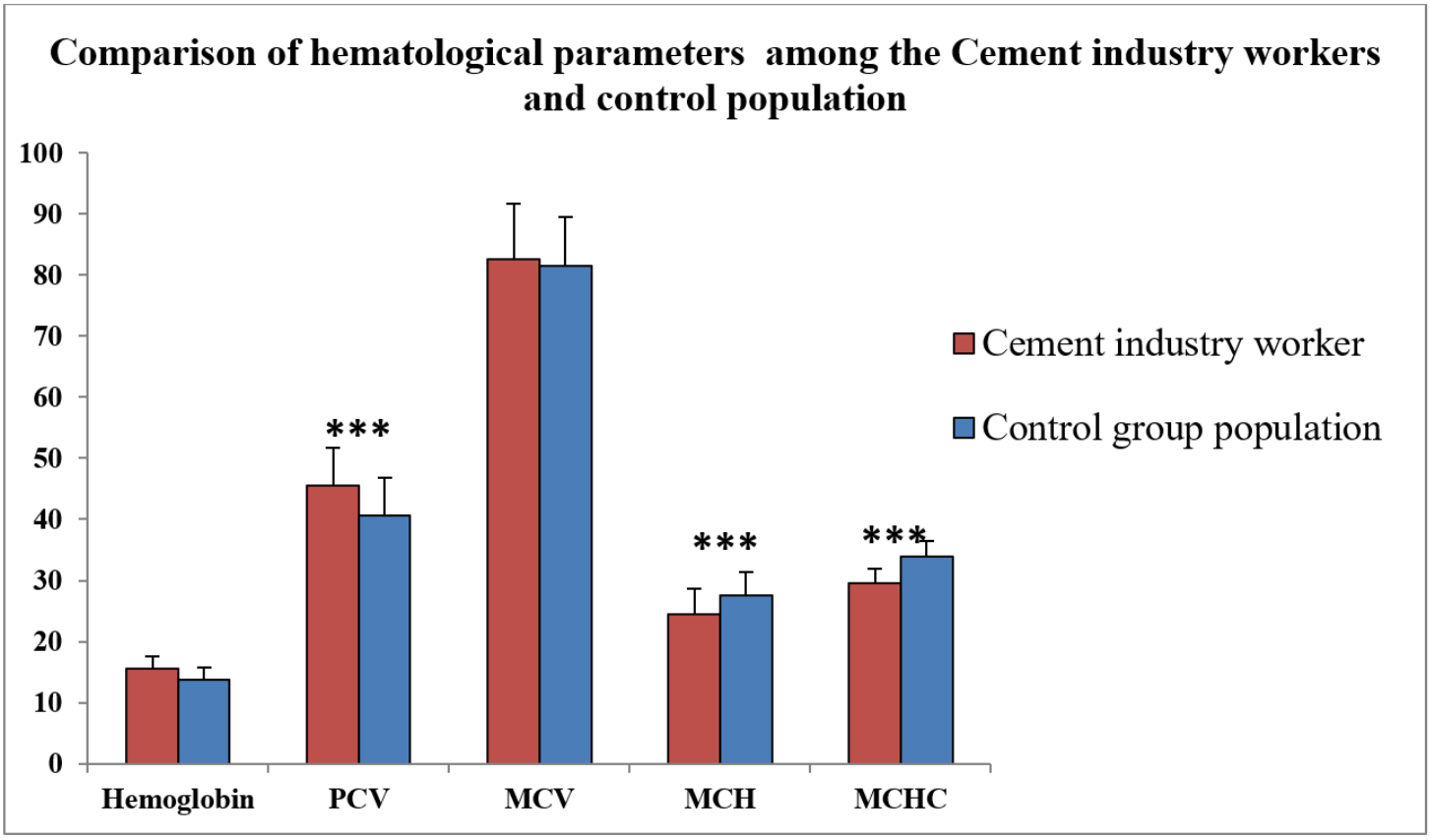


Figure 4

Comparison of hematological parameters among the Cement industry workers and control group population