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Full-length Research Article

Cardiorespiratory and Haematological Indices in Factory Workers Exposed to Cement Dust

Yussuf O.E. and *Ige A.O.

Applied and Environmental Physiology Unit, Department of Physiology, University of Ibadan, Ibadan, Nigeria.

Summary: The Nigerian cement industry, which produces 58.9 million metric tonnes annually, creates occupational health risks for workers who regularly encounter cement dust containing fine particulates with crystalline silica and heavy metals. This study aims to investigate the effects of cement dust on cardiorespiratory and haematological indices among exposed factory workers. The research design utilized a cross-sectional approach on 111 participants (60 cement factory workers (53 male, 7 female) who had at least three years of exposure and 51 control subjects (44 male, 7 female), matched by demographic characteristics and unexposed to cement dust. Pulmonary function was assessed via spirometry; cardiovascular health was evaluated using electrocardiography and blood pressure measurement with a digital sphygmomanometer; and a haematological analysis (full blood count) was performed using standard methods. The study data demonstrated that exposed workers experienced significant decreases in forced vital capacity (FVC), forced expiratory volume in one second (FEV1), Forced Expiratory Flow at 75% of FVC (FEF75), and peak expiratory flow, which point towards restrictive pulmonary damage. The cardiovascular evaluations revealed elevated systolic blood pressure, prolonged QTc intervals, and reduced T-wave amplitudes. Simultaneously, the haematological assessments revealed increased red cell distribution width and changes in white blood cell counts. The results demonstrate the occupational risk factors (marked declines in lung function, disruptions in cardiac indices, and haematological abnormalities) associated with exposure to cement dust, highlighting the need for stronger industrial regulations and protective measures.

Keywords*: cement dust, electrocardiography, spirometry, haematological indices.*

**Authors for correspondence: aby_ige@yahoo.com, Tel: +234-08033787617*

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INTRODUCTION

The cement industry plays a major role in a nation's global economic development, contributing significantly to infrastructural development (Etim *et al*., 2021). However, the industry's rapid growth has introduced numerous occupational hazards, particularly for workers exposed to cement dust. Cement dust exposure has been implicated in respiratory, haematological, and cardiovascular health issues due to its composition of fine particulates, crystalline silica, and heavy metals such as cadmium and chromium (Ogunbileje *et al*., 2014; Rahmani *et al*., 2018; Omidianidost *et al*., 2019; Obaji-Ogar *et al*., 2020; Al-Shamery & Jankeer, 2021).

Chronic exposure to cement dust is associated with an increased risk of cardiovascular disease, owing to systemic inflammation and oxidative stress (Obaji-Ogar *et al*., 2020). Factory workers exposed to cement dust exhibit a high prevalence of respiratory symptoms and reduced lung function, indicating pronounced adverse respiratory effects (Aweto *et al*., 2018). In addition to affecting the cardiovascular and respiratory systems, cement dust exposure also alters haematological parameters. These alterations lead to immune activation and inflammation, which may cause systemic health problems over time (Emmanuel *et al*., 2015).

Although the cement industry is economically important, protective measures for workers remain suboptimal, particularly in low- and middle-income settings. This study aimed to compare cardiorespiratory and haematological indices between factory workers with prolonged exposure to cement dust and an unexposed control group, thereby providing evidence to inform occupational health policies and intervention strategies.

MATERIALS AND METHODS

Study Design and Setting: This study employed a cross-sectional design to investigate the effects of cement dust exposure on cardiorespiratory and haematological indices among cement factory workers. The research was conducted at the Dangote Cement Factory in Obajana, Kogi State, Nigeria.

Sample Size and Justification: The sample size for this study was determined using the standard formula for comparing two independent means as described by Charan and Biswas (2013). The parameters applied in the

calculation were derived from the findings of Emmanuel *et al*. (2015), who evaluated the physiological effects of occupational exposure to cement dust.
The formula used is:

$$n = \frac{2\sigma^2\left(Z_{\frac{\alpha}{2}} + Z_{\beta}\right)^2}{d^2}$$

Where n = *Minimum sample size required per group*

- $Z_{\frac{\alpha}{2}}$ = Standard normal deviate corresponding to a 95% confidence level (1.96)
- Z_{β} = Standard normal deviate corresponding to 80% statistical power (0.84)
- σ = Pooled standard deviation obtained from the reference study
- d = Minimum detectable difference between the two group means

$$n = \frac{2\sigma^2(1.96 + 0.84)^2}{d^2}$$

$$n = \frac{2\sigma^2(7.84)^2}{d^2}$$

$$n = \frac{15.68\sigma^2}{d^2}$$

Using the variance (σ^2) of 1.0 and a mean difference (d) of 0.56 derived from the Red Blood Cell (RBC) indices reported in the reference study (Emmanuel *et al*., 2015), the calculated minimum sample size was approximately 50 participants per arm. To account for potential non-response or attrition, a total of 111 participants were recruited (60 factory workers and 51 controls).

$$n = \frac{15.68}{0.3136} = 50$$

Sampling Technique: Participants were selected using a stratified random sampling method to ensure representation across different factory departments, including production, packaging, and loading units. Control participants, drawn from a farming population approximately 11 km from the factory, were matched for age, sex, and physical activity levels to minimize confounding variables. This distance was selected to ensure that the participants were not in the immediate area of high particulate fallout and industrial air pollution dispersion, yet were still within the same geoclimatic area to control for environmental confounders such as temperature and humidity.

Eligibility Criteria: The participants in the study were male and female aged between 18 and 60 years who had at least 3 years of continuous occupational exposure to cement dust. Moreover, every participant had to give voluntary written informed consent before being enrolled in the study. The participants excluded in the study were those with pre-existing cardiovascular, respiratory, or haematological conditions not related to exposure to cement dust. To make valid comparisons of occupational hazards, workers with less than 3 years of exposure history were not recruited. Moreover, to reduce the number of confounding factors, the study excluded smokers, alcohol-dependent people, individuals with other chronic diseases, pregnant women, and those who were taking drugs that are known to alter physiological indices.

Bias and Error Minimization: The study had several measures that were taken to reduce measurement bias and experimental error. Data collection of the exposed and control groups was done simultaneously during the same week to control environmental confounders like temperature and humidity. Calibration of all diagnostic devices was done prior to use, and procedures were strictly followed to standard protocols to ensure consistency. To further reduce observer bias, the technicians performing electrocardiography and spirometry assessments were blinded to the participants' group allocations. Also, the participants were thoroughly briefed and guided in the course of testing to guarantee performance validity, and quality control samples were used in the course of haematological analyses to guarantee data integrity.

MATERIALS AND METHODS

Equipment included: ECG (Biocare ECG-9801), Sphygmomanometer (Omicron), Spirometer (CONTEC SP10), Pulse oximeter, Haematology analyzer (Abacus Junior), and standard phlebotomy materials (e.g., EDTA tubes, sterile syringes, alcohol swabs).

Data Collection Procedures

Cardiovascular Assessment: A validated digital sphygmomanometer (Omicron) was used to measure blood pressure based on standard procedures. The participants were made to sit in a comfortable position with their back supported and their arms at the level of the heart. At least 10 minutes of rest were given to them before measurement to ascertain hemodynamic stability. Three consecutive readings were taken at 2-minute intervals for each participant. To reduce variability, the mean of the two final readings was taken as the final systolic and diastolic blood pressure reading. A 12-lead electrocardiogram (ECG) was performed using a Biocare ECG-9801 machine in accordance with American Heart Association (AHA) guidelines. The parameters analyzed included heart rate, QT interval, PR interval, QRS duration, QRS axis, and T-wave characteristics.

Respiratory Function Testing: Pulmonary function was assessed using a CONTEC SP10 spirometer to measure Forced Vital apacity (FVC), Forced Expiratory Volume in one second (FEV_1), the FEV_1/FVC ratio, Peak Expiratory Flow (PEF), and Forced Expiratory Flow at 25–75% of FVC (FEF25–75%). The spirometer was calibrated before each session to ensure accuracy. Participants were tested in a rested state using disposable mouthpieces. Three trials were performed for each participant, and the best reading was recorded for analysis. Additionally, peripheral oxygen saturation (SpO_2) was recorded using a pulse oximeter under resting conditions.

Haematological Assessment: Venous blood samples (3–5 mL) were collected from the antecubital vein into ethylenediaminetetraacetic acid (EDTA) tubes using sterile techniques. The samples were analyzed within four hours of collection using an Abacus Junior haematology analyzer. The parameters assessed included total White Blood Cell (WBC) count and differential counts (lymphocytes, monocytes, granulocytes), as well as Red Blood Cell (RBC) indices (RBC count, haematocrit, Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH),

Mean Corpuscular Haemoglobin Concentration (MCHC), and Red Cell Distribution Width (RDW), and platelet count
Statistical Analysis: Data were analysed using descriptive statistics (mean ± SEM). Student's independent t-test was used to compare group means, and Pearson's correlation assessed the relationships among variables. The statistical significance threshold was set at $p < 0.05$.

Ethical Approval
Ethical approval for this study was obtained from the UI/UCH Institutional Review Committee-(UI/EC/24/0342). Written informed consent was obtained from each participant after the study objectives, procedures, potential risks, and benefits were clearly explained. Participation was strictly voluntary, and individuals had the right to withdraw from the study at any time without penalty. Confidentiality of participants' information was ensured throughout the research process.

RESULTS

Anthropometric Measurements: No significant differences were observed between the exposed and control groups in age, height, or weight (Table 1).

Respiratory Parameters: The analysis of pulmonary functions revealed evident impairments in the exposed workers, and the degree of decrease varied depending on gender. In general, factory workers had a much lower Forced Vital Capacity (FVC) (3.10 ± 0.08 L; p = 0.012) and Forced Expiratory Volume in one second (FEV1) (2.51 ± 0.06 L; p = 0.005) than controls. This trend was also reflected in male workers, who had much lower FVC (p = 0.019) and peak expiratory flow (PEF) (p = 0.011) compared with the male controls in the gender-specific analysis. However, female workers did not show any significant changes in FVC (p = 0.439) or FEV1 (p = 0.285) compared with the female controls. There was no gender difference in small-airway functioning. Flow at 75% (FEF_{75}) was significantly lower in the overall exposed group (p < 0.001) and significantly suppressed in both male (1.18 ± 0.08 L/s; p < 0.001) and female (1.30 ± 0.06 L/s; p = 0.022) subgroups

Cardiovascular Parameters: Cardiovascular assessment revealed significant physiological changes. In general, systolic blood pressure (135.58 ± 2.05 mmHg; p = 0.005) was much higher in exposed workers than in controls. This pattern of hypertension became significant in both males (p = 0.021) and females (p = 0.025). The ECG disturbances were more pronounced in males. Male employees had a much lower QRS axis (28.04 ± 4.76; p = 0.007) and a longer QTcF interval (p = 0.014), whereas there was no significant difference in these parameters between males and females (p = 0.369 and p = 0.867, respectively). Only the male workers also had significantly higher left ventricular hypertrophy indices (SV 1 + RV 5) (p = 0.036). T-wave amplitude was reduced in the general exposed group (p < 0.001) and among males (p < 0.001), with females showing only a borderline decline (p = 0.074).

Haematological Parameters: Hematologic assessment showed signs of systemic inflammation and erythropoietic disruption, and some of the changes were more severe in male factory workers. In general, the exposed group had a much greater total white blood cell count (5.93 ± 0.28 × 10 9/L; $p = 0.046$) and a much lower percentage of mixed-cell/monocyte (MI) (6.46 ± 0.41%; $p < 0.001$). The decrease in the percentage of MI was very significant in both males ($p = 0.001$) and females ($p = 0.001$). Red Cell Distribution Width (RDW) was also significantly higher in the total exposed group (17.60 ± 0.27%; $p < 0.001$), but only in males (17.63 ± 0.29%; $p < 0.001$), and no significant change in females ($p = 0.297$). Mean Corpuscular Haemoglobin (MCH) was significantly lower in the general exposed group ($p = 0.045$), though subgroup analysis showed no significant difference within males ($p = 0.089$) or females ($p = 0.510$) independently.

Correlation Analysis: Higher BMI was linked to prolonged P-wave duration, greater mean corpuscular volume, and reduced T-wave amplitude. Height correlated with FVC, which was strongly associated with FEV_1 and moderately with PEF and T-wave duration. FEV_1 correlated with red blood cell count, haematocrit, and mid-expiratory flow rates. QRS duration was negatively related to QRS axis and positively to QT interval and QTc. Red blood cell count correlated positively with haematocrit but inversely with mean corpuscular volume and haemoglobin; red cell distribution width showed similar patterns. Haematocrit was negatively associated with peripheral oxygen saturation (Table 5).

Table 1:
Shows the demographics among the Factory workers and the control group

Group	Age	Height	Weight	BMI	p-value
Factory	48.13 ± 0.90	1.71 ± 0.01	75.82 ± 1.45	26.01 ± 0.45	NS
Control	46.98 ± 1.19	1.70 ± 0.01	76.20 ± 1.83	26.38 ± 0.70	NS
Male Factory	48.53 ± 0.88	1.71 ± 0.01	76.38 ± 1.59	26.02 ± 0.48	NS
Male Control	47.30 ± 1.30	1.71 ± 0.01	78.23 ± 1.90	26.96 ± 0.72	NS
Female Factory	45.14 ± 3.89	1.66 ± 0.02	71.57 ± 2.72	25.93 ± 1.26	NS
Female Control	45.00 ± 2.91	1.66 ± 0.04	63.42 ± 2.82	22.70 ± 1.79	NS

Values are expressed as mean ± SEM. BMI = Body Mass Index. All comparisons between groups showed no statistically significant differences (NS, p > 0.05) (P-value derived from t-test).

Table 2:
Independent Samples T-test Analysis of Lung Function Parameters in Factory Workers vs. Controls

Parameter	Controls	FW	Diff	T-value	p-value	Male Controls	Male FW	Male Diff	Male T-value	Male p-value	Female Controls	Female FW	Female Diff	Female T-value	Female p-value
FVC	3.46 ± 0.12	3.10 ± 0.08	-0.364	-2.545	0.012 *	3.46 ± 0.14	3.09 ± 0.08	0.371	-2.28	0.019 *	3.47 ± 0.27	3.16 ± 0.28	-0.311	-0.8	0.439 (NS)
FEV1	2.84 ± 0.10	2.51 ± 0.06	-0.327	-2.873	0.005 **	2.82 ± 0.11	2.49 ± 0.06	0.322	-2.52	0.011 *	2.97 ± 0.23	2.63 ± 0.20	-0.337	-1.12	0.285 (NS)
FEV1/FVC	82.17 ± 0.86	81.55 ± 1.02	-0.621	-0.455	0.650 (NS)	81.61 ± 0.96	81.21 ± 1.14	0.397	-0.35	0.795 (NS)	85.71 ± 1.14	84.11 ± 1.53	-1.596	-0.84	0.419 (NS)
PEF	7.34 ± 0.28	6.41 ± 0.22	-0.930	-2.646	0.009 **	7.45 ± 0.30	6.48 ± 0.23	0.969	-2.56	0.011 *	6.60 ± 0.78	5.81 ± 0.74	-0.783	-0.73	0.480 (NS)
FEF75	1.72 ± 0.10	1.20 ± 0.07	-0.521	-4.385	0.000 **	1.66 ± 0.11	1.18 ± 0.08	0.476	-3.61	0.000 **	2.09 ± 0.26	1.30 ± 0.06	-0.790	-2.97	0.022 **

*Values are presented as mean ± SEM. FW = Factory Workers; Diff = Mean Difference; t-value = t-statistic from Student's independent t-test; NS = Not significant ($p > 0.05$). Asterisks indicate statistical significance: *$p < 0.05$ (significant); **$p < 0.01$ (highly significant). FVC = Forced Vital Capacity; FEV_1 = Forced Expiratory Volume in 1 second; FEV_1/FVC = Ratio of FEV_1 to FVC; PEF = Peak Expiratory Flow; FEF_{75} = Forced Expiratory Flow at 75% of FVC.*

Table 3:
Independent Samples T-test Analysis of Cardiovascular Parameters (Blood Pressure, Heart Rate, and ECG Indices) Between Factory Workers and Controls

Parameter	Controls	FW	Diff	T-value	p-value	Male Controls	Male FW	Male Diff	Male T-value	Male p-value	Female Controls	Female FW	Female Diff	Female T-value	Female p-value
Systole (mmHg)	127.12 ± 2.14	135.58 ± 2.05	8.47	2.849	0.005 **	126.77 ± 2.47	134.43 ± 2.17	7.66	2.34	0.021 *	129.29 ± 1.21	144.29 ± 5.71	15.00	2.57	0.025 *
Diastole (mmHg)	80.90 ± 1.04	83.22 ± 1.17	2.31	1.457	0.148 NS	81.39 ± 1.13	82.89 ± 1.24	1.50	0.88	0.380 NS	77.86 ± 2.53	85.71 ± 3.71	7.86	1.75	0.106 NS
Heart Rate (bpm)	69.75 ± 0.98	66.33 ± 1.24	-3.41	-2.105	0.038 *	69.64 ± 1.11	66.94 ± 1.27	-2.69	-1.57	0.121 NS	70.43 ± 1.63	61.71 ± 4.52	-8.71	-1.81	0.095 NS
QRS Axis (°)	47.31 ± 5.20	26.93 ± 4.69	-20.38	-2.915	0.004 **	48.27 ± 5.57	28.04 ± 4.76	-20.24	-2.78	0.007 **	41.29 ± 15.41	18.57 ± 18.80	-22.71	-0.93	0.369 NS
T-wave Amplitude (Limb Leads, mV)	0.396 ± 0.028	0.225 ± 0.015	-0.1715	-5.614	0.000 **	0.392 ± 0.029	0.223 ± 0.017	-0.16918	-4.98	0.000 **	0.426 ± 0.091	0.243 ± 0.023	-0.183	-1.95	0.074 *
QTcF	0.423 ± 0.005	0.440 ± 0.006	0.0172	2.144	0.034 *	0.421 ± 0.006	0.442 ± 0.006	0.02048	2.51	0.014 *	0.432 ± 0.020	0.427 ± 0.023	-0.005	-0.17	0.867 NS
SV1 + RV5 (mV)	2.424 ± 0.102	2.617 ± .083	0.1931	1.481	0.142 NS	2.361 ± 0.114	2.657 ±0.084	0.29524	2.08	0.036 *	2.81 ± 0.15	2.31 ± 0.31	-0.500	-2.25	0.172 NS

*Values are expressed as mean ± SEM. FW = Factory Workers; Diff = Mean Difference; NS = Not significant ($p > 0.05$). Asterisks indicate statistical significance: *$p < 0.05$ (significant); **$p < 0.01$ (highly significant). QRS Axis = Electrical axis of the QRS complex (degrees); T-wave Amplitude (Limb Leads) = Amplitude of the T wave in limb leads (mV); QTcF = QT interval corrected using Fridericia's formula; $SV_1 + RV_5$ = Sum of S-wave amplitude in V_1 and R-wave amplitude in V_5 (mV).*

Table 4:
Independent Samples T-test Analysis of Haematological Parameters (Blood Pressure, Heart Rate, and ECG Indices) Between Factory Workers and Controls

Parameter	Controls	FW	Diff	T-value	p-value	Male Controls	Male FW	Male Diff	Male T-value	Male p-value	Female Controls	Female FW	Female Diff	Female T-value	Female p-value
WBC (10^9/L)	5.29 ± 0.15	5.93 ± 0.28	0.64	2.037	0.046 *	5.31 ± 0.16	5.84 ± 0.30	0.53	1.53	0.133 (NS)	5.22 ± 0.40	6.35 ± 0.69	1.13	1.35	0.204 (NS)
MID (10^9/L)	0.45 ± 0.02	0.37 ± 0.03	-0.08	-2.198	0.032 *	0.44 ± 0.03	0.37 ± 0.03	-0.07	-1.74	0.088 (NS)	0.50 ± 0.05	0.40 ± 0.08	-0.10	-1.05	0.314 (NS)
M I (%)	10.27 ± 0.66	6.46 ± 0.41	-3.81	-4.935	0.000 **	10.05 ± 0.80	6.54 ± 0.49	-3.52	-3.99	0.001 **	11.02 ± 1.02	6.10 ± 0.57	-4.92	-4.38	0.001 **
MCH (pg)	29.75 ± 0.71	28.21 ± 0.39	-1.54	-2.045	0.045 *	29.81 ± 0.83	28.16 ± 0.44	-1.65	-1.76	0.089 (NS)	29.53 ± 1.45	28.41 ± 0.89	-1.12	-0.68	0.510 (NS)
RDW (%)	15.24 ± 0.42	17.60 ± 0.27	2.36	4.765	0.000 **	14.87 ± 0.49	17.63 ± 0.29	2.75	5.1	0.000 **	16.50 ± 0.53	17.46 ± 0.67	0.96	1.09	0.297 (NS)

*Values are presented as mean ± SEM. FW = Factory Workers; Diff = Mean Difference; t-value = t-statistic from Student's independent t-test; NS = Not significant (p > 0.05). Asterisks denote significance levels: *p < 0.05 (significant); **p < 0.01 (highly significant). WBC (10^9/L) = White Blood Cell count; MID (10^9/L) = Combined count of monocytes, eosinophils, and basophils; M I (%) = Percentage of monocytes, eosinophils, and basophils; MCH (pg) = Mean Corpuscular Haemoglobin; RDW (%) = Red Cell Distribution Width.*

Table 5:
Key Significant Correlations Across Anthropometric, Respiratory, Cardiovascular, and Haematological Parameters

Variables	Pearson Correlation	p-value	Direction
Height → PR Interval	0.265	0.05	Weak positive
Weight → PR Interval	0.295	0.05	Weak positive
Weight → T Wave Amplitude (Precordial Leads)	-0.283	0.05	Weak negative
BMI → P Wave Duration	0.291	0.05	Weak positive
BMI → T Wave Amplitude (Precordial Leads)	-0.358	0.001	Moderate negative
BMI → MCV	0.330	0.01	Moderate positive
Height → FVC	0.320	0.05	Moderate positive
FVC → FEV1/FVC	-0.393	0.05	Moderate negative
FVC → PEF	0.410	0.01	Moderate positive
FVC → T Wave Duration	0.313	0.05	Moderate positive
FEV_1 → T Wave Duration	0.386	0.01	Moderate positive
FEV_1 → RBC	0.389	0.05	Moderate positive
FEV_1 → HCT	0.454	0.01	Moderate positive
PEF → PR Interval	-0.320	0.05	Moderate negative
QRS Duration → QRS Axis	-0.421	0.01	Moderate negative
QRS Duration → QT Interval	0.317	0.05	Moderate positive
QRS Axis → T Wave Amplitude (Limb Leads)	0.382	0.01	Moderate positive
QT Interval → QTc	0.537	0.01	Moderate positive
RDW → MCV	-0.386	0.05	Moderate negative
RDW → MCH	-0.475	0.01	Moderate negative
HCT → SpO_2	-0.322	0.05	Moderate negative

BMI – Body Mass Index; BP – Blood Pressure; FVC – Forced Vital Capacity; FEV_1 – Forced Expiratory Volume in 1 second; FEV_1/FVC – Ratio of Forced Expiratory Volume in 1 second to Forced Vital Capacity; PEF – Peak Expiratory Flow; PR Interval – Time between onset of atrial and ventricular depolarisation; P Wave Duration – Duration of atrial depolarisation; T Wave Duration – Duration of ventricular repolarisation; T Wave Amplitude – Height of T wave on electrocardiogram; QRS Duration – Duration of ventricular depolarisation; QRS Axis – Electrical axis of ventricular depolarisation; QT Interval – Time from ventricular depolarisation to repolarisation; QTc – Corrected QT Interval; RBC (10^{12}/L) – Red Blood Cell count; HCT (%) – Haematocrit; MCV (fL) – Mean Corpuscular Volume; MCH (pg) – Mean Corpuscular Haemoglobin; RDW (%) – Red Cell Distribution Width; SpO_2 (%) – Peripheral Oxygen Saturation

DISCUSSION

Baseline demographics were similar between groups, so observed differences are unlikely to reflect age, height, or weight discrepancies. This study demonstrates that cement dust exposure adversely affects several lung function parameters, including Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV_1), Peak Expiratory Flow (PEF), and Forced Expiratory Flow at 75% of FVC (FEF_{75}). These reductions reflect diminished lung capacity and expiratory flow efficiency among factory workers compared with healthy controls (Meo, 2004; Shanshal and Al-Qazaz, 2021; Akhter *et al*., 2021; Dushyant *et al*., 2023). The lower FVC in workers suggests restrictive lung changes, possibly from chronic dust inhalation, causing airway inflammation and reduced pulmonary compliance (Al-Neaimi *et al*., 2001). Although FEV_1 was reduced, the FEV_1/FVC ratio remained similar between groups, supporting a restrictive rather than obstructive pattern. This finding aligns with that of Shanshal and Al-Qazaz (2021) in a study conducted in Mosul, Iraq, who reported restriction as the predominant pattern in 45.4% of factory workers compared with 6.2% of controls recruited from academic and administrative sectors in Mosul.

The FEF_{75} values seen in this study amongst workers was markedly declined, indicating small-airway involvement, consistent with cumulative dust deposition. Among females, only FEF_{75} was significantly reduced, suggesting localized small-airway disease. The absence of significant changes in other lung indices may reflect lower exposure levels or physiological differences, as noted in earlier studies such as that of Hall *et al* (2024) who reported that women experience fewer respiratory declines in such settings due to shorter work hours and roles. However, the risk of chronic respiratory infection remains, as evidenced by long-term risk reports in industrial environments (Nku *et al*., 2005).

In line with other studies that brings to fore occupational hazards associated with working in the cement industry, hypertension marked by elevations in systolic blood pressure (SBP) was observed among factory workers. This aligns with previous occupational studies that have linked the exposure to particulate matter exposure to hypertension through mechanisms that are triggered by systemic inflammation, oxidative stress, and endothelial dysfunction (Rahmani *et al*., 2018; Li *et al*., 2024). Furthermore, electrocardiographic patterns observed in this study suggests that exposure to cement dust also significantly alters ventricular depolarization and repolarization dynamics. Reduced QRS axis seen in this study may reflect left ventricular hypertrophy or conduction disturbance linked to particulate exposure (Leigh *et al*., 2016; Seko *et al*., 2021). The lower T-wave amplitude seen in workers suggests compromised myocardial repolarization, possibly from ischemia or subclinical myocardial stress (Baoum *et al*., 2022; Hill *et al*., 2021). In addition to this, the prolonged QTcF observed is also concerning as it has suggested to reflect a predisposition to ventricular arrhythmias such as Torsades de Pointes (Cubeddu, 2003; Cai *et al*., 2019).

The SV_1+RV_5 index was higher in male workers, consistent with left ventricular hypertrophy (LVH), the results are an extension of the earlier reports of respiratory dysfunction among cement workers (Mwaiselage *et al*., 2004; Nordby *et al*., 2011), which offers specific evidence of cardiac structural remodelling. Although previous findings mainly focused on pulmonary dysfunction and subsequent right heart strain (Mwaiselage *et al*., 2004; Nordby *et al*., 2011), the finding of LVH in this study, in addition to increased SBP, indicates that cement dust exposure can also cause remodeling of the systemic cardiovascular system, which could be via hypertensive or inflammatory mechanisms.

Haematological changes were also evident. Workers had elevated white blood cell (WBC) counts, indicating systemic inflammation, consistent with prior findings by Farheen *et al*. (2017) involving 65 construction workers in Hyderabad, India. Both monocyte count and percentage were reduced, suggesting immune suppression due to cytotoxic dust effects, as previously reported by Priya and Suja (2012) in a study of 30 cement factory workers in Coimbatore, India.

Red Blood Cell Distribution Width was significantly increased, particularly among males, suggesting haematological stress despite no rise in RBC count. This may represent early adaptive responses to oxidative stress and inflammation (Priya and Suja, 2012; Ahmad and Akhter, 2018; Pan *et al*., 2021). Although haematocrit (HCT) and mean corpuscular volume (MCV) were unchanged, mean corpuscular haemoglobin (MCH) was lower in workers, suggesting risk of microcytic hypochromic anaemia, consistent with impaired haemoglobin synthesis under chronic dust exposure (Ahmad and Akhter, 2018; Zhao and Lv, 2018; Pan *et al*., 2021; Peters *et al*., 2021; Jin *et al*., 2022).

Correlation analysis revealed significant associations between anthropometric, respiratory, cardiovascular, and haematological variables. Higher BMI correlated with prolonged P-wave duration, larger MCV, and reduced T-wave amplitude, supporting links between adiposity and altered cardiac conduction (Rautaharju *et al*., 2006; Aro *et al*., 2012; Ebong *et al*., 2014; Thavaraputta *et al*., 2020). Height correlated positively with FVC, which was itself associated with FEV_1 and PEF, reflecting the expected relationships between lung volume and these parameters (Dyer, 2012; Thomas *et al*., 2019).

The FEV_1 correlated with RBC count and haematocrit, underscoring the interplay between pulmonary function and oxygen transport. The QRS duration correlated negatively with QRS axis and positively with QT/QTc, showing links between ventricular conduction and repolarization (Bazett, 1997; Dilaveris *et al*., 2017; Malik and Hnatkova, 2020;). The RBC count correlated positively with haematocrit but inversely with MCV and MCH, suggesting smaller, less haemoglobin-dense cells typical of microcytic anaemia (DeLoughery, 2014; Rampon, 2023). Haematocrit also correlated negatively with SpO_2, possibly due to viscosity-related impairments in oxygen delivery (Variste *et al*., 2008).

This research had certain limitations. First, the cross-sectional design does not permit conclusions about causal relationships between cement dust exposure and the observed physiological changes. Second, the sample size was relatively small and unevenly distributed across genders because of the higher proportion of male workers, which may limit the generalizability of the results. Third, exposure

information was evaluated based on occupational category rather than direct environmental or personal exposure measurements, which may result in exposure misclassification. Additionally, potential confounding factors such as nutritional status, comorbidities, or air quality were not comprehensively controlled. To confirm and expand these results, future research should employ longitudinal designs, larger and more diverse populations, and direct quantification of cement dust exposure.

In conclusion, this study demonstrates that continuous exposure to cement dust may pose health risks to the respiratory, cardiovascular, and haematological systems. Thus, factory workers exposed to cement dust require enhanced occupational safety measures. Taken together, these findings underscore the health risk posed by cement dust exposure and emphasize the need for greater occupational health measures to diminish long-term health risks.

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Conflict of Interest Statement
The authors declare no conflict of interest.

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REFERENCES

Ahmad, R. and Akhter, Q. (2018). Effects of exposure to cement dust on haemoglobin concentration and total count of RBC in cement factory workers. J. Bangladesh Soc. Physiol. 13: 68-72.

Akhter, M., Noor, N., Begum, M., Akhter, S. and Datta, A. (2021). Effect of exposure to cement dust on peak expiratory flow rate of cement factory workers. J. Chittagong Med. Coll. Teach. Assoc. 32: 72-77.

Al-Neaimi, Y. I., Gomes, J. and Lloyd, O. L. (2001). Respiratory illnesses and ventilatory function among workers at a cement factory in a rapidly developing country. Occup. Med. (Lond.). 51: 367-373.

Al-Shamery, H. and Jankeer, M. (2021). Study the effect of cement dust exposure on the hematological variables in the workers of new Badoosh cement factory in Mosul city, Iraq. J. Phys. Conf. Ser. 1999: 012030.

Alter, P., Watz, H., Kahnert, K., Rabe, K. F., Biertz, F., Fischer, R. *et al*. (2019). Effects of airway obstruction and hyperinflation on electrocardiographic axes in COPD. Respir. Res. 20: 61.

Aro, A. L., Anttonen, O., Tikkanen, J. T., Junttila, M. J., Kerola, T., Rissanen, H. A. *et al*. (2012). Prevalence and prognostic significance of T-wave inversions in right precordial leads of a 12-lead electrocardiogram in middle-aged subjects. Circulation. 125: 2572-2577.

Aweto, H. A., Tella, B. A. and Lateef, A. I. (2018). Work-related respiratory symptoms and cardiopulmonary function impairment of factory workers in a cement company in South-West Nigeria. Afr. J. Med. Health Sci. 17: 47-53.

Baoum, S. O., Matrafi, T. M. A., Alshaikh, T. T., Alluhaibi, H. N., Alkhaldi, G. N., Alotaibi, M. H. *et al*. (2022). Common electrocardiogram manifestations in electrolyte imbalance. Int. J. Community Med. Public Health. 9: 3902-3906.

Bazett, H. C. (1997). An analysis of the time-relations of electrocardiograms. Ann. Noninvasive Electrocardiol. 2: 177-194.

Bhatt, S. P. and Dransfield, M. T. (2013). Chronic obstructive pulmonary disease and cardiovascular disease. Transl. Res. 162: 237-251.

Cai, C., Huang, J., Lin, Y., Miao, W., Chen, P., Chen, X. *et al*. (2019). Particulate matter 2.5 induced arrhythmogenesis mediated by TRPC3 in human induced pluripotent stem cell-derived cardiomyocytes. Arch. Toxicol. 93: 1009-1020.

Charan, J., and Biswas, T. (2013). How to calculate sample size for different study designs in medical research?. Indian journal of psychological medicine, 35: 121–126. https://doi.org/10.4103/0253-7176.116232

Chen, Z., Ma, J., Lei, J., Li, Y., Zhao, R. and Zhao, L. (2024). Fixed airflow obstruction in asthma can be identified early by low FEF25-75% and is associated with environmental exposure. J. Asthma Allergy. 17: 1001-1014.

Churg, A., Wright, J. L., Wiggs, B., Paré, P. D. and Lazar, N. (1985). Small airways disease and mineral dust exposure. Am. Rev. Respir. Dis. 131: 139-143.

Cubeddu, L. X. (2003). QT prolongation and fatal arrhythmias: a review of clinical implications and effects of drugs. Am. J. Ther. 10: 452-457.

DeLoughery, T. G. (2014). Microcytic anemia. N. Engl. J. Med. 371: 1324-1331.

Dilaveris, P., Antoniou, C. K., Gatzoulis, K. and Tousoulis, D. (2017). T wave axis deviation and QRS-T angle – controversial indicators of incident coronary heart events. J. Electrocardiol. 50: 466-475.

Dushyant, K., Walia, G. K. and 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.

Dyer, C. (2012). The interaction of ageing and lung disease. Chron. Respir. Dis. 9: 63-67.

Ebong, I. A., Goff, D. C. Jr, Rodriguez, C. J., Chen, H. and Bertoni, A. G. (2014). Mechanisms of heart failure in obesity. Obes. Res. Clin. Pract. 8: e540-e548.

Emmanuel, T., Ibiam, U., Okaka, A. and Alabi, O. (2015). Effects of cement dust on the hematological parameters in Obajana cement factory workers. Eur. Sci. J. 11.

Etim, M.-A., Babaremu, K., Lazarus, J. and Omole, D. (2021). Health risk and environmental assessment of cement production in Nigeria. Atmosphere. 12: 1111.

Farheen, A., Hazari, M. A. H., Khatoon, F., Sultana, F. and Qudsiya, S. M. (2017). Hematological parameters are acutely affected by cement dust exposure in construction workers. Ann. Med. Physiol. 1: 31-35.

Hall, N. B., Myers, N. T., Reynolds, L. E., Blackley, D. J. and Laney, A. S. (2024). Women in coal mining – radiographic findings of women participants in the coal workers' health surveillance program 1970-2022. J. Womens Health (Larchmt). 33: 1219-1223.

Hill, B. G., Rood, B., Ribble, A. and Haberzettl, P. (2021). Fine particulate matter (PM2.5) inhalation-induced alterations in the plasma lipidome as promoters of vascular inflammation and insulin resistance. Am. J. Physiol. Heart Circ. Physiol. 320: H1836-H1850.

Hogg, J. C., Paré, P. D. and Hackett, T. L. (2017). The contribution of small airway obstruction to the pathogenesis of COPD. Physiol. Rev. 97: 529-552.

Jin, Q., Yao, C., Bian, Y. and Pi, J. (2022). Pb-induced eryptosis may provoke thrombosis prior to hemolysis. Int. J. Mol. Sci. 23: 7008.

Leigh, J. A., O'Neal, W. T. and Soliman, E. Z. (2016). Electrocardiographic left ventricular hypertrophy as a predictor of cardiovascular disease independent of left ventricular anatomy in subjects aged ≥65 years. Am. J. Cardiol. 117: 1831-1835.

Li, Y., Yu, B., Yin, L., Li, X. and Nima, Q. (2024). Long-term exposure to particulate matter is associated with elevated blood pressure: Evidence from the Chinese plateau area. J. Glob. Health. 14: 04039.

Malik, M. and Hnatkova, K. (2020). QT interval duration and QT/heart rate relationship. In: Malik, M. and Camm, A. J. (eds.) Dynamic Electrocardiography. 2nd ed. London: Elsevier. pp. 163-182.

Mall, M. A. (2016). Unplugging mucus in cystic fibrosis and chronic obstructive pulmonary disease. Ann. Am. Thorac. Soc. 13 Suppl. 2: S177-S185.

Meo, S. A. (2004). Health hazards of cement dust. Saudi Med. J. 25: 1153-1159.

Mwaiselage, J., Bråtveit, M., Moen, B. and Mashalla, Y. (2004). Cement dust exposure and ventilatory function impairment: an exposure-response study. J. Occup. Environ. Med. 46: 658-667.

Nku, C. O., Peters, E. J., Eshiet, A. I., Oku, O. and Osim, E. E. (2005). Lung function, oxygen saturation, and symptoms among street sweepers in Calabar, Nigeria. Niger. J. Physiol. Sci. 20: 79-84.

Nordby, K. C., Fell, A. K., Notø, H., Eduard, W., Skogstad, M., Thomassen, Y. *et al*. (2011). Exposure to thoracic dust, airway symptoms and lung function in cement production workers. Eur. Respir. J. 38: 1278-1286.

Obaji-Ogar, L. T., Nsonwu-Anyanwu, A. C. and Odum, F. A. (2020). Oxidative DNA damage and pro-inflammatory response in chronic exposure to cement dust. Asia Pac. J. Med. Toxicol. 9: 3-10.

Ogunbileje, J. O., Nawgiri, R. S., Anetor, J. I., Akinosun, O. M., Farombi, E. O. and Okorodudu, A. O. (2014). Particles internalization, oxidative stress, apoptosis and pro-inflammatory cytokines in alveolar macrophages exposed to cement dust. Environ. Toxicol. Pharmacol. 37: 1060-1070.

Omidianidost, A., Gharavandi, S., Azari, M. R., Hashemian, A. H., Ghasemkhani, M., Rajati, F. *et al*. (2019). Occupational exposure to respirable dust, crystalline silica and its pulmonary effects among workers of a cement factory in Kermanshah, Iran. Tanaffos. 18: 157-162.

Pan, C., Yan, M., Jin, H., Guo, H. and Han, X. (2021). Chronic exposure to MC-LR increases the risks of microcytic anemia: Evidence from human and mice. Environ. Pollut. 288: 117966.

Peters, J. L., Perry, M. J., McNeely, E., Wright, R. O., Heiger-Bernays, W. and Weuve, J. (2021). The association of cadmium and lead exposures with red cell distribution width. PLoS One. 16: e0245173.

Priya, S. and Suja, S. (2012). The effect of cement dust exposure on hematological and cytogenetic studies of cement workers. Res. J. Pharm. Biol. Chem. Sci. 3: 615-620.

Rahmani, A. H., Almatroudi, A., Babiker, A. Y., Khan, A. A. and Alsahly, M. A. (2018). Effect of exposure to cement dust among the workers: An evaluation of health related complications. Open Access Maced. J. Med. Sci. 6: 1159-1162.

Rampon, K. (2023). Anemia: microcytic anemia. FP Essent. 530: 12-16.

Rautaharju, P. M., Kooperberg, C., Larson, J. C. and LaCroix, A. (2006). Electrocardiographic predictors of incident congestive heart failure and all-cause mortality in postmenopausal women: the Women's Health Initiative. Circulation. 113: 481-489.

Seko, Y., Kato, T., Yamaji, Y., Haruna, Y., Nakane, E., Haruna, T. *et al*. (2021). Clinical impact of left and right axis deviations with narrow QRS complex on 3-year outcomes in a hospital-based population in Japan. Sci. Rep. 11: 8892.

Shanshal, S. A. and Al-Qazaz, H. K. (2021). Consequences of cement dust exposure on pulmonary function in cement factory workers. Am. J. Ind. Med. 64: 192-197.

Thavaraputta, S., Dennis, J. A., Ball, S., Laoveeravat, P. and Nugent, K. (2020). Relation of hematologic inflammatory markers and obesity in otherwise healthy participants in the National Health and Nutrition Examination Survey, 2011-2016. Proc. (Bayl. Univ. Med. Cent.). 34: 17-21.

Thomas, E., Guppy, M., Straus, S., Bell, K. and Glasziou, P. (2019). Rate of normal lung function decline in ageing adults: a systematic review of prospective cohort studies. BMJ Open. 9: e028150.

Variste, J., Dowling, M. M. and Quinn, C. T. (2008). Daytime steady-state hemoglobin saturation is a determinant of cerebral artery blood flow velocity in children with sickle cell anemia. Blood. 112: 1424.

Zhao, Y. and Lv, J. (2018). Basophilic stippling and chronic lead poisoning. Turk. J. Haematol. 35: 298-299.