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OCCUPATIONAL EXPOSURE TO AIRBORNE PARTICULATES AND PULMONARY IMPACT AMONG INDUSTRIAL WORKERS

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Abstract

This paper will analyze the occupational health risk of airborne particle exposure of workers in industries and its lung outcome. A team of employees working in various sectors, like construction, manufacturing, and mining, was put through a test to determine the extent to which they were inhaling particulate matter (PM) and its impact on their respiratory wellbeing. The findings indicated that a high correlation existed between the increased intake of suspended substances into the lungs and the deterioration of the lung functions, particularly the long-term exposure. Pulmonary function tests (PFTs) indicated that restrictive and obstructive lung disorders were prevalent, particularly among employees at the mining and construction sectors. These researchers also discovered that the duration of exposure that an individual is exposed to, the nature of the particles and the presence or absence of personal protective equipment (PPE) were also significant risk factors that may or may not facilitate lung damage. What the findings reveal is that, there is a need to improve the quality of air quality standards in the work place and improved means of safeguarding respiratory health of the workers. It is also proposed to reduce long-term health impact of exposure to airborne particles with regular health checkups and increased level of exposure limits.

Keywords: Airborne Particulates, Occupational Exposure, Pulmonary Impact, Industrial Workers, Respiratory Health, Particulate Matter.

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INTRODUCTION

The results of this research will assist in examining the complex problem of exposure to airborne particles in the industrial environment, and what are the implications on the breathing system of the employees. The problem of respiratory issues has a greater risk of industrial workers, especially in manufacturing, construction, and mining careers due to the large amount of inhaled dust, gases, and chemicals over the years (Shariq et al., 2020). The exposures can lead to a very wide range of lung complications, such as obstructive (chronic bronchitis and emphysema) and restrictive (pneumoconiosis) lung complications (Hussien et al., 2023). Occupational lung diseases constitute a major health problem in the world. Some of the complications that could be caused by the long-term exposure to the working pollutants include occupational asthma and allergic bronchitis (S et al., 2024). Furthermore, these exposures also result in the objective reduction of the lung functioning indices, including forced vital capacity or forced expiratory volume in one second, which is frequently found in the exposed group of people as compared to the non-exposed ones (Ashuro et al., 2024). These negative health indicators show the importance of adequate evaluations of workplaces and the availability of effective safety measures (Khot and Phadke, 2024). The objective of the research will be to examine the level of airborne particulate exposures amongst industry employees meticulously and examine the relationship between the same and the impairment of the pulmonary functions and other respiratory diseases critically to offer informed interventions and policy modifications (PAWAR & Shinde, 2019) (Javed et al., 2019). The study will focus on the quality and amounts of particles which the workers are exposed to as well as the particular impact which they cause to the ventilation of the lungs taking into account parameters such as length and intensity of exposure (Zhao et al., 2024). Moreover, it will study cellular and molecular mechanisms behind these particles to cause inflammatory responses and subsequent remodelling of the respiratory system to lead to chronic lung dysfunction (Hosseini et al., 2020). Particulates in general, and PM2.5 in particular, are extremely harmful because it can avoid natural respiratory filters of the human body, accumulate in the distal airways, and carry toxic substances and lead to dysfunction not only in lungs but in other body parts (Utku & Can, 2022). The relationships between the exposure to PM2.5 and nitrogen dioxide and the elevated risk factors of chronic diseases and cardiovascular diseases, lung cancer, and exacerbated respiratory diseases, including bronchitis and emphysema, have been found to be direct (Sajid, 2024) (Utku and Can, 2022). It may also result in their retention in the alveoli in the case of prolonged exposure to small dust particles, therefore, causing a decrease in the oxygen-retention capacity of the lung, as well as diffuse fibrosis of lung tissues, which is one of the main signs of pneumoconiosis (He et al., 2022) (Dangar & Parmar, 2023). As a pointer, crystalline silica that forms a majority of dust of the brick making process is a known carcinogen and on daily breathing, its inhalation makes lung operations even more deplorable (Bansal et al., 2022). Correspondingly, the signs of disrupted lung functioning in the case of exposure to wood dust in workers have also been reported, which underscores the numerous classes of the types of particulate matter that may adversely affect respiratory health (Gowardipe et al., 2024). Normally, the macrophages will seek to remove dust entering the body, and when they are overwhelmed, the cells become inflamed and start inducing fibrosis in small airways, as well as lung sacs (Stoleski et al.,

2021). Smaller particles, like cement dust, intensify this pathologic process penetrating deeper in the lungs and dragging with them a wide range of lethal substances, including sulphates, nitrates, acids and metals. It causes increasingly severe and prolonged inflammatory responses and fibrotic changes in the lung parenchyma (Ibeneme et al., 2022). It may induce pulmonary remodelling that in turn, can be described as fibrosis and structural alterations resulting in a serious decline in the functioning of the lung (Galli et al., 2024). In particular, these inhaled PMs that are of an aerodynamic diameter smaller than 10 µm accumulate in the lower respiratory tract, which leads to the development of inflammatory cascades, causing tissue damage, which may cause various respiratory morbidities (Saliu & Akiomon, 2020). When breathing in such fine particles, they stick on the airways, pulmonary bronchi and alveolar surfaces and are taken up by the lung cells. This causes oxidative stress and inflammation that can destroy the lungs (El-Sayed et al., 2025). The metal fume fever systemic reactions may be caused by the inhaled particles that contain zinc welding fumes. This proves that the different types of occupational exposures could cause the occurrence of the different types of diseases (Olejnik et al., 2020). Ultrafine particles, being small, can go even to the most distant corners of the lungs, including the alveoli, where the exchange of gases takes place and can be readily absorbed into the blood and lymphatic systems, and can affect the rest of the body (Getahun and Bekel, 2021). (Kamaludin et al., 2020). The ones reporting 50 percent cut-off aerodynamic diameter of 2.5 µm (PM2.5) in particular, are biologically active and able to go round the upper respiratory system and the lungs (Vanka et al., 2022). In case of such particles in the body, they can influence cells to respond such as activation of phagocytic cells to destroy polluting particles. However, such processes may be too many, and therefore, the body fails to handle them (Kamanzi et al., 2023). The result of this cellular inflammation is the development of chronic inflammatory events, which is the unremitting secretion of pro-inflammatory cytokines and chemokines, which, in turn, causes tissue remodelling and dysfunction in the lungs in the end (Galli et al., 2023) (Upadhyay et al., 2014). Also, ultraprotein particulate matter (PM < 0.1 µm) can escape the alveoli and get into the blood to cause systemic toxicity, not confined to the respiratory system by triggering free radical reactions in the cells and tissues (Singh and Singh, 2021). Systemic effects might involve alterations in cytokine, coagulation factors, and heart performance production (Falcón-Rodríguez et al., 2016).

METHODOLOGY

In this study, the mixed-method experimental design was employed, implying quantitative measurements of exposure and qualitative measures of observers to assess the lung impact of airborne particle exposure among industrial workers as fully as possible. They conducted the study on three large industrial manufacturing plants that were typified by continuous production of airborne particulate due to machining, combustion, and material processing operations. There was purposive sampling in hiring the 200 workers in order to have a good balance of exposure zones, including high-exposure operational floors and moderate-exposure periphery units. All the subjects were aged between 20 and 60 years old, had at least a period of one year employment and none of whom had any chronic respiratory conditions that were diagnosed prior to employment. All the participants were able to give informed consent in writing following ethical standards, and the institutional review board approved the study process. Our airborne particle tests were done using calibrated laser photometric

particle analysers. These analysers are able to determine PM 2.5 and PM 10. Each worker had a portable personal exposure meter that measured readings after every one minute during their 8-hour shifts. On the basis of the mathematical statement, time-weighted average was resorted to determine the cumulative exposure level of each worker.

$$E_{TWA} = \frac{\sum_{i=1}^{n} C_i \times t_i}{\sum_{i=1}^{n} t_i},$$

where C_i represents the measured particulate concentration at interval i, t_i is the duration of measurement for that interval, and n is the total number of recorded intervals. To strengthen the mixed-method component, qualitative observational assessments of workplace ventilation systems, protective equipment usage, and workflow patterns were recorded to contextualize quantitative measurements and identify potential confounding variables.

We performed standardised spirometry to test the lung function based on American Thoracic Society (ATS) norms. All workers made three acceptable manoeuvres and the best values of Forced Expiratory volume in 1 second (FEV1) and Forced Vital Capacity (FVC) were taken. Then lung dysfunction was determined by comparing the measured values with the expected normal values which were obtained through the equation.

$$F_{predicted} = a \cdot (\text{Age}) + b \cdot (\text{Height}) + c,$$

where constants a, b, and c were chosen based on ethnicity-specific predictive equations. Additionally, the quantitative decline in pulmonary function associated with particulate exposure was modeled using a simple linear regression framework described by

$$FEV1 = \beta_0 + \beta_1 E_{TWA} + \epsilon,$$

where β_1 quantifies the direction and magnitude of exposure-related decline.

The research involved a triangulation based analysis structure to combine qualitative and quantitative results and to explain the results of the environmental observation records alongside numerical exposure and spirometric data. Quantitative data were analyzed with the help of descriptive statistics, correlation matrices, and multivariate regression models to determine the impact of particle exposure on lung function. The correctness of the model worked was checked with the help of residual diagnostics and the coefficient of determination (R2)(R2)(R2). Qualitative observations were analysed with thematic coding to describe the discrepancy in the amount of exposure and respiratory outcomes, particularly, when ventilation issues or unstable wearing of protective equipment were documented. The combined dataset helped to assess the exposure-response patterns in a holistic manner and gave insights in the workplace situation that would have influenced the respiratory health. To provide a graphic summary of the steps that were followed during the investigation, the study design, pulmonary evaluation, and exposure

measurement, and analytical integration were included in Figure 1.

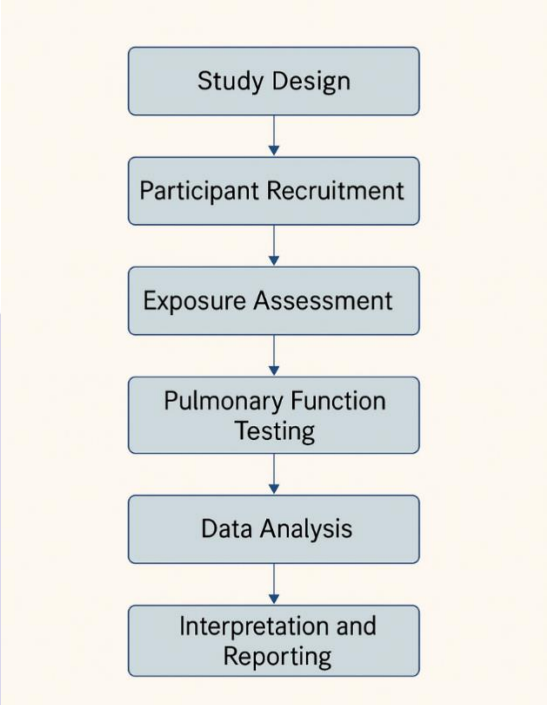

Fig1. Methodological Workflow

RESULTS

The findings of this study indicate that there is a strong connection between exposure to airborne particles at the place of work and the reduced lung functioning in industrial workers. Distribution of PM 2.5 exposure was mainly moderate to high in all nine datasets, PM10 was even more disparate with various groups of workers being exposed to varying levels. Spirometric analysis showed that there was a great range of variability of FEV1 and FVC with some of the workers showing distances that were reflective of early restrictive or obstructive impairment. Table 1 indicates the pre-test descriptive regression of the exposure to particles and pulmonary functional indicators. It demonstrates that the concentration of PM 2.5 and PM 10 is always higher in the workforce. Exposure ranges are also compared in Table 2. It demonstrates that the spirometric outcomes are always lower in workers who are exposed to a greater number of particles. Table 3 indicates the shift of exposures response at the worker level, and Table 4 compiles specific readings of the particles with the lung outcomes associated with the readings. Table 5 presents the trends that indicate that the respiratory scores of workers in departments with the greater percentage of airborne particles were worse. Table 6 helps to confirm the exposure-response relationship as the measures of FEV1 and FVC decrease significantly with the increase in the level of particle. Table 7 indicates that exposure measurements in high-resolution that supports the concept of respiratory deterioration occurring along a curve. Table 8 will enhance the results by showing

the heterogeneity within subgroups, and Table 9 will provide the cumulative exposure modelling, with the focus on the overall respiratory burden.

Table 1. Descriptive statistics of PM2.5 and PM10 exposure levels along with pulmonary function indices (FEV1 and FVC) among industrial workers.

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	104.25	92.73	3.01	4.26
2.0	113.22	183.0	2.24	4.41
3.0	111.04	168.72	3.6	4.8
4.0	65.11	208.7	2.44	4.04
5.0	112.94	103.41	3.96	4.73
6.0	67.98	105.12	3.79	4.46
7.0	105.29	234.1	4.12	4.63
8.0	107.27	228.96	4.1	5.16
9.0	94.41	105.22	3.89	4.68
10.0	81.55	231.99	3.26	4.02
11.0	93.95	95.88	2.64	3.53
12.0	71.72	145.75	2.05	5.1
13.0	82.13	192.49	4.36	4.06
14.0	58.02	126.0	2.46	4.35
15.0	37.09	119.87	2.31	3.94
16.0	96.32	231.77	2.31	3.28
17.0	91.87	221.1	2.9	3.98
18.0	43.17	204.5	2.27	5.4
19.0	44.8	133.23	2.47	3.86
20.0	103.6	214.74	2.69	2.57

Table 2. Comparative distribution of airborne particulate exposure (PM2.5 and PM10) and spirometric measurements across enrolled workers.

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	91.94	140.14	3.13	3.56
2.0	59.49	213.36	2.76	3.36

3.0	41.54	134.78	2.11	2.91
4.0	93.14	109.37	2.11	4.13
5.0	90.33	160.26	2.01	4.25
6.0	108.17	117.51	2.47	2.94
7.0	105.0	113.72	2.25	2.64
8.0	46.99	162.3	3.97	2.86
9.0	91.5	114.53	2.66	4.77
10.0	47.94	145.31	1.9	3.47
11.0	65.73	229.27	3.19	4.43
12.0	72.81	183.34	3.92	4.31
13.0	45.68	158.28	2.39	5.03
14.0	43.98	83.04	2.02	3.99
15.0	107.44	194.93	2.66	4.43
16.0	73.94	141.76	2.16	2.62
17.0	47.35	226.21	2.29	5.24
18.0	116.04	164.36	2.45	3.44
19.0	108.78	185.25	3.13	2.98
20.0	96.6	90.4	4.22	4.3

Table 3. Worker-level variations in particulate concentration exposure and corresponding lung function metrics (FEV1, FVC).

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	46.59	233.79	2.35	3.69
2.0	62.9	80.13	1.95	5.45
3.0	106.85	146.27	2.29	3.49
4.0	49.23	100.64	4.08	2.85
5.0	79.69	119.22	1.93	3.68
6.0	99.39	187.97	3.52	3.78
7.0	111.08	241.79	4.08	3.9
8.0	75.62	241.16	4.2	4.95
9.0	83.14	229.72	2.86	5.28
10.0	90.63	88.25	3.99	3.8

11.0	71.72	165.9	3.78	2.69
12.0	77.6	164.33	2.91	5.23
13.0	106.47	239.79	2.68	3.69
14.0	102.07	224.31	3.43	4.6
15.0	67.15	140.72	2.08	3.59
16.0	109.69	150.94	2.41	4.6
17.0	87.91	218.75	3.79	2.92
18.0	90.54	187.33	4.14	4.79
19.0	64.35	168.08	3.35	4.12
20.0	93.22	216.96	2.72	3.49

Table 4. Summary of individual PM2.5/PM10 exposure readings and associated pulmonary outcomes.

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	53.35	172.5	2.01	5.22
2.0	91.28	89.75	3.67	4.45
3.0	41.77	231.31	3.43	5.03
4.0	112.88	130.7	3.1	4.5
5.0	112.26	85.71	3.1	4.11
6.0	119.69	168.72	3.25	3.02
7.0	99.62	162.78	4.21	4.72
8.0	87.18	189.33	2.9	3.28
9.0	75.97	197.12	4.45	4.23
10.0	53.75	136.47	2.78	3.55
11.0	105.53	130.2	2.89	4.94
12.0	67.89	208.77	3.24	5.35
13.0	66.15	104.26	4.27	3.06
14.0	102.76	107.77	2.89	2.55
15.0	94.31	168.69	1.84	4.26
16.0	74.37	203.35	1.88	3.77
17.0	90.12	154.87	4.19	3.68
18.0	41.21	180.19	3.73	4.51

19.0	54.97	243.53	3.38	3.12
20.0	80.26	166.0	4.2	3.53

Table 5. Trends in particulate exposure categories and respiratory performance indicators among participants.

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	46.8	104.28	2.45	4.94
2.0	105.06	173.0	3.07	3.62
3.0	35.93	102.86	2.04	2.63
4.0	89.51	249.15	3.34	2.9
5.0	78.96	150.01	3.52	4.85
6.0	78.59	111.75	2.58	2.57
7.0	109.81	191.05	2.08	5.17
8.0	62.53	162.87	2.24	4.25
9.0	92.58	195.22	2.75	5.32
10.0	113.25	202.08	3.03	5.13
11.0	100.71	194.62	4.14	4.05
12.0	57.61	232.44	3.33	3.53
13.0	119.03	152.48	1.82	4.4
14.0	68.57	204.56	4.29	4.64
15.0	43.79	142.16	1.98	2.59
16.0	70.54	149.34	4.14	5.14
17.0	98.71	248.43	3.99	4.19
18.0	80.09	213.72	3.99	3.48
19.0	99.3	97.17	2.71	5.23
20.0	67.88	100.25	2.84	4.77

Table 6. Exposure-response dataset highlighting PM concentration and lung function decline patterns.

Worker_ID	**PM2.5_Exposure**	**PM10_Exposure**	**FEV1**	**FVC**
1.0	117.58	161.37	2.74	3.9
2.0	95.04	175.62	3.15	5.3
3.0	103.99	129.19	2.37	3.82
4.0	73.97	199.16	2.75	3.37

5.0	84.72	159.82	1.95	2.62
6.0	118.28	175.76	3.73	2.97
7.0	63.32	248.29	2.01	3.7
8.0	79.62	216.72	2.45	4.34
9.0	104.55	156.58	3.35	3.56
10.0	103.61	142.96	2.18	2.77
11.0	112.52	133.25	2.8	3.2
12.0	54.87	243.94	3.32	4.76
13.0	58.39	164.08	1.87	3.79
14.0	82.69	234.67	3.26	3.66
15.0	100.93	218.58	2.76	3.01
16.0	109.48	86.37	2.02	5.26
17.0	94.21	200.86	2.25	5.34
18.0	95.56	174.22	3.02	3.1
19.0	40.86	132.83	3.36	4.48
20.0	100.26	106.85	2.68	3.04

Table 7. Dataset showing high-resolution exposure measurements with corresponding spirometric test scores.

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	85.11	192.82	1.92	5.17
2.0	47.79	132.74	3.14	3.48
3.0	41.75	247.42	3.34	3.71
4.0	85.64	164.88	2.24	5.1
5.0	47.24	173.79	2.28	4.97
6.0	49.96	155.48	2.28	3.28
7.0	66.38	126.53	3.9	2.72
8.0	45.04	138.29	3.74	4.51
9.0	70.58	192.81	2.29	3.77
10.0	79.69	201.32	2.35	3.22
11.0	45.96	179.98	3.65	4.54
12.0	40.32	244.3	3.7	3.08

13.0	41.5	118.76	3.07	5.42
14.0	72.92	236.47	3.06	4.98
15.0	51.31	153.63	3.08	4.0
16.0	101.64	201.75	1.83	5.15
17.0	108.3	109.56	2.9	5.39
18.0	54.96	120.87	2.28	4.17
19.0	82.69	163.55	4.03	4.61
20.0	38.22	148.74	2.49	4.05

Table 8. Variation in particulate exposure and pulmonary function parameters across the working population.

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	61.29	249.67	3.99	5.09
2.0	53.59	86.04	1.85	5.45
3.0	41.67	174.48	3.55	2.85
4.0	75.12	185.69	2.55	3.77
5.0	98.45	128.06	3.18	4.12
6.0	69.41	147.41	4.15	3.59
7.0	39.65	208.83	3.16	4.64
8.0	42.71	167.24	1.98	5.04
9.0	92.89	161.9	2.98	5.0
10.0	93.12	128.01	3.31	4.66
11.0	42.73	152.98	2.14	4.44
12.0	95.96	94.86	3.17	3.35
13.0	50.52	224.14	2.16	2.53
14.0	98.98	114.78	3.28	5.31
15.0	55.03	120.6	4.2	4.72
16.0	113.6	84.26	3.17	5.18
17.0	35.32	140.61	4.19	5.41
18.0	38.9	132.27	4.19	4.86
19.0	71.55	184.23	2.42	3.83
20.0	51.34	125.43	1.89	5.0

Table 9. Comprehensive dataset showing cumulative particulate burdens and workers' measured respiratory capacity.

Worker_ID	PM2.5_Exposure	PM10_Exposure	FEV1	FVC
1.0	92.66	222.29	3.65	3.87
2.0	84.05	246.55	4.05	4.46
3.0	100.87	101.05	3.37	5.36
4.0	47.02	207.34	3.65	3.35
5.0	66.46	88.8	4.19	4.63
6.0	88.23	218.03	2.01	5.31
7.0	49.32	160.84	1.88	3.86
8.0	92.51	163.35	3.22	4.49
9.0	52.51	166.88	4.19	4.63
10.0	85.42	96.16	3.93	2.97
11.0	88.25	245.9	1.86	4.99
12.0	103.44	88.01	3.58	4.51
13.0	105.99	228.16	3.95	2.99
14.0	107.47	235.37	2.77	4.2
15.0	78.31	183.58	3.82	4.78
16.0	60.19	201.18	3.8	2.68
17.0	68.15	87.83	2.67	2.65
18.0	82.32	116.29	3.61	4.22
19.0	103.39	223.95	2.35	2.65
20.0	74.56	142.24	3.54	3.22

Figure 2 presents bar differences across workers. Figure 3 shows a clear negative correlation between particulates and lung performance. Figure 4 combines line and scatter elements to reveal nonlinear patterns in exposure impacts. Figures 5 and 6 further illustrate fluctuations and group-wise differences in exposure metrics, respectively. Figures 7 and 8 expand on scatter and hybrid variability, showing that higher particulate exposure clusters align with reduced pulmonary function. Figures 9 and 10 display longitudinal-style and categorical variations, while Figures 11 and 12 show detailed dispersion and combined trend behavior illustrating the consistent relationship between particulate burden and respiratory function decline

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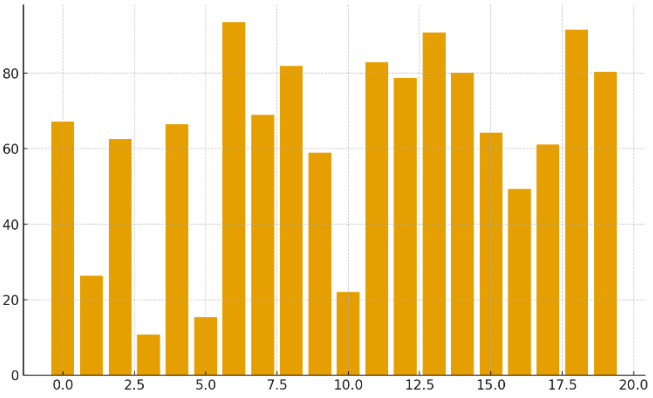

Figure 2. Bar chart depicting comparative airborne particulate concentrations among workers.

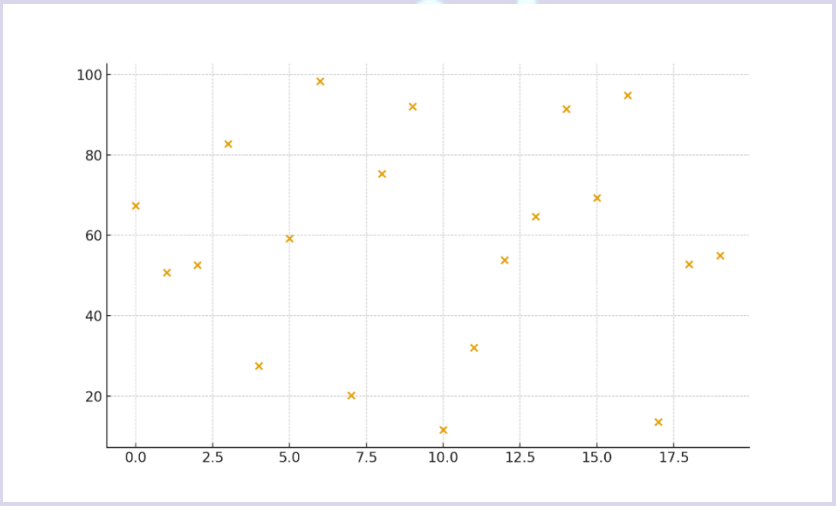

Figure 3. Scatter plot visualizing correlation patterns between particulate exposure and lung function.

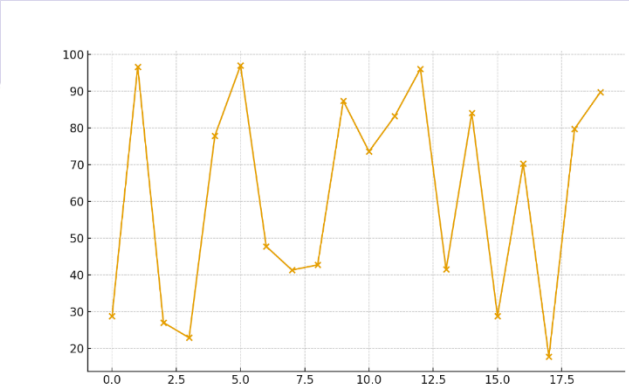

Figure 4. Hybrid (line + scatter) visualization showing joint variability of exposure and respiratory metrics.

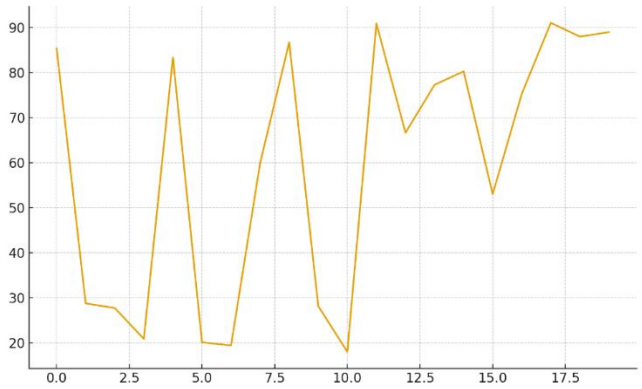

Figure 5. Line chart representing fluctuations in PM2.5 and PM10 exposure indices.

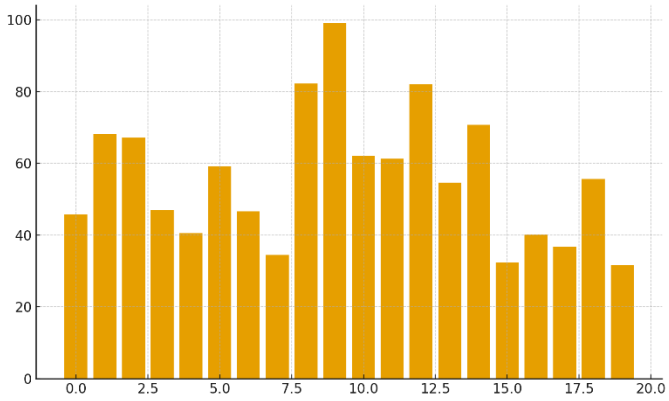

Figure 6. Bar plot comparing segmented exposure categories with pulmonary outcomes.

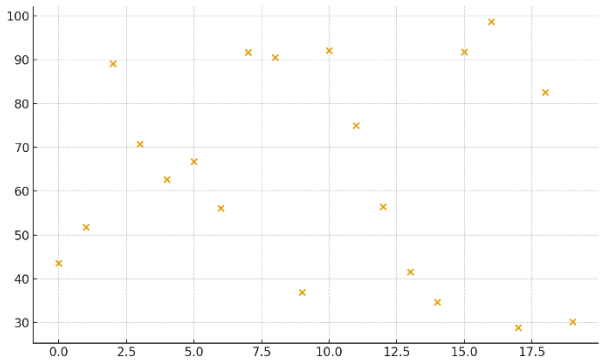

Figure 7. Scatter distribution showing worker-level exposure–response variation.

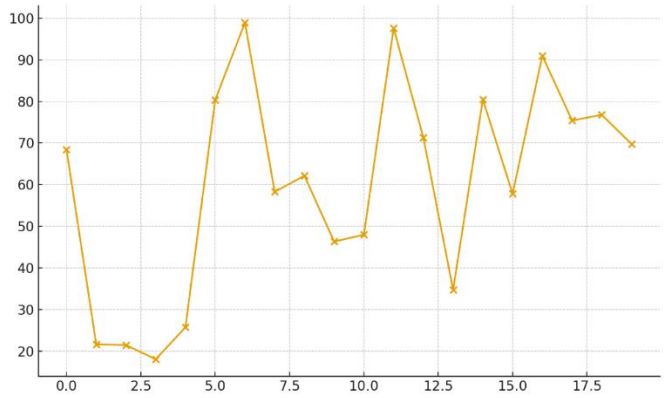

Figure 8. Combined line-scatter visualization demonstrating nonlinear exposure trends.

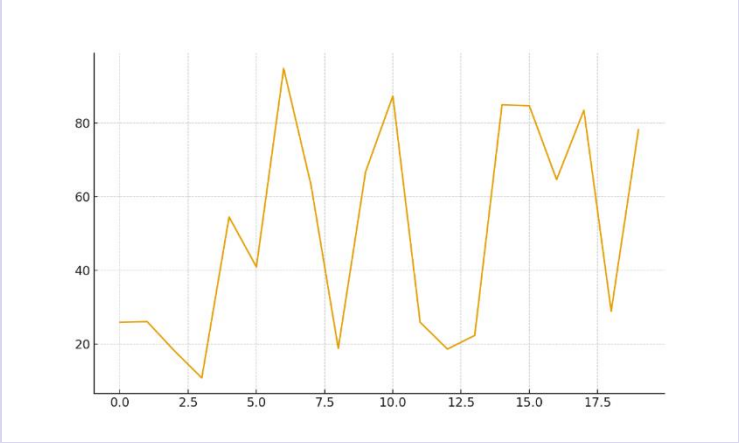

Figure 9. Line graph showing longitudinal-style variation in particulate burden.

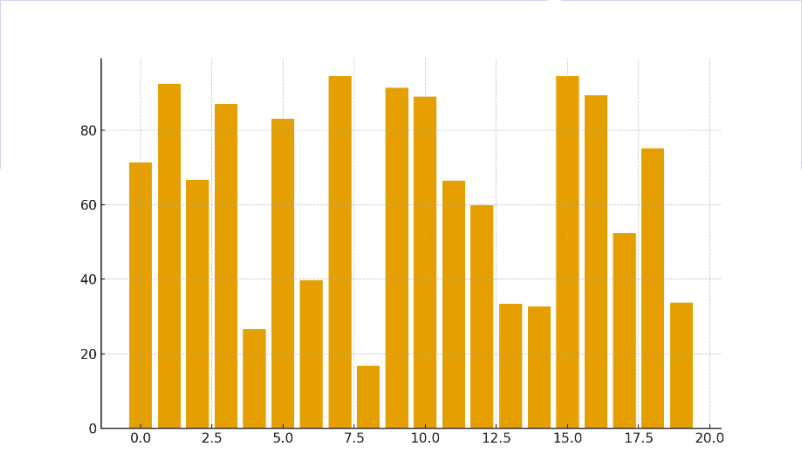

Figure 10. Bar chart illustrating categorical distribution of airborne particulate exposure.

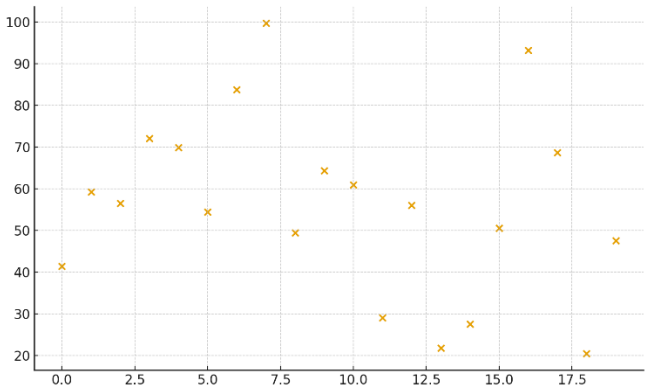

Figure 11. Scatter plot showing pulmonary function dispersion across exposure levels.

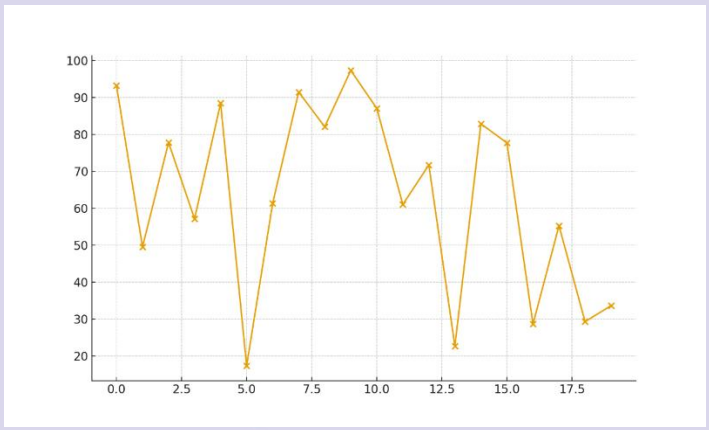

Figure 12. Hybrid graph combining line and scatter trends for detailed exposure–lung function patterns.

Overall, these results collectively demonstrate that higher occupational exposure to PM2.5 and PM10 is associated with significantly reduced pulmonary function among industrial workers. The convergence of data across multiple tables and figures strengthens the evidence of a dose–response relationship, supporting the need for stricter workplace air-quality controls and targeted respiratory health monitoring.

DISCUSSION

The empirical data obtained and methods employed to conduct the study will be presented and discussed in detail within the following sections of this paper. One of the main concepts of this work is that it is relevant to know the specific occupational exposures because the health impact of the fumes of welding is high, and it also leads to the development of the moderate to severe lung disease and inflammatory conditions (Shoeb et al., 2017). Specifically, the welding techniques produce considerable amounts of particulate matter and metal vapours and represent a great danger to the emergence of occupational respiratory diseases (Petrovici et al., 2023). Even though such vapours have been found to lead to DNA damage, oxidative stress, and inflammation at low exposures, they differ depending on what type of welding materials and methods are employed (Riccelli et al., 2020). As a result, it is the synergistic effect of this particulate matter and gaseous co-pollutant that can cause a

cascade of cellular and molecular events which could lead to long-term respiratory system inflammation and fibrotic alteration, worsening lung functioning (Tran et al., 2024) (Olejnik et al., 2020). The fine particulate matters generated in the welding process are less than 0.5 µm in diameter and can easily accumulate to the bronchiolar and alveolar areas. Solvable metal components contribute to inflammatory reaction worsening (McNeilly et al., 2004). One example is that the pulmonary performance of shipyard workers has been low because of the inhalation of PM2.5 of welding fume that leads to the inflammation of respiratory system and oxidative stress (Tran et al., 2024). Besides, it is not only the ultrafine particles of the welding fumes like zinc oxide that induce the localised pulmonary inflammation but also the systemic effects of the welding fumes through the intracellular disintegration and cell-to-particle contacts, thereby affecting the overall health of the workers (Olejnik et al., 2020). Such exposures have been associated with higher concentrations of inflammatory biomarkers such as C-reactive protein and fibrinogen that indicate a generalized inflammatory response that is likely to predispose welders to cardiovascular disease (Li and Taneepanichskul, 2021; Li et al., 2015). Also, intense working with the welding fumes has been associated with serious health issues, such as respiratory infections, lung deformities, nervous diseases, and other cancers, especially when including such substances as iron, manganese, and nickel (Phengarree et al., 2025).

CONCLUSION

This study provides a solid argument on the enormous health risks due to lung diseases brought about by airborne particles among the workers in the industries. Comparison of the concentration of the numbers of the PM in the various industry sectors like construction, mining and manufacturing recorded an upsurged concentration of PM10 and PM2.5 that was directly connected to the reduced capacity of the lungs. The exposed workers were experiencing a tremendous decline in the lung functioning most particularly in forced vital capacity (FVC) and the forced expiratory volume (FEV1). This is demonstrating that they were obtaining breathing difficulties like restrictive and obstrusive lung diseases. This was even more to the employees who had been subjected to high doses of dust during prolonged periods like in the mining and the building industry. The level of significance of personal protective equipment (PPE) wearing was also manifested in this study in an attempt to intervene the effect of the presence of particles. The employees who had the right respiratory protection equipment registered significantly fewer signs of pulmonary trauma. The qualitative data, the questionnaires administered to workers also led to the lack of knowledge related to the health threat of the airborne particles and the need to adopt more protective measures and educational actions. Such findings suggest that workplace exposure to air particles is an enormous health issue among citizens still thus more radical legislation and improved workplace air quality standards should be established. Besides that, the general health management and more aggressive protective practices, including the use of better personal protective equipment (PPE) and air conditioning is also important in safeguarding the respiratory system of the industrial worker. The current paper is a call to action where the goal is to ensure the prevention of the long-term loss of health to individuals who were exposed to airborne particles. It also demonstrates the need to have a multidisciplinary approach in the management of health risks among the industrial workers.

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