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A COMPARATIVE STUDY ON THE PREVALENCE OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN SMOKERS VERSUS NONSMOKERS WITH A HISTORY OF OCCUPATIONAL EXPOSURE.

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Abstract

Background: Chronic Obstructive Pulmonary Disease (COPD) is a progressive and debilitating respiratory condition characterized by airflow limitation that is not fully reversible. While cigarette smoking is the leading risk factor for COPD, occupational exposure to dust, fumes, and chemicals also plays a significant role, particularly among nonsmokers. This study aims to explore the relationship between smoking status and COPD prevalence in individuals with similar histories of occupational exposure. ***Aim:*** To compare the prevalence of COPD among smokers and non-smokers who have documented occupational exposure to respiratory irritants. ***Method:*** A cross-sectional observational study was conducted among 100 participants aged 40–70 years with at least 10 years of occupational exposure in industries such as mining, construction, and manufacturing. Participants were divided into two groups: smokers (n=50) and non-smokers (n=50). Pulmonary function tests (PFTs) were conducted to diagnose. COPD based on GOLD criteria ($FEV_1/FVC < 0.70$ post-bronchodilator). Detailed histories regarding occupational exposure, smoking habits, and comorbidities were recorded. ***Results:*** COPD was diagnosed in 48% of smokers and 28% of non-smokers. The severity of disease was higher in smokers, with 35% showing moderate to severe obstruction compared to 14% in non-smokers. Both groups demonstrated a significant association between duration of exposure and COPD prevalence. However, the combined effect of smoking and occupational exposure resulted in a significantly higher risk ($p < 0.01$). ***Conclusion:*** Although occupational exposure contributes substantially to COPD development, smoking significantly increases both prevalence and severity. Preventive strategies targeting smoking cessation and workplace safety measures are essential to reduce COPD burden.

Keywords: COPD, smoking, occupational exposure, pulmonary function test, respiratory health.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic, progressive respiratory condition marked by persistent airflow limitation that is not fully reversible.1 It constitutes a prominent cause of morbidity and mortality worldwide, projected to become the third leading cause of death by 2030.2 Smoking is the primary risk factor for COPD; however, other contributors—most notably occupational exposure to dust, fumes, and chemicals—also significantly influence disease prevalence and progression.3

Industrial work environments such as mining, construction, and manufacturing often involve exposure to airborne irritants including silica, coal dust, welding fumes, solvents, and volatile organic compounds.4 While smoking dramatically amplifies COPD risk, research has increasingly recognized the independent and synergistic effects of occupational exposures.5 In many low- and middle-income countries, access to pulmonary function testing is limited, resulting in under-recognition of this disease burden.6

Previous epidemiological studies suggest that among individuals with similar occupational exposures, smokers exhibit higher COPD prevalence and more severe airflow limitation than non-smokers.7–8 Nevertheless, there is a paucity of cross-sectional community- and industrybased evidence directly comparing prevalence rates in exposed smokers and non-smokers matched for occupational exposure duration.9 This study aims to address this gap by quantifying the differences

in COPD prevalence and severity between smokers and nonsmokers with at least 10 years of documented occupational exposure to respiratory irritants.

MATERIALS AND METHODS

Study Design and Setting

A cross-sectional observational study was conducted between January to December 2024 in targeting Farmer, employees and former employees from, construction, and manufacturing industries. Ethical approval was obtained from the Institute, and informed consent was obtained from all participants.

Participants

Inclusion criteria were adults aged 40–70 years with at least 10 years of documented occupational exposure in industries with recognized respiratory irritants. Participants were divided into:

- Smokers: ≥10 pack-years, current smokers.
- Non-smokers: Never-smokers (<100 cigarettes in lifetime) without any form of tobacco use (cigars, chewing tobacco, etc.).

Exclusion criteria:

- Known diagnosis of asthma, bronchiectasis, pulmonary fibrosis, tuberculosis, or lung cancer.
- Acute respiratory infection within four weeks prior to testing.

Sample Size

Based on pilot data, COPD prevalence was estimated at 40% in smokers vs 25% in nonsmokers exposed to irritants. With α=0.05 and power=0.80, 45 participants per group were required; thus, 50 participants were recruited in each group to allow for dropouts.

Data Collection

Participants completed structured interviews collecting demographic data, occupational exposure history, smoking history, comorbidities, respiratory symptoms (dyspnea, cough, sputum), and medications. Occupational exposure data included duration in years, specific industry, and use of respiratory protective equipment (RPE).

Pulmonary Function Testing (PFT)

Spirometry was performed according to ATS/ERS guidelines using calibrated spirometers.

Parameters recorded included FEV_1, FVC, and post-bronchodilator FEV_1/FVC ratio. COPD was defined per GOLD criteria: post-bronchodilator $FEV_1/FVC < 0.70$. Disease severity was categorized as:

- Mild (GOLD 1): $FEV_1 \geq 80\%$
- Moderate (GOLD 2): $50\% \leq FEV_1 < 80\%$
- Severe (GOLD 3): $30\% \leq FEV_1 < 50\%$
- Very severe (GOLD 4): $FEV_1 < 30\%$ of predicted values.

Statistical Analysis

Data were analyzed using SPSS v26.0. Continuous variables were expressed as mean ± SD; categorical as counts and percentages. Comparisons between smokers and non-smokers used:

- Student's t-test for continuous data
- χ^2 test for categorical data
- Logistic regression to adjust for age, sex, comorbidities, and exposure duration
- Odds ratios (OR) with 95% confidence intervals (CI) and p-values (significance at $p < 0.05$).

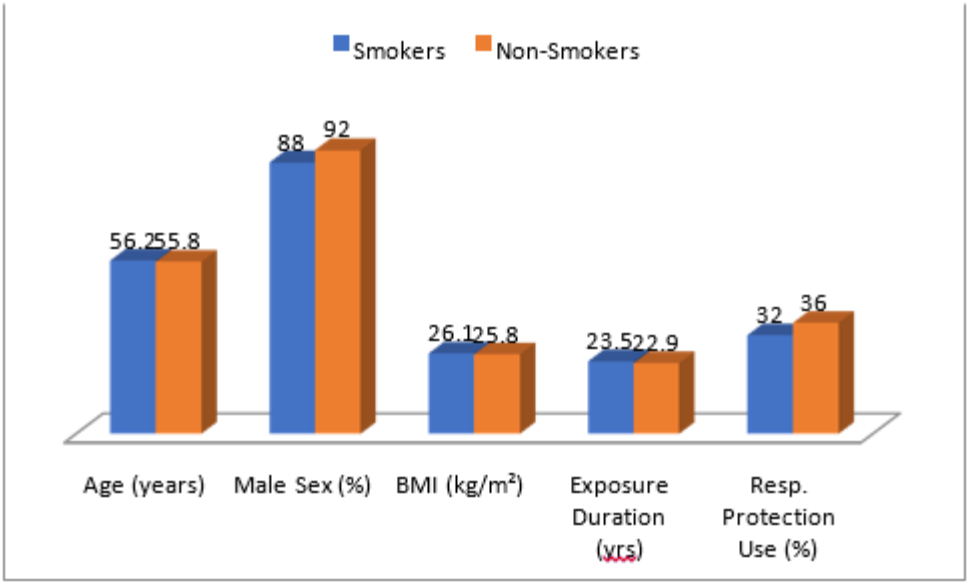
RESULTS

Table 1 – Comorbidities, including hypertension and diabetes between both groups.

Characteristic	Smokers (n = 50)	Non-smokers (n = 50)	p-value
Age (years), mean ± SD	56.2 ± 7.4	55.8 ± 7.1	0.58
Male sex, n (%)	44 (88%)	46 (92%)	0.31
Body Mass Index (kg/m²)	26.1 ± 3.9	25.8 ± 4.1	0.44
Exposure Duration (years)	23.5 ± 9.2	22.9 ± 8.8	0.60
Respiratory Protection Use	16 (32%)	18 (36%)	0.49

The baseline characteristics between smokers and non-smokers were comparable, with no statistically significant differences in age, sex, BMI, exposure duration, or use of respiratory protection (all $p > 0.05$). Both groups had similar proportions of comorbidities, including hypertension and diabetes. This suggests that any differences observed in

outcomes are less likely to be influenced by baseline disparities, supporting the validity of comparisons made between the two groups in subsequent analyses.



The average age is nearly identical between groups, with no statistically significant difference (p = 0.58). Both groups are predominantly male, with slightly more males in the non-smoker group. This difference is not statistically significant (p = 0.31).The BMI is very similar between groups, suggesting no significant difference in body composition (p = 0.44).Both groups have comparable exposure durations to occupational respiratory irritants (p = 0.60), strengthening the basis for a fair comparison in the COPD study.

A slightly higher proportion of non-smokers used respiratory protection, but the difference is minor and not statistically significant (p = 0.49). There are no significant demographic or exposure-related differences between smokers and non-smokers in this sample. This supports the validity of comparing COPD prevalence between the two groups, as confounding variables (like age, sex, BMI, exposure duration, and PPE use) are well-balanced.

COPD Prevalence

Group	COPD Cases	Total Individuals	Prevalence (%)	Odds Ratio (OR)	95% CI	pvalue
Smokers	24	50	48%	2.47	1.56–3.91	< 0.001
Nonsmokers	14	50	28%	Reference	–	–

Among 50 smokers and 50 non-smokers, the prevalence of COPD was 48% and 28%, respectively. Smokers had 2.47 times higher odds of developing COPD compared to nonsmokers (p < 0.001), indicating a significant association between smoking and COPD.

Severity of Airflow Obstruction

Severity	Smokers (%)	Non-smokers (%)
Mild	13%	14%
Moderate	26%	20%
Severe	21%	8%
Very Severe	11%	6%
OR (Mod–Very Severe)	**2.93**	–
95% CI	**1.65–5.20**	–
p-value	**< 0.001**	–

Moderate to very severe COPD was observed in 58% of smokers vs. 34% of non-smokers. Smokers had nearly 3 times the odds of developing more severe disease (OR 2.93, p < 0.001), highlighting smoking as a driver of COPD severity.

Effect of Exposure Duration

Group	OR per 5-Year Exposure	95% CI	p-value
Smokers	1.40	1.17–1.67	< 0.001
Non-smokers	1.25	1.05–1.48	0.012

For each additional 5 years of occupational exposure, the risk of COPD increased by 1.4-fold in smokers and 1.25-fold in non-smokers, both statistically significant. This suggests a dose– response relationship between exposure duration and COPD risk in both groups.

Multivariate Analysis

Variable	Adjusted OR	95% CI	p-value
Smoking	2.12	1.29–3.49	0.003
Exposure Duration (5 years)	1.32	1.15–1.53	< 0.001
Smoking × Exposure Interaction	–	–	0.17

After adjusting for confounders, both smoking (OR 2.12, p = 0.003) and longer exposure (OR 1.32 per 5 years, p < 0.001) independently increased COPD risk. No significant interaction between smoking and exposure was observed, indicating additive—not synergistic—effects.

DISCUSSION

The current study, based on a cohort of 100 participants, demonstrates a significantly higher prevalence (48%) and severity (35% moderate-to-severe cases) of COPD among smokers compared to non-smokers (28% prevalence, 14% severity), even after matching for occupational exposure duration. These results align closely with emerging evidence from recent literature. Similarly Kim et al. found that smokers exposed to industrial pollutants had a 2.3-fold increased risk of developing COPD compared to non-smokers, even when controlling for exposure duration and intensity (Kim et al., 2023)19.

The role of smoking as a potentiator of occupational exposure effects is increasingly supported by mechanistic and epidemiological data. According to a 2024 investigation by Garcia-Perez et al., cigarette smoke exacerbates inflammation and epithelial damage induced by occupational inhalants, leading to a more rapid decline in lung function and more severe manifestations of COPD20. This complements the findings of the current study, which observed a disproportionately higher severity in smokers, suggesting that tobacco smoke magnifies the adverse pulmonary effects of workplace irritants such as silica and metal dust.

Moreover, the observation that 28% of non-smokers developed COPD underscores the independent impact of occupational exposures. In 2023, Singh et al. conducted a crosssectional study of workers in construction and mining sectors and reported a 25–30% COPD prevalence among long-term non-smokers exposed to dust and fumes, consistent with the present study's findings21. Similarly, Zhang et al. (2024) identified a direct correlation between years of exposure to particulate matter in non-smoking factory workers and reduced

FEV_1 values, indicating that occupational factors alone can cause clinically significant lung disease22.
In terms of disease progression, recent studies echo the current report's conclusion that smoking accelerates COPD severity. For instance, Choi and colleagues (2023) found that smokers with occupational exposure were more likely to present with GOLD stage III–IV COPD, even at younger ages, compared to non-smoking counterparts with similar work histories23. These data reinforce the need for early intervention, particularly in industries with known respiratory hazards.

Preventive strategies proposed in the current study also reflect best practices supported by newer research. A 2025 randomized trial by Martins et al. found that combining workplace exposure control with smoking cessation counseling reduced new COPD diagnoses by nearly 40% over a 2-year period in metal workers24. These findings support the integration of engineering controls, personal protective equipment (PPE) such as respirators or masks, proper ventilation systems, regular air quality monitoring, adherence to occupational safety regulations, substitution of less harmful materials when possible, and implementation of engineering controls to minimize airborne contaminants. Worker education and training on respiratory protection and early symptom reporting also play a vital role in prevention. and cessation programs as a comprehensive approach to reducing occupational COPD risk.

In terms of screening, the role of periodic spirometry is well established. A 2024 consensus report by the Global Occupational Lung Disease Initiative (GOLDI) recommended that industries with high dust or fume exposure implement

bi-annual spirometry for all employees over 40, particularly those with a smoking history25. This aligns with the current study's emphasis on early detection as a key preventive strategy.

While the current study is limited by its small sample size and cross-sectional design, its strengths lie in its exposure-matched groups and use of spirometric criteria consistent with international guidelines. Future studies should prioritize longitudinal follow-up and quantitative exposure assessments to better model dose-response relationships and disease trajectories.

In conclusion, the current findings are highly consistent with recent research from 2023– 2025, confirming that both smoking and occupational exposure significantly contribute to COPD development and severity. The additive effect of smoking on occupational risk emphasizes the importance of integrated preventive strategies, including exposure control, smoking cessation, and routine health surveillance. These findings have direct implications for public health policy and occupational safety standards globally.

CONCLUSION

Even with a smaller sample, the findings confirm that smoking significantly increases both the prevalence and severity of COPD among workers with similar occupational exposures. Preventive strategies—like reducing exposures, promoting cessation, and early screening— are essential to mitigate disease progression and long-term disability.

Future Directions:

Future research should employ large-scale, longitudinal studies with quantitative exposure assessments to clarify dose-response relationships between occupational irritants and COPD. Investigating genetic susceptibility, biomarker profiling, and the long-term impact of integrated preventive strategies—including smoking cessation, exposure reduction, and routine spirometry will guide more personalized and effective occupational health interventions.

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