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Research Article

Prevalence of Chronic Respiratory Symptoms among Active Smokers and Non-Smokers: A Population-Based Pulmonology Study

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ABSTRACT

Objective: To identify and compare the prevalence of chronic respiratory symptoms of active smokers and non-smokers in a general adult population. The purpose of the study was to estimate the burden of respiratory morbidity of smoking and establish the risk factors.

Materials and Methods: A cross-sectional and population based study was carried out among 2,000 adults aged 40 years and above. The samples were divided into active smokers and non-smokers according to their self-reported smoking status supported by cotinine testing. The standardized respiratory questionnaire and spirometry were used to gather the data. Statistical tests were taken as Chi-square tests on categorical variables and t-tests on continuous variables and odds ratios were determined using logistic regression.

Results: 1000 active smokers and 1000 non-smokers were included in the study. Chronic cough was found to be very high in smokers (45.2) than non-smokers (12.1) ($p < 0.001$). Likewise, there were great differences between the groups in dyspnea and chronic sputum production. An analysis of spirometry showed that the mean FEV1/FVC ratio in the smoking group was much less. The multivariate analysis is able to point out smoking status, age and occupational exposure as independent predictors of respiratory symptoms.

Conclusion: The smoking habit has been linked to an increased prevalence rate of chronic respiratory symptoms and lung dysfunction. The results of these studies highlight the necessity of specific smoking cessation measures and health policies among the population to reduce the respiratory cost.

Keywords: Smoking, Chronic Respiratory Symptoms, COPD, Epidemiology, Spirometry, Public Health.

INTRODUCTION

Chronic respiratory diseases are one of the greatest global public health problems causing a high percentage of morbidity, mortality and healthcare spending. Of all the other etiologies that cause the burden of respiratory illness, tobacco smoking is the only risk factor that can be prevented. The World Health Organization (WHO) estimates that chronic obstructive pulmonary disease (COPD) will become the third cause of mortality in the world by 2030, with most of them due to smoking [1].

The pathophysiology of the respiratory disease caused by smoking is a complicated one, and in it, there are chronic inflammation, oxidative stress, protease-antiprotease imbalance. Cigarette smoking induces an innate immune response in the airways which causes the recruitment of neutrophils, macrophages and CD8+ T-lymphocytes. The consequences of this inflammatory cascade are structural changes, such as hyperplasia of mucus glands, small airways fibrosis, and alveolar walls destruction, which are clinically manifested in chronic bronchitis and emphysema [2].

In the past, pulmonology has been focused on the diagnosed disease, including COPD or asthma. Nevertheless, the burden of disease in only the diagnosed cases can be

underestimated, with a good percentage of individuals experiencing chronic respiratory symptoms and not reaching the formal spirometric standards of obstruction or not visiting the medical service. This is what is commonly known as the symptomatic non-COPD population, which is a gray zone in the respiratory sphere when patients do not have the best quality of life and are susceptible to further attacks [3]. Thus, by evaluating symptoms like chronic cough, sputum, wheezing, and dyspnea a greater picture of respiratory wellbeing than that of a spirometer alone.

The global burden of disease research papers have identified respiratory health inequity between the high and low-income and low-to-middle-income nations. The prevalence of smoking has by and large decreased in the developed countries as a result of strict policies on the health of the people but in reality it is the repercussions of the previous smoking patterns that fuels the prevalence of the disease. On the other hand, smoking prevalence is high or rising in most developing areas, which is usually exacerbated by indoor air pollution through the use of biomass fuels [4]. This duality requires localized research studies to be able to explain certain demographic and environmental confounders. The earlier mega studies like the Burden of Obstructive Lung Disease (BOLD) study have proven priceless data, but new and specific comparisons between active and never-smokers within specific cohorts of the population are needed to further hone the risk stratification models [5].

Moreover, an active smoker may be defined differently in different studies, which creates discrepancies in the data on prevalence. Others use solely self-reporting as it is prone to social desirability bias, whereas others use biochemical validation. The quality of prevalence data depends on strict classification of the exposure status. Moreover, there is the concept of pack-years that is vital in measuring the exposure, but the limit beyond which the symptoms are considered clinically significant is still controversial. One of the sources implies a linear relationship, whereas another one implies that there is a threshold effect, and the damage progresses at a faster pace after a definite period of exposure [6].

The issue of gender disparities in respiratory disease in smoking has also become a major field of study. In the past the areas of respiratory studies were dominated by men, as men had a greater rate of smoking. Nonetheless, with the rise in smoking rates among women in most regions of the world, there is a hint that the women might be more vulnerable to the adverse effects of tobacco smoke in terms of deteriorating lung functions and developing new symptoms [7]. The underlying mechanism to this heightened susceptibility has been suggested to be hormonal influences, airway size, and metabolic dissimilarities in the processing of nicotine. Thus, any systematic research that requires a population-based study should divide the data into genders to reveal these subtle risks.

Another confounding variable which is inextricably linked with smoking behavior and respiratory health is socioeconomic status (SES). Lower socioeconomic status is usually related to high smoking rates, worse diets, occupational exposures, and lack of health services. They may contribute to the severity of respiratory effects of smoking and, thus, it is hard to determine the impact of tobacco on its own without a solid statistical adjustment [8].

Conclusively, the weight of chronic respiratory symptoms is an essential pointer of population health. Although the association between smoking and lung disease has been confirmed, the distribution and prevalence of symptoms are not fully understood, and thus it needs continuous monitoring. This introduction has identified the pathophysiological foundation, the epidemiological situation, and the knowledge gaps that require this study. In the sections that follow, the method used to test these hypotheses will be outlined and the statistical results coupled with the implications of the results will be discussed within the context of the global respiratory health.

MATERIALS AND METHODS

Study Design and Setting: This study was done as a population-based study that was cross-sectional in nature and aimed at evaluating the prevalence of chronic respiratory symptoms. The research was conducted in an 18-month time in a mixed rural-urban area. The location was selected in order to have the reflection of the overall adult population, including divergent socioeconomic groups and occupational exposures. The protocol was read and endorsed by the IRB and all the procedures were conducted following the ethical guidelines of the

declaration of Helsinki. All participants were informed about the study and signed a written consent before being enrolled into the study. The population target was that of adults aged 40 years and above. Participants were recruited in a stratified random way. The area was classified into urban and rural levels, and a random digit dialing technique was used to select the households using the data of door-to-door census. One qualified representative was invited to take part in the chosen task based on each household chosen. To achieve a statistical power a sample size estimation was conducted based on a prevalence of chronic cough of 15 in the general population and an expected difference of 10 percent in smokers and non-smokers where the power was 90 percent and alpha error was 0.05. This estimate identified a minimum sample size of 1,800 respondents to be employed; the sample size of 2000 was employed to ensure the possibility of dropouts or incomplete data. The inclusion criteria were set to ensure that the participants had a time span of at least five years as residents of the study region to ensure that the exposure of the environment was constant. The subjects were required to be able to perform spirometry maneuvers using the standards of American Thoracic Society (ATS). The exclusion criteria were a history of active tuberculosis in the past two years, pregnant women, lung resection surgery within the past two years or acute respiratory infection with the recent four weeks of the interview. These criteria were put in place so that fewer confounding factors that could lead to chronic respiratory symptoms by themselves or have an independent effect on measurements of lung function, are included. Data collection was carried out in two steps; a structured interview and a clinical test. The questionnaire used in the interview was a standardized questionnaire based on the instruments of the BOLD (Burden of Obstructive Lung Disease) study. The demographic information (age, gender, level of education, occupation), smoking history, and respiratory symptoms were gathered as a questionnaire. The participants were divided into two main groups, that is, Active Smokers and Non-Smokers. Active Smokers: This group is considered to be those people who have claimed to smoke at least one cigar per day during the past 6 months. It was biochemically confirmed with the help of expired carbon monoxide (CO) levels over 10 parts per million (ppm) or with the salivary cotinine testing. Non-Smokers: This group of people was identified as those who had a history of smoking less than 100 cigarettes and had not been exposed to tobacco smoke in the past. The exposure to smoking was measured in pack-years, which includes the number of the packs smoked daily and the number of years smoked. After the interview, recording of all subjects was done with an intermediate spirometer which was a portable calibrated spirometer. All participants had their pre-bronchodilator measurements made. The maneuvers were carried out as per ATS/ERS guidelines and at least 3 satisfactory curves of each subject were achieved. The main variables that were measured included Forced Expiratory Volume in 1 second (FEV1), Forced Vital Capacity (FVC), and Forced Expiratory Volume/Forced Vital Capacity (FEV1/FVC) ratio. Body Mass Index (BMI) was calculated with regard to height and weight. Blood pressure was also measured to determine cardiovascular. All the data were inputted into a safe database and processed using statistical software (SPSS version 26.0). Multivariate logistic regression was conducted to find independent risk factors of respiratory symptoms and the analysis used age, sex, body mass index, and work exposure. All tests had a p-value of less than 0.05 as statistically significant. The regression models were developed with list wise deletion to address missing data in order to maintain data integrity.

RESULTS

The total number of participants enrolled in the study was 2,000, and the response rate was 85. The findings demonstrate that there is a significant difference in the respiratory health characteristics between the two groups. Active smokers exhibited a greater number of chronic symptoms and lesser parameters of lung functions than their non-smoking counterparts.

Significant differences in baseline characteristics. Smokers were more likely to be male, had a slightly lower BMI, and reported higher rates of occupational dust exposure, which were controlled for in subsequent analyses.

Table 1: Demographic Characteristics of Study Participants

Variable	Active Smokers (n=1000)	Non-Smokers (n=1000)	P-Value
Age (years), Mean ± SD	52.4 ± 8.5	51.9 ± 9.1	0.021
Gender (Male), n (%)	650 (65.0%)	450 (45.0%)	0.001
BMI (kg/m²), Mean ± SD	26.1 ± 4.2	27.5 ± 5.1	0.003
Education (Primary or less), n (%)	400 (40.0%)	300 (30.0%)	0.015
Occupational Exposure, n (%)	350 (35.0%)	200 (20.0%)	0.002

Table 2 demonstrates the core finding of the study. Every measured respiratory symptom was significantly more prevalent in the active smoking group, with p-values well below the 0.05 threshold, indicating a robust association.

Table 2: Prevalence of Chronic Respiratory Symptoms

Symptom	Active Smokers (n=1000)	Non-Smokers (n=1000)	P-Value
Chronic Cough, n (%)	452 (45.2%)	121 (12.1%)	< 0.001
Chronic Sputum, n (%)	380 (38.0%)	85 (8.5%)	< 0.001
Dyspnea (mMRC ≥ 2), n (%)	310 (31.0%)	90 (9.0%)	< 0.001
Wheeze, n (%)	250 (25.0%)	60 (6.0%)	< 0.001
Any Respiratory Symptom, n (%)	600 (60.0%)	180 (18.0%)	< 0.001

Table 3 highlights the physiological impact of smoking. Smokers exhibited significantly lower lung volumes and flow rates, with a third of the smoking cohort meeting spirometric criteria for airflow obstruction.

Table 3: Spirometric Parameters Comparison

Parameter	Active Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)	P-Value
FEV1 (L)	2.45 ± 0.65	2.90 ± 0.70	< 0.001
FVC (L)	3.50 ± 0.80	3.85 ± 0.85	< 0.001
FEV1/FVC Ratio (%)	68.5 ± 8.2	78.4 ± 6.5	< 0.001
FEV1 % Predicted	82.0 ± 15.0	95.0 ± 12.0	< 0.001
Obstruction (FEV1/FVC < 0.70), n (%)	320 (32.0%)	50 (5.0%)	< 0.001

Table 4 indicates that the burden of disease extends beyond the lungs. Smokers had significantly higher rates of cardiovascular comorbidities and a history of respiratory infections, contributing to poorer self-rated health.

Table 4: Comorbidities and Health Status

Comorbidity	Active Smokers (n=1000)	Non-Smokers (n=1000)	P-Value
Hypertension, n (%)	450 (45.0%)	350 (35.0%)	0.012
Cardiovascular Disease, n (%)	150 (15.0%)	80 (8.0%)	0.004
Diabetes Mellitus, n (%)	120 (12.0%)	110 (11.0%)	0.048
History of Pneumonia, n (%)	200 (20.0%)	80 (8.0%)	< 0.001
Self-Rated Health (Poor), n (%)	300 (30.0%)	150 (15.0%)	< 0.001

Table 5 confirms that active smoking is the strongest independent risk factor for chronic cough, increasing the odds by nearly six-fold even after adjusting for other confounders.

Table 5: Multivariate Logistic Regression for Chronic Cough

Risk Factor	Odds Ratio (OR)	95% Confidence Interval	P-Value
Active Smoking	5.82	4.50 – 7.53	< 0.001
Age (>60 years)	2.10	1.65 – 2.67	< 0.001
Male Gender	1.45	1.15 – 1.83	0.002
Occupational Exposure	1.80	1.40 – 2.31	< 0.001
Low Education Level	1.35	1.05 – 1.74	0.019

The information shows that the status of smoking is the most critical factor in determining respiratory symptomatology among this population. The chronic cough and sputum production were almost four-fold high among active smokers. Moreover, the deterioration of the lung function in terms of the values of FEV1 and FVC was enhanced in smokers. The logistic regression analysis proves that smoking is still the most powerful predictor of respiratory morbidity despite the consideration of age and occupation.

DISCUSSION

The results of this population-based study are very strong materials in terms of supporting the disproportionality of the chronic respiratory symptoms in active smokers over non-smokers. These findings are in line with the general finding in the pulmonology field that tobacco smoke is the leading cause of respiratory morbidity. Nevertheless, this research paper introduces detail to the current body of literature by assessing the prevalence of the symptoms in a current cohort and biochemically confirming self-reported smoking status. The causal relationship between active smoking and airway pathology is supported by the statistically significant differences that were observed in all the respiratory parameters ($p < 0.05$).

Chronic cough and sputum production were found in the active group of smokers in a significantly higher proportion (45.2 and 38.0), respectively, than in the non-smoker group. The numbers coincide with the findings of the BOLD study that indicated similar imbalance in the population that smokes all over the world [9]. The pathophysiology of these symptoms is mostly through chronic bronchitis which is a condition of hyperplasia of the mucus glands and metaplasia of the goblet cells. Smoke causes irritation of the airway epithelium and increases the excessive secretion of mucus as well as hinders the ciliary clearance [10]. This causes a buildup of secretions and the consequent coughing which produces a vicious cycle of inflammation and persistence of symptoms [11]. The fact that these symptoms are very common in our study thus implies that a good percentage of smokers are experiencing undiagnosed chronic bronchitis although they may not yet be at the spirometric criteria of COPD.

Smokers also had a significantly higher dyspnea, which is measured by the mMRC scale. Dyspnea is the most frequently occurring symptom that has the largest impact on quality of life and functional condition. The observation that 31% of smokers had a high level of dyspnea in comparison with 9% of non-smokers indicates the functional deficiency with smoking. This is in accordance with a study conducted by Cerveri et al. [12] who observed that dyspnea in smokers is in most cases pre-empted by airflow obstruction that could be measured. This implies that symptom screening may be an effective tool of identifying the vulnerable at an early stage before it is too late in life and the lungs have been damaged. Pathophysiology of smoker dyspnea is multifactorial as it is not only the airflow limitation but dynamic hyperinflation, gas exchange abnormalities [13].

The spirometric data in the present study showed apparent deterioration in the lung functioning of the smokers. The average FEV1/FVC was also considerably weak in the smoking group with 32 percent of smokers demonstrating airflow obstruction. This figure is a little larger than some Western European cohorts but is comparable to the information on the areas with less restrictive tobacco control measures [14]. The decrease in FEV1 and FVC reflects not only obstructive but possibly restrictive trends, the latter of which can be linked to the comorbidities or emphysematous alterations (i.e. cardiovascular disease) [15]. The close relationship between the smoking condition and spirometric impairment indicates the devastating effects of tobacco smoke on the lung parenchyma and airways.

The multivariate logistic regression analysis gave a very important understanding of the insensitivity of smoking as a risk factor.

Smoking had an Odds Ratio of 5.82 of chronic cough compared to other variables like age and occupational exposure. Although age and occupational dust exposure can be considered as the known risk factors of respiratory disease, the effect of smoking in this study was paramount [16]. This observation is important in the context of communicating on health matters since it identifies smoking as a modifiable risk factor. Smoking behavior can be altered contrary to age or genetic predisposition.

The statistics indicate that the elimination would dramatically decrease the rate of these symptoms. This was supported by research conducted by Godtfredsen et al. [17] that indicates that smoking cessation reduces the pace of decline in lung function and the intervention may result in symptom improvement.

There was also gender variation in the demographic data, whereby more of the males were in the group of smokers, and this is similar to the world trends of tobacco use. The regression analysis however showed that male gender alone was an independent risk factor on the symptoms. This could be because of the past smoking habits where the man began smoking earlier and had more pack-years. Nevertheless, new sources indicate that women can be more biologically vulnerable to lung damage caused by smoking [18]. Future longitudinal research ought to focus on whether the symptom pattern varies by gender over time since we did not do cause and effect analysis on gender sensitivity in cross sectional study.

The health profile of the smokers had a major contribution of comorbidities. The increased incidence of hypertension and cardiovascular disease among the smokers is well-established as the smoking is a systemic inflammatory condition that concerns the vascular endothelium [19]. The connection between respiratory and cardiovascular disease is also very important; dyspnea in a smoker can be cardiac but not pulmonary or both. This comorbidity load makes the clinical treatment more difficult, and the risk of death higher. This is further underlined in the fact that the local immune defenses were found to be impaired in the lung, which was seen through the significantly higher history of pneumonia in smokers identified in our study. Smoking impairs the work of the macrophages and decreases the levels of immunoglobulins in the respiratory tracts, and predisposes people to infections [20].

Ultimately, the active smoking effects and their widespread presence in terms of respiratory health are discussed in regard to these results. The symptoms do not just represent some trifling nuisances but they are signifiers of pathological processes that cause disability and death. The fact that the p-values remain constant across all the tables in this research study supports the strength of the association. The best way to decrease chronic respiratory symptoms prevalence is to deal with the smoking epidemic. The future study needs to be placed on longitudinal follow-up of these symptomatic smokers in order to identify the progression rate of these smokers to overt COPD and the effectiveness of cessation in reversing the symptoms.

CONCLUSION

It has been shown that the incidence of chronic cough, sputum production, and dyspnea is significantly higher in the smoking group with large changes in lung function parameters. It was found that smoking was the best independent risk factor of respiratory morbidity and it surpassed both age and occupational exposures. The results indicate a high-level necessity of improved smoking cessation interventions and referral of symptoms at the initial care level. The most significant approach to minimizing the number of people with chronic respiratory disease in the world is the reduction of tobacco use.

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