

ASSOCIATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) WITH SMOKING IN PATIENTS ATTENDING THE MEDICAL OUT-PATIENT DEPARTMENT OF A TERTIARY CARE HOSPITAL IN A DEVELOPING COUNTRY

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ABSTRACT

Objective: To study the association of chronic obstructive pulmonary disease with smoking in patients attending the Medical out-patient department of Darul Sehat Hospital, Karachi. **Study Design:** Analytical Cross-Sectional study. **Place and Duration of study:** A 2-month duration study was conducted in the demographical area of Karachi, Sindh, Pakistan. The duration of the study is from September 1, 2019 to November 1, 2019. **Methodology:** The data was collected through a semi-structured questionnaire consisting of open and closed-ended questions. The questionnaire was filled by the participants with a 100% response rate. Final data were analyzed with the help of SPSS version 20 and the descriptive statistics e.g. percentage and frequencies were calculated. **Results:** A total of 60 patients (males 50, females 10) were enrolled in our study. 58(96.67%) patients were older than 40 years. Current or past smoking history was present in 53(83.3%). The majority of the patients had cough 53(83.3%) with phlegm production in 44(73.3%) patients. All of the patients had undergone CXR with 47(78.33%) patients had signs of significant lung damage (all belonging in the smoker category). **Conclusion:** Our study concluded that there is a significant relationship between smoking and chronic obstructive lung disease. Cigarette smoke induces a strong inflammatory response causing the destruction of lung parenchyma and airways leading to a massive drop in oxygen saturation levels. Non-smokers have significantly lower chances of developing lung disease as compared to smokers.

KEYWORDS: COPD, smoke, Oxygen saturation, lung damage.

INTRODUCTION

COPD is characterized by an abnormal response of the lungs to toxic particles and gases, resulting in progressive and often irreversible airway obstruction. The main pathologic hallmarks are bronchiolitis and lung parenchymal emphysematous destruction.^[1] Despite tobacco smoke (TS) being the environmental risk factor most frequently associated with COPD, exposure to biomass smoke (BS) has also been proposed as one of the main risk factors for developing the disease, especially among non-smokers.^[2,3] In this respect, the existing evidence of clinical, functional, radiologic, and histopathology differences between TS and BS-exposed COPD patients has raised the hypothesis of two distinct phenotypes of COPD, according to the main triggering

risk factor. Thus, in addition to the known gender difference on exposure chance, BS COPD patients exhibit a milder airflow obstruction—measured by FEV1 and FEV1/FVC—than TS COPD subjects, as well as a higher DLCO value.^[4-7]

On the other hand, BS COPD patients show more anthracosis and pulmonary fibrosis and less emphysema than TS COPD patients.^[8-10] The evidence of different pathologic features in COPD patients exposed to BS or TS underpin the existence of a third potential phenotype which remains largely uncharacterized: COPD patients with significant exposure to both TS and BS (TS + BS COPD).

Given the toxicity of both kinds of smoke, higher severity of pulmonary damage could be expected in double-exposed subjects. Following this line of thought, Dogan *et al.*^[11] compared lung histopathological alterations among rats exposed to BS and a group with combined exposure to TS and BS. They found an increased severity of perivascular inflammation, as well as higher parenchymal infiltration, number of alveolar macrophages, and vascular wall thickness in the lungs of rats exposed to TS and BS than in those only exposed to BS. With data from the PREPOCOL study,^[12] Torres-Duque *et al.*^[13] compared some demographical and functional parameters among 114 COPD patients exposed to wood smoke, 81 COPD patients exposed to TS, and 264 COPD patients exposed to both types of smoke; reporting significant decreases in FEV1 and FEV1/FVC values in patients with a combined exposure^[13] In both studies, the authors suggested that combined exposure to TS and BS could have a noxious additive effect in COPD patients and could increase the severity of COPD symptoms.

OBJECTIVES

1. Assess and monitor diseases

Diagnosis of COPD is based on a history of exposure to risk factors and the presence of airflow limitation that is not fully reversible, with or without the presence of symptoms. Patients who have chronic cough and sputum production with a history of exposure to the risk factors should be tested for airflow limitation even if they do not have dyspnea. For the assessment of COPD, spirometry is the gold standard as it is the most reproducible, standardized, and objective way of measuring airflow limitation. Health care workers involved in the diagnosis and management of patients with COPD should have access to spirometry.

2. Reduce Risk Factors

Reduction of the total personal exposure to tobacco smoke, occupational dust, chemicals, and indoor/outdoor air pollutants are important goals to prevent the onset and progression of COPD. Smoking cessation is the most effective way to reduce the risk of developing COPD and stopping its progression. Tobacco users should be offered this treatment at every visit by the health care providers. Highly effective counseling types include practical counseling and social support arranged outside of treatment.

3. Manage Stable COPD

The overall approach to managing stable COPD should be characterized by the stepwise increase in treatment, depending on the severity of the diseases. For patients with COPD, health education can play a role in improving skills and the ability to cope with illness. None of the existing medications for COPD has been shown to modify the long-term decline in lung function that is the hallmark of the disease. Bronchodilator medications are central to the symptomatic management of COPD. They are given on an as-needed basis or

regularly to prevent or reduced symptoms. Chronic treatment is with glucocorticoids. All patients with COPD get benefit from exercise training programs.

4. Manage Exacerbations

Exacerbation of the respiratory symptoms and requiring medical intervention are important clinical events in COPD. The most common causes of COPD exacerbation are infection of the tracheobronchial tree and air pollution, but the causes of approximately one-third of severe exacerbations are unidentified. Short-acting inhaled bronchodilators are effective.

Research Methodology

Study design: Cross-Sectional Study Design

Study population: 60 patients suffering from COPD presenting in the out-patient department of Darul Sehat Teaching Hospital, Karachi.

Place and duration of study: A 2-month duration study was conducted in the demographical area of Karachi, Sindh, Pakistan. The duration of the study is from September 1, 2019 to November 1, 2019.

Methodology: The data was collected through a semi-structured questionnaire consisting of open and closed-ended questions. The questionnaire was filled by the participants with a 100% response rate. Final data were analyzed with the help of SPSS version 20 and the descriptive statistics e.g. percentage and frequencies were calculated.

RESULTS

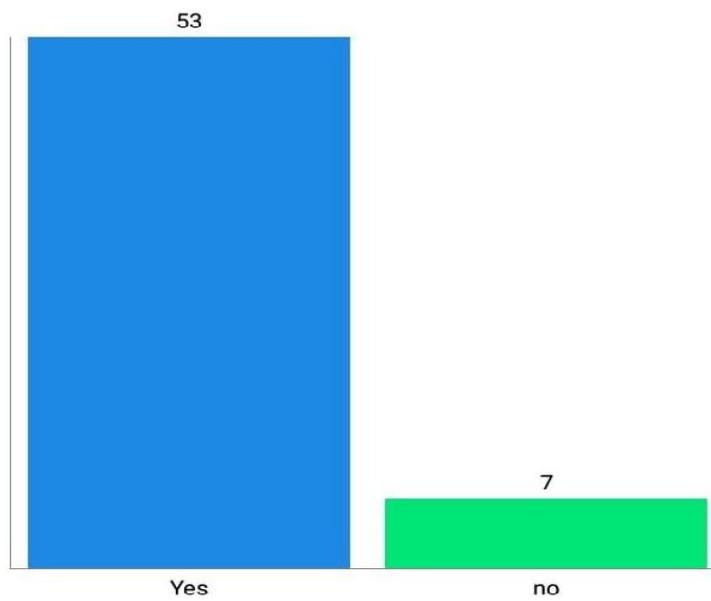
A total of 60 patients (males 50, females 10) were enrolled in our study. 58(96.67%) patients were older than 40 years. Current or past smoking history was present in 53(83.3%). The majority of the patients had cough 53(83.3%) with phlegm production in 44(73.3%) patients. All of the patients had undergone CXR with 47(78.33%) patients had signs of significant lung damage [45/53 (84.9%) in smokers and 2/7 (28.6%) in non-smokers.

1. Do you cough several times a most days?

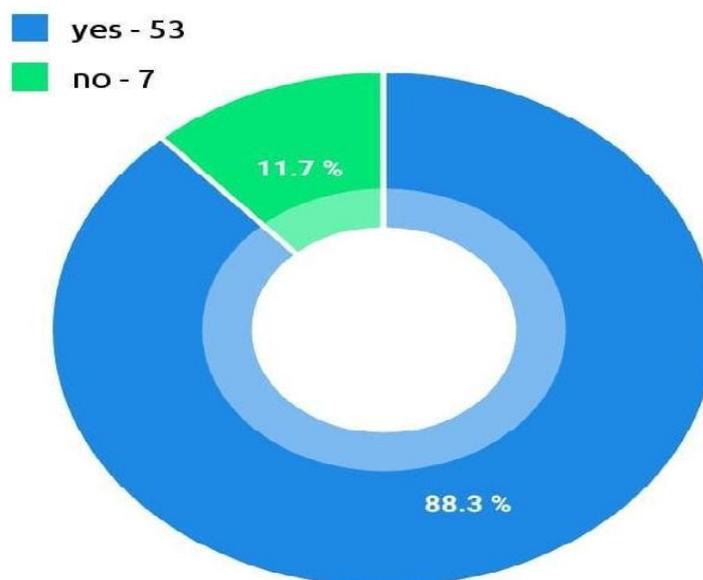
RESULTS

Choices	%	Count
Yes	88.33 %	53
no	11.67 %	7

BAR CHART



PIE CHART

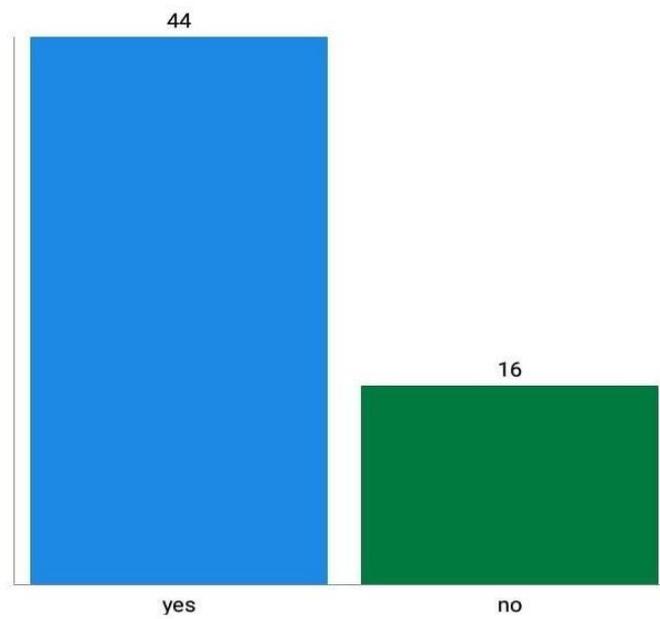


2. Do you bring up phlegm or mucus most days?

RESULTS

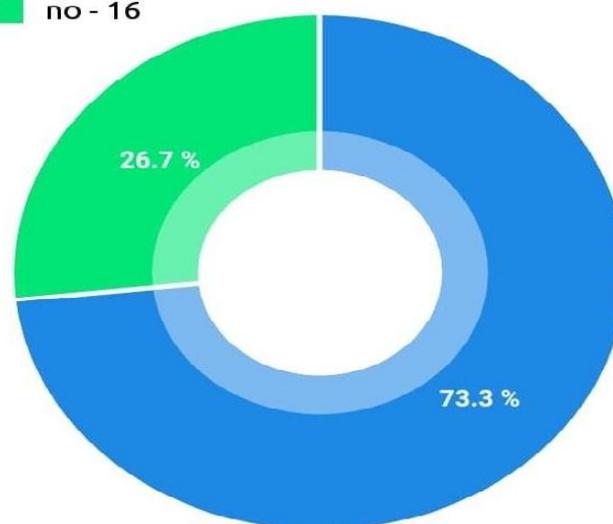
Choices	%	Count
yes	73.33 %	44
no	26.67 %	16

BAR CHART



PIE CHART

■ yes - 44
■ no - 16

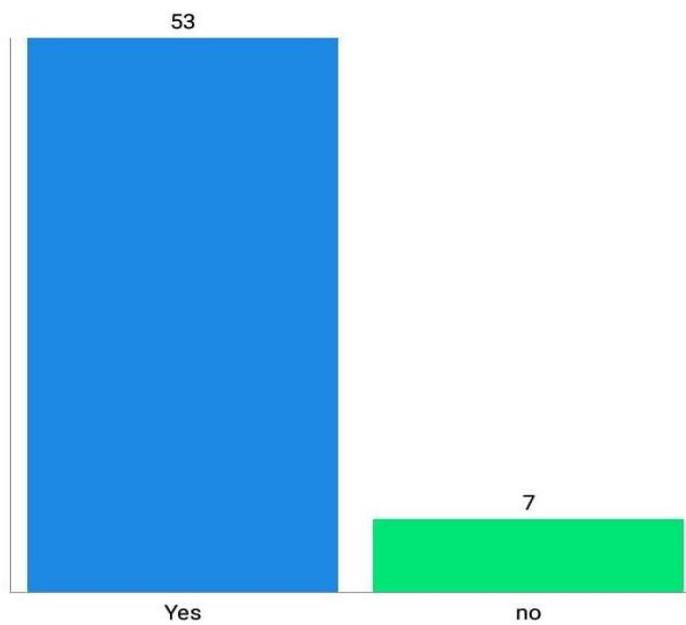


3.Are you a current smoker or a ex-smoker

RESULTS

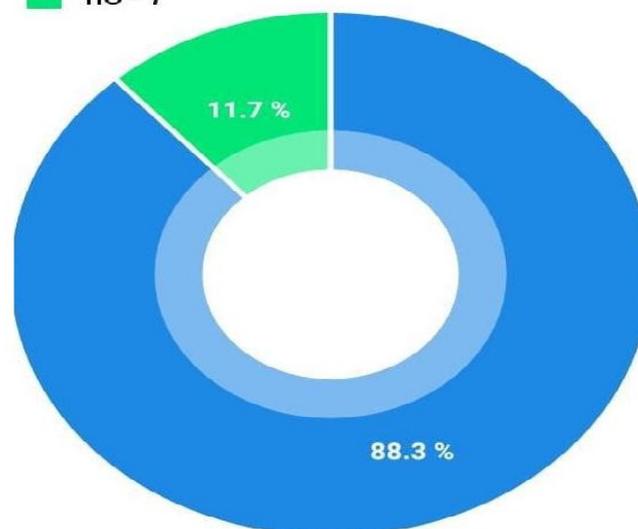
Choices	%	Count
Yes	88.33 %	53
no	11.67 %	7

BAR CHART



PIE CHART

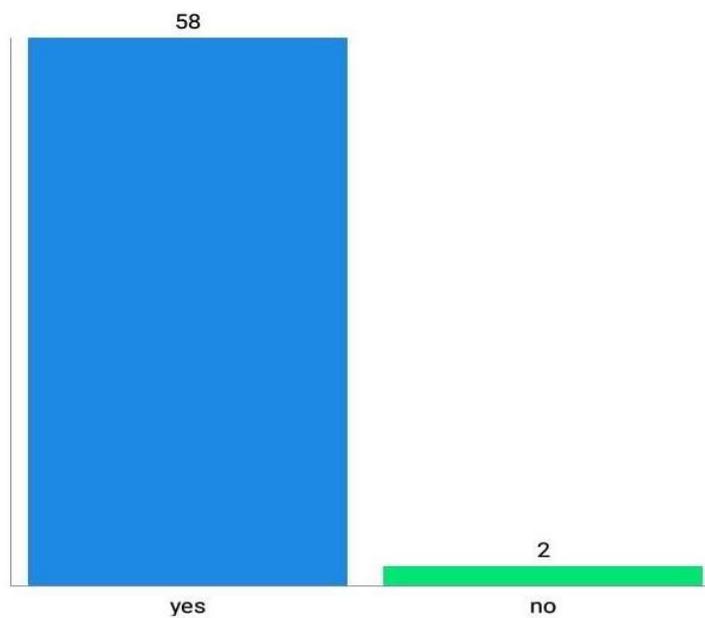
■ yes - 53
■ no - 7



4.Are you older than 40 years?

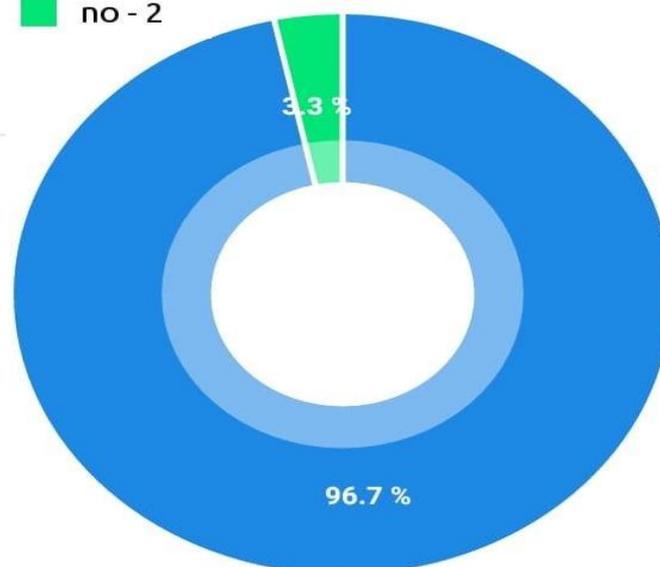
RESULTS

Choices	%	Count
yes	96.67 %	58
no	3.33 %	2

BAR CHART**PIE CHART**

■ yes - 58

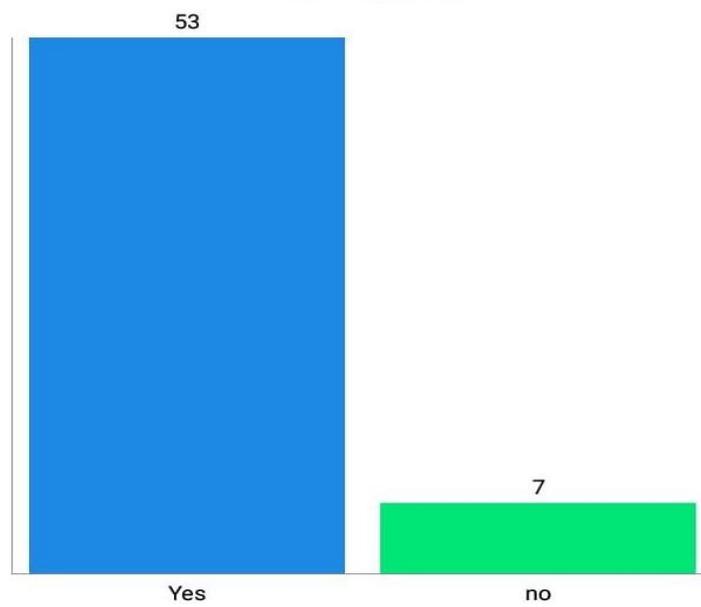
■ no - 2



5Did you cough RESULTS

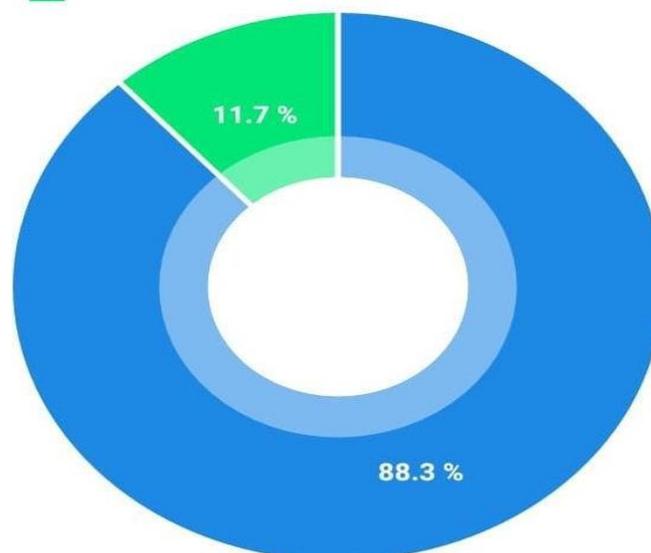
Choices	%	Count
Yes	88.33 %	53
no	11.67 %	7

BAR CHART



PIE CHART

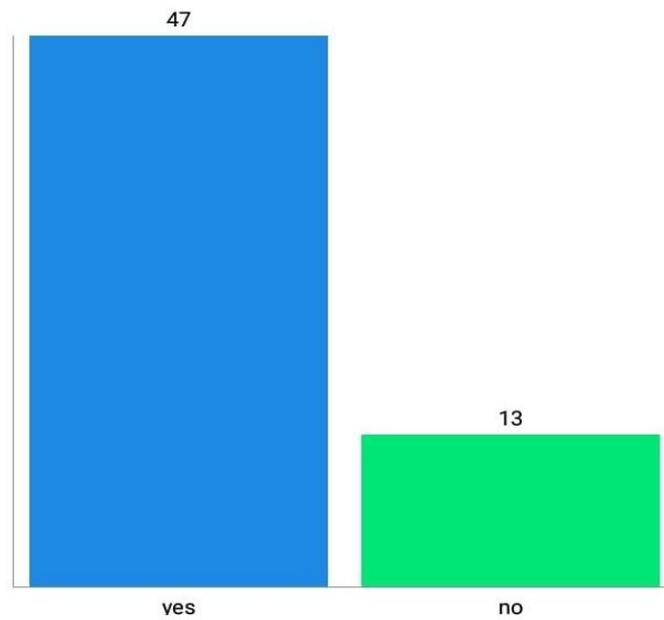
■ yes - 53
■ no - 7



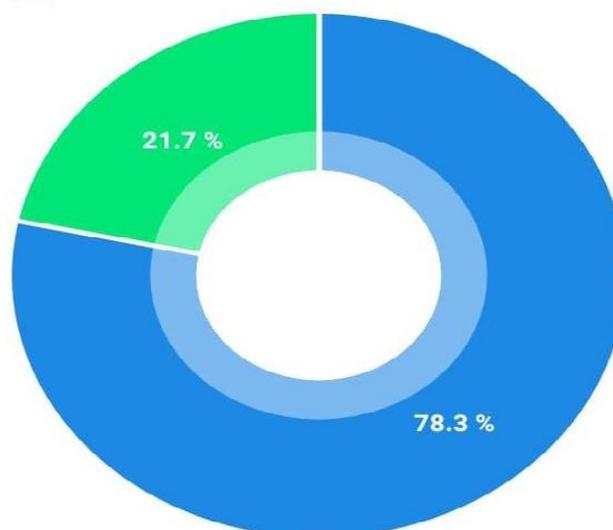
6. Abnormal chest X-Ray

RESULTS

Choices	%	Count
yes	78.33 %	47
no	21.67 %	13

BAR CHART**PIE CHART**

- yes - 47
- no - 13



DISCUSSION

Although COPD is primarily a disease of lungs and airways, it also has several extra-pulmonary manifestations and comorbidities. Indeed, systemic inflammation is a hallmark of COPD and it may be involved in cardiovascular complications in COPD patients.^[14] In general, lung function was more severely impaired in COPD patients exposed to TS, who also showed higher levels of fibrinogen, circulating leukocytes, and monocytes, whereas COPD patients exposed to BS had significantly higher levels of blood IgE antibodies. Increased number of inflammatory cells have been reported not only in the small airways and lung parenchyma of COPD patients.^[15-17] but also in the peripheral blood.^[18-19] Moreover, several inflammatory markers are also increased in the blood of COPD patients; one of the most widely studied is C-reactive protein, an acute-phase reactant secreted by the liver during inflammatory processes, which is increased in both stable and exacerbated COPD patients.^[20] The results of this study show that there are significant clinical, functional, and systemic differences between COPD patients exposed to TS, BS, or both. These results suggest different inflammatory responses to different inhaled pollutants in COPD. There is a synergistic damaging effect of the combined exposure to Tobacco smoke and Biomass smoke. The systemic inflammatory response in these two types of COPD is, however, distinct. Given that Biomass Smoke and Tobacco Smoke have many components in common, further characterization at a molecular level would be necessary to elucidate what concentrations could be responsible for different inflammatory features leading to the development of COPD.

We suggest adopting a healthy lifestyle to cope with COPD which will lead to improved functionality and exercise tolerance of the patients. Eating a healthy diet and exercising are important to improve your fitness level. Always talk with your provider before starting a diet or exercise plan and start slowly. Smoking cessation and long term home oxygen therapy have been linked with an improved prognosis of the disease. Learning techniques to bring up mucus and clearing airways have also been linked to fewer hospital visits as depicted by our study.

CONCLUSIONS

Our study concluded that there is a significant relationship between smoking and chronic obstructive lung disease. Cigarette smoke induces a strong inflammatory response causing the destruction of lung parenchyma and airways leading to a massive drop in oxygen saturation levels. Non-smokers have significantly lower chances of developing lung disease as compared to smokers. Biomass smoke also leads to lung inflammation but the rate of development of chronic lung diseases is significantly lower in this group. No-Tobacco policies should be implied at governmental levels and support

should be offered to smokers to encourage them to quit smoking.

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