RESEARCH PAPER

# A comparative study of arterial blood gas and lung function parameters between exposure to biomass fuel smoke and tobacco smoke in a rural population of north India

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## ABSTRACT

**Background:** Nearly half of the global population relies on biomass fuel for cooking, heating, and lighting due to its low cost and easy availability, making it a major source of household air pollution. This study aimed to compare lung function and arterial blood gas (ABG) parameters among individuals exposed to biomass fuel smoke and tobacco smoke.

**Methods:** A total of 165 individuals were included, comprising 83 biomass fuel users (BS) and 82 tobacco smokers (TS). Sociodemographic variables, vital parameters, pulmonary function tests, and ABG measurements were assessed and compared between two groups.

**Results:** Lung function indices were significantly lower in biomass fuel users compared to tobacco smokers ($p < 0.001$). Mean arterial oxygen tension ($pO_2$) was marginally higher in biomass users than smokers, though this difference was not statistically significant. Biomass users demonstrated significantly lower $pCO_2$ and lower pH values compared to smokers ($p < 0.001$).

**Conclusion:** Biomass fuel exposure is predominantly associated with impaired oxygenation and hypoxemia, while tobacco smoke exposure is more closely linked to hypercapnia resulting from ventilatory impairment. These distinct physiological patterns underscore the need for targeted preventive and therapeutic strategies, particularly in rural populations exposed to biomass smoke.

**Keywords:** Biomass fuel smoke, tobacco smoke index, lung function test, environmental pollution

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## INTRODUCTION

The incomplete and the inefficient combustion of biomass fuels1 has been a known risk factor for development of respiratory disorders, such as Chronic Obstructive Pulmonary Disease (COPD), chronic bronchitis, asthma, lung cancer, lung fibrosis, and tuberculosis etc., COPD due to biomass smoke is especially increasing in women of the Middle East Asian, and African2-4 countries, indirectly pointing towards the low- and middle-income countries. Estimated deaths due to indoor air pollution from biomass exposure amounts to 2 million women and children annually5. Biomass fuel smoke was composed of organic particulate matter6 of less than 10µm (Particulate Matter- 10) in aerodynamic diameter, carbon monoxide, nitrogen dioxide, formaldehyde, sulphur dioxide7, and polycyclic organic matter including carcinogens like benzopyrene8-10. And the particles with diameter lesser than 2.5µm (PM2.5) will easily penetrate into the lungs causing respiratory dysfunction. Arterial blood gas test used to measure arterial partial pressure11 of oxygen (pO2), the arterial partial pressure of carbon dioxide (pCO2), pH and the arterial oxygen saturation12 and some electrolytes values can also be determined. It is one of the most common

tests performed in intensive-care units.

To quantify the impact of the biomass fuel on the blood gas parameters, such as pO2, pCO2, and pH, we devised biomass exposure index13 by multiplying the average hours spent by the person on cooking and the number of years, the person cooking with biomass fuel. A very few studies only been conducted on blood gas parameter analysis of biomass fuel users, and in rural areas the awareness of harmful impact of biomass fuel also been less recognised and addressed in comparison to urban areas, so in order to fill that lacunae with some useful information this study been devised. Therefore, this research aims to provide a comprehensive analysis of arterial blood gas and lung function parameters in a rural North Indian population, comparing the physiological effects of chronic biomass fuel smoke exposure against those observed with

tobacco smoke, thereby illuminating the distinct and overlapping respiratory morbidities associated with these prevalent environmental insults.

**Methodology:**

Study population:

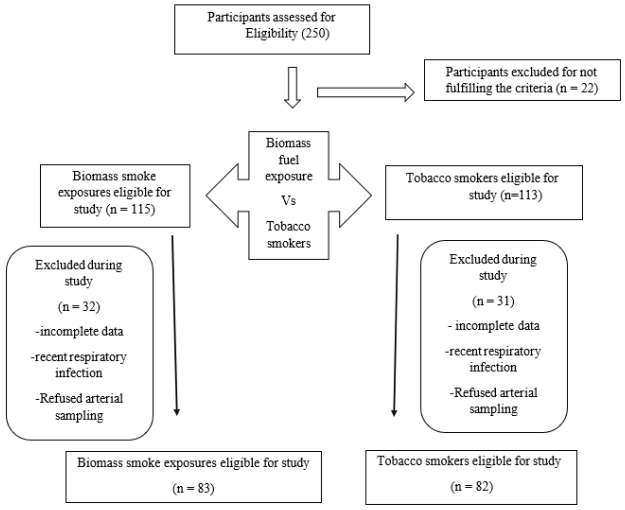
The ethics committee of institute approved the study (BPSGMC/WRC390/IEC/2018) and written informed consent was obtained from all the subjects. The study adhered to the principles of the declaration of Helsinki. We studied 250 individuals who had either biomass smoke exposure or tobacco smokers at suburban hospital of Haryana. All the individuals were recruited at the respiratory department of the Hospital, who had come for respiratory disease monitoring. Subjects in the age group of 18 – 45 years were included. After collecting baseline characteristics, subjects were classified into 2 groups: 83 individuals who had biomass fuel smoke exposure (BS without tobacco smoke exposure) and 82 individuals who were tobacco smokers (TS without biomass fuel smoke)(Figure 1). Biomass exposure index was calculated by the average hours spent on cooking per day X the number of years spent in cooking (13). Smoking index was calculated by the average number of cigarettes/bidis smoked per day X the number of years a person had smoked(13).

Participants with known COPD due to exposure of biomass or tobacco smoke, Patients with known COPD due to exposure other than biomass or tobacco, Subjects having anatomical defects in respiratory system, which created hindrance in performing pulmonary function tests, previous history of surgery, recent Myocardial infarction were not included for the study. The study excluded the participants who were exposed to passive tobacco smoking.

Sample size calculation: The required sample size was estimated for detecting differences in lung function parameters between biomass smoke–exposed and tobacco smoke–exposed individuals. Assuming a two-sided $\alpha = 0.05$ and 80% power, a difference of 0.30 L in $FEV_1$

with a standard deviation of 0.60 L would require approximately 63 subjects in each group. Similarly, to detect a 10% difference in predicted $FEV_1$ values with a standard deviation of 15% would require 36 subjects per group. Our study included 83 individuals in the biomass group and 82 in the tobacco group, thereby exceeding the minimum requirement and ensuring adequate statistical power to detect clinically meaningful differences.

**Figure 1**: STROBE flowchart for recruitment of participants




All parameters including age, sex, height, weight, body mass index (BMI), type of exposure (biomass fuel smoke or tobacco smoke), blood pressure (BP), pulse rate (PR), temperature, pulmonary function test (PFT) indices and Arterial blood gas (ABG) were obtained and recorded in the proforma. Standard procedures were used to assess pulmonary function in all individuals, including measurements of forced expiratory volume in 1 s (FEV1), Forced vital capacity (FVC) and FEV1/FVC ratio and ABG.

**Pulmonary function Test:**

All study subjects were explained the procedure in detail. Reports were obtained after taking all the precautions. Pulmonary functions were measured by a forced vital capacity (FVC) maneuver on a computed spirometer with automated quality checks- BTL-08 Spiro PC, manufactured by Health and Medical Industry, United Kingdom.

**Arterial blood gas analysis (ABG):**

Arterial blood sample (0.5 ml) were collected after performing all the precautionary measures, from the radial artery by using a heparinised syringe after conducting the modified Allen's test to prevent ischemic complications to find the gas indices (pO2, pCO2, and pH) and subjects who were not willing and having peripheral vascular disorders and end stage diseased subjects were excluded. ABG parameters were analysed

by auto analyser- pH ox Ultra by Nova Biomedical Corporation, USA.
Statistical analysis:

Data are expressed as Mean + SD. The p-value obtained < 0.05 was considered as
statistically significant and with value < 0.001 considered highly statistically significant. Statistical analysis was done by using Microsoft Office Excel and standard software (IBM Statistical Package for the Social Sciences (SPSS), Statistics for Windows, Version 20.0. Armonk, NY: USA).

**Results:**
The mean ± SD of Sociodemographic and vitals among biomass fuel smoke exposed individuals Vs tobacco smokers had no statistical significant difference (Table - I). The unpaired t-test revealed significant ($p<0.001$) differences in predicted FEV1, FVC, FEV1/FVC, MEF75, MEF50 , MEF25 between biomass fuel exposure group and tobacco smokers (Table -II). Mean $pO_2$ was slightly higher in biomass users (103.1 ± 11.2 mmHg) than in tobacco smokers (100.0 ± 11.5 mmHg), but the difference was not significant ($p = 0.118$). Mean $pCO_2$ was significantly lower in biomass users (40.3 ± 3.3 mmHg) compared to tobacco smokers (45.3 ± 5.9 mmHg, $p < 0.001$). Mean pH was significantly lower in biomass users (7.38 ± 0.04) than in tobacco smokers (7.41 ± 0.05, $p < 0.001$) (Table III).

BMEI (biomass exposure index) was significantly negatively correlated with $pO_2$ ($r = –0.382$, $p = 0.002$), indicating higher biomass exposure is associated with lower oxygen levels. No significant correlations were observed with $pCO_2$ or pH. Smoking Index was strongly positively correlated with $pCO_2$ ($r = 0.840$, $p = 0.001$), indicating higher tobacco exposure is associated with $CO_2$ retention. No significant correlations were observed with $pO_2$ or pH. Biomass exposure primarily affects oxygenation, while tobacco exposure primarily affects $CO_2$ retention, with minimal effect on pH in both cases (Table IV).

Pearson's correlation revealed the (BS) group (n = 83), lung function parameters showed a significant positive correlation with arterial oxygen tension. Predicted FEV1 correlated positively with $pO_2$ ($r = 0.313$, $p = 0.011$), as did predicted FVC ($r = 0.323$, $p = 0.004$) and FEV1/FVC ratio ($r = 0.319$, $p = 0.010$). No significant associations were observed with $pCO_2$ or pH. These findings indicate that reduced lung function in biomass-exposed individuals is primarily associated with impaired oxygenation.

In contrast, in the tobacco smoke (TS) group (n = 82), lung function parameters demonstrated significant negative correlations with arterial carbon dioxide levels. Predicted FEV1 ($r = – 0.384$, $p = 0.002$), predicted FVC ($r = –0.327$, $p = 0.008$), and FEV1/FVC ratio ($r = –0.521$, $p < 0.001$) all correlated inversely with $pCO_2$, while no significant relationship was observed with $pO_2$ or pH. This pattern suggests that tobacco-related airway obstruction is strongly associated with carbon dioxide retention rather than hypoxemia (Table V).

Taken together, these results highlight a differential pathophysiological effect of biomass and tobacco smoke exposure on gas exchange: biomass smoke is more strongly associated with hypoxemia, whereas tobacco smoke is predominantly linked to hypercapnia due to impaired ventilation.

**Table 1:**
**Comparison of Sociodemographic and vitals:**

| Parameters | BS (n = 83) | TS (n = 82) | *p* value |
|---|---|---|---|
| Age (Years) | 35.0781 ± 7.3209 | 33.2031 ± 7.9525 | 0.168 |
| Height (cms) | 152.5468 ± 6.2384 | 154.5625 ± 6.6377 | 0.079 |
| Weight (Kgs) | 54.8281 ± 12.2507 | 55.1875 ± 0.5075 | 0.859 |
| BMI | 23.5859 ± 5.0108 | 23.1 ± 4.221 | 0.554 |
| Biomass exposure, hours-years | 310.8±110.8 | -- | -- |
| Smoking history, pack-years | -- | 41.57±25.62 | -- |
| SBP (mmHg) | 124 ± 16 | 128 ± 14 | 0.621 |
| DBP (mmHg) | 74 ± 12 | 78 ± 10 | 0.23 |
| MAP (mmHg) | 88 ± 2.6 | 90 ± 1.5 | 0.12 |
| HR (bpm) | 95 ± 7.8 | 89 ± 8.2 | 0.12 |

SBP – Systolic blood pressure; DBP – Diastolic blood pressure; MAP – Mean Arterial Pressure; HR – Heart Rate;
Table 2:
Comparison of predicted FEV1, predicted FVC, predicted FEV1/FVC, predicted MEF75, predicted MEF50 and predicted MEF25 among biomass fuel users and tobacco smokers

| S.No | Parameter | Biomass fuel smokers (BS) (Mean ± SD) | Tobacco smokers (TS) (Mean ± SD) | p-value |
|---|---|---|---|---|
| 1 | Predicted $FEV_1$ (l) | 2.3553 ± 0.3471 | 3.1777 ± 0.39001 | 0.001[b] |
| 2 | Predicted FVC (l) | 2.6927 ± 0.3598 | 3.7905 ± 0.4584 | 0.001[b] |
| 3 | Predicted $FEV_1$/ FVC (%) | 82.2045 ± 1.4967 | 80.3484 ± 1.5070 | 0.001[b] |
| 4 | Predicted MEF75 (l/s) | 5.6352 ± 0.3694 | 7.4577 ± 0.5646 | 0.001[b] |
| 5 | Predicted MEF50 (l/s) | 4.0386 ± 0.2816 | 4.7508 ± 0.4050 | 0.001[b] |
| 6 | Predicted MEF25 (l/s) | 1.7941 ± 0.2762 | 2.0058 ± 0.2947 | 0.001[b] |

([b]p<0.005 Highly Significant)

Table 3: Comparison of pO2, pCO2, pH among biomass users and tobacco smokers:

| S.No. | Parameter | Biomass users (Mean ± SD) | Tobacco smokers' (Mean ± SD) | p-value |
|---|---|---|---|---|
| 1 | $pO_2$ | 103.1188 ± 11.1790 | 99.9672 ± 11.5007 | 0.118 |
| 2 | $pCO_2$ | 40.2797 ± 3.3091 | 45.2719 ± 5.8949 | 0.001[b] |
| 3 | pH | 7.3834 ± 0.0372 | 7.4138 ± 0.0478 | 0.001[b] |

([b]p<0.005 – Significant

Table 4:
Correlation of biomass exposure index and smoking index with the arterial blood gas parameters.


| S.No. | Parameter | Biomass users (Mean ± SD) | Tobacco smokers' (Mean ± SD) | p-value |
|---|---|---|---|---|
| 1 | $pO_2$ | 103.1188 ± 11.1790 | 99.9672 ± 11.5007 | 0.118 |
| 2 | $pCO_2$ | 40.2797 ± 3.3091 | 45.2719 ± 5.8949 | 0.001[b] |
| 3 | pH | 7.3834 ± 0.0372 | 7.4138 ± 0.0478 | 0.001[b] |

([b]p<0.005 – Significant


Table 5:
Association between lung function and arterial blood gas

| | | BS (n = 83) | | | TS (n = 82) | | |
|---|---|---|---|---|---|---|---|
| Lung function | | **pO2** | **pCO2** | **pH** | **pO2** | **pCO2** | **pH** |
| Predicted FEV1 | *r* | *0.313* | *-0.087* | *0.121* | *-0.027* | *-0.3838* | *-0.026* |
| | *p* | *0.011[a]* | *0.494* | *0.34* | *0.83* | *0.0017[b]* | *0.838* |
| Predicted FVC | *r* | *0.3232* | *-0.102* | *0.125* | *-0.042* | *-0.327* | *-0.043* |
| | *p* | *0.0041[b]* | *0.422* | *0.325* | *0.74* | *0.0083* | *0.735* |
| Predicted FEV1/FVC | *r* | *0.319* | *-0.166* | *0.0302* | *0.057* | *-0.521* | *0.1889* |
| | *p* | *0.010[a]* | *0.1898* | *0.812* | *0.64* | *0.00001[c]* | *0.134* |

*[a]**p<0.05** Statistically significant [b]**p<0.005** highly significant*

*[c]**p<0.005** extremely significant*

**Discussion:**

This study provides a comparative analysis of arterial blood gas (ABG) and lung function parameters in individuals exposed to biomass fuel smoke (BS) versus tobacco smoke (TS) in a rural North Indian population. Both biomass fuel and tobacco smoke are well-established environmental risk factors for chronic respiratory diseases, yet they appear to exert distinct pathophysiological effects on the respiratory system.

Our findings indicate that biomass smoke exposure primarily affects oxygenation, as reflected by a significant negative correlation between biomass exposure index (BMEI) and arterial $pO_2$ ($r = –0.382$, $p = 0.002$), whereas $pCO_2$ and pH remain largely unaffected. This suggests that chronic inhalation of particulate matter and toxic gases from biomass combustion may lead to hypoxemia due to impaired alveolar gas exchange, consistent with previous studies reporting lower oxygen saturation and increased risk of hypoxemia in biomass-exposed populations(14,15). The small, respirable particulate matter ($PM_{2.5}$) and carbon monoxide in biomass smoke may penetrate deep into the lungs, causing alveolar inflammation and ventilation-perfusion mismatch, leading to reduced oxygenation without significant carbon dioxide retention (16,17).

In contrast, tobacco smoke exposure showed a strong positive correlation between smoking index and $pCO_2$ ($r = 0.84$, $p < 0.001$), while $pO_2$ did not show significant changes. This pattern suggests that chronic tobacco smoking predominantly impairs ventilation rather than oxygen uptake, leading to $CO_2$ retention. The observed hypercapnia may result from airway obstruction and reduced expiratory airflow, as evidenced by significant reductions in FEV1, FVC, and FEV1/FVC ratio in tobacco smokers. These findings align with established mechanisms of tobacco-induced chronic obstructive pulmonary disease (COPD), where small airway remodeling, mucus hypersecretion, and loss of elastic recoil contribute to alveolar hypoventilation and $CO_2$ retention(18,19).

Comparisons of lung function parameters further highlight differential effects of the two exposures. While both BS and TS groups demonstrated reductions in FEV1, FVC, and mid-expiratory flows (MEF25–75), tobacco smokers exhibited more pronounced impairment in airway flow rates, suggesting greater obstructive changes. In the biomass-exposed group, lung function parameters correlated positively with $pO_2$, but not with $pCO_2$ or pH, indicating that decreased ventilatory function is primarily associated with hypoxemia rather than hypercapnia. Conversely, in the tobacco group, lung function correlated negatively with $pCO_2$, emphasizing the role of airway obstruction in $CO_2$ retention rather than oxygenation deficits.

Interestingly, the mean pH in biomass users was slightly lower ($7.38 \pm 0.04$) than in tobacco smokers ($7.41 \pm 0.05$, $p < 0.001$), though both remained within physiological limits. This mild acidotic tendency in biomass users may reflect subtle respiratory compensation for chronic hypoxemia. In tobacco smokers, pH remained near normal despite hypercapnia, suggesting effective renal compensation over time.

Our results underscore that biomass smoke and tobacco smoke, while both harmful, impact gas exchange and lung function via distinct mechanisms. Biomass smoke exposure, often in poorly ventilated rural kitchens, predominantly causes hypoxemia due to alveolar impairment, whereas tobacco smoke primarily induces hypercapnia through chronic airway obstruction. These findings have important public health implications, particularly for rural populations where biomass use is prevalent and awareness of its respiratory hazards is limited. Interventions such as improved ventilation, use of cleaner cooking fuels, and tobacco cessation programs may mitigate these physiologic impairments.

A limitation of this study is its cross-sectional design, which precludes causal inference. Longitudinal studies could further clarify the temporal relationship between exposure and respiratory dysfunction. Additionally,

inclusion of female participants predominantly exposed to biomass smoke could provide gender-specific insights, as previous research has highlighted higher biomass-related COPD risk in women.

In conclusion, our study demonstrates that biomass fuel and tobacco smoke exposures differentially affect lung function and arterial blood gases. Biomass exposure is associated with impaired oxygenation, whereas tobacco smoke is linked to $CO_2$ retention and obstructive lung changes. These distinct patterns highlight the need for targeted preventive strategies and emphasize the physiological consequences of chronic exposure to environmental respiratory pollutants in rural India.

**Conflict of interest:**
The authors declare that they have no conflict of interests.
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Author contribution:
N.Vinoth kumar – Conceptualization, Data Collection
A.Sangeetha – Statistical Analysis, Manuscript writing
B.Arun kumar - Conceptualization, Data Collection, Plagiarism

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