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Original Research Article

Effect of Biomass Smoke Pollutants on Respiratory Health of Food Vendors in Maiduguri, Northeast Nigeria

Abdullahi O. Amali[1,2]*, Umar Loskurima[3], Jamila Audu Idrisa[1,4], Hadiza Umar Muktar[2], Ahmed Hammangabdo[1,2], AbdulFattah Alhaji Hassan[1,5], Musa Dungus Musa[6], Adamu Abba[7], Mustapha Lawan[1,3], Mustapha Muhammad[7], Zakiyyah Zakariyya[8], Ijuptil Chiroma[1,5], Bukar Bakki[1,2]

[1]Department of Medicine, University of Maiduguri
[2]Division of Pulmonology, Department of Internal Medicine, University of Maiduguri Teaching Hospital
[3]Division of Nephrology, Department of Internal Medicine, University of Maiduguri Teaching Hospital
[4]Division of Dermatology, Department of Internal Medicine, University of Maiduguri Teaching Hospital
[5]Division of Endocrinology, Department of Internal Medicine, University of Maiduguri Teaching Hospital
[6]Department of Chemical Pathology, University of Maiduguri Teaching Hospital
[7]Division of Infectious Diseases, Department of Internal Medicine, University of Maiduguri Teaching Hospital
[8]Division of Cardiology, Department of Internal Medicine, University of Maiduguri Teaching Hospital

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Abstract: ***Background*:** Chronic exposure to biofuel smoke from biomass fuels like wood and charcoal poses major respiratory health risks, especially in low- and middle-income countries. Commercial food vendors in Maiduguri, Northeast Nigeria, often use these fuels for cooking, but their respiratory health effects are under-researched. ***Objective***: This study examined the association between exposure to biofuel pollutants (PM2.5, CO, CO2, NO2, VOCs) and respiratory symptoms and lung function among commercial food vendors compared to controls without biomass smoke exposure. ***Methods***: A cross-sectional comparative study enrolled 195 adult food vendors and 195 matched controls. Air pollutants were measured during peak cooking hours using portable sensors at vendor workplaces and control sites. Respiratory symptoms were assessed using the Modified Medical Research Council questionnaire; ventilatory function was measured by spirometry (PEF, FEV1, FVC, FEV1/FVC). Logistic regression and correlation analyses assessed pollutant effects. ***Results***: Vendors were exposed to significantly higher median levels of PM2.5 (162 vs. 14 µg/m³), CO (36 vs. 2 ppm), CO2 (836 vs. 580 ppm), and VOCs (908 vs. 139 ppb) compared to controls ($p < 0.001$); NO2 levels were similar. PM2.5 exposure was significantly linked to increased odds of dyspnoea (OR=1.018; p=0.002). CO exposure correlated with cough (OR=1.043; p=0.051) and phlegm (OR=1.056; p=0.034). Lung function measures (PEF, FEV1, FVC) correlated positively with PM2.5 but negatively with CO2. Oxygen saturation did not differ between groups. ***Conclusions***: Commercial food vendors in Maiduguri experience high biofuel pollutant exposure associated with increased respiratory symptoms and altered lung function, underscoring the urgent need for interventions to reduce occupational exposure.

Keywords: Biofuel smoke, Respiratory health, Occupational exposure, Lung function, Spirometry.

INTRODUCTION

Chronic exposure to biofuel smoke, generated from the combustion of biomass fuels such as wood, charcoal, and animal dung, represents a significant public health challenge, particularly in low- and middle-income countries where it is a primary source of household and occupational air pollution [1, 2]. Globally, over 3 billion people rely on biomass fuels for cooking and heating, contributing to household air pollution that the World Health Organization (WHO) estimates causes approximately 3.8 million premature deaths annually, predominantly from respiratory and cardiovascular diseases [1]. In sub-Saharan Africa, where more than 70% of households depend on biomass fuels, the respiratory burden is substantial, with conditions like chronic obstructive pulmonary disease (COPD), chronic bronchitis, and asthma increasingly linked to prolonged exposure [3, 4]. Biofuel smoke is a complex mixture of

***Corresponding Author:** Abdullahi O. Amali
Department of Medicine, University of Maiduguri

pollutants, including particulate matter (PM2.5, PM10), carbon monoxide (CO), carbon dioxide (CO2), nitrogen dioxide (NO2), and volatile organic compounds (VOCs) [5], which penetrate deep into the respiratory tract, triggering inflammatory responses mediated by cytokines such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and granulocyte-monocyte colony-stimulating factor (GM-CSF) [6, 9]. These pollutants induce oxidative stress, epithelial damage, and airway remodelling, leading to symptoms such as dyspnoea, cough, and phlegm, and impair ventilatory function, measured by parameters like peak expiratory flow (PEF), forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), and FEV1/FVC ratio [8, 9]. The significance of this issue lies in its widespread impact on vulnerable populations, particularly in regions with limited access to cleaner energy sources and inadequate occupational health protections, underscoring the urgent need for targeted research and interventions to mitigate these risks. In Nigeria, commercial food vending is a prevalent occupation, particularly in urban centers like Maiduguri, Northeast Nigeria, where vendors rely on open fires fuelled by wood or charcoal for extended daily cooking activities [10]. Previous studies across Nigeria and sub-Saharan Africa have explored the respiratory impacts of biomass fuel exposure, providing a foundation for understanding its health effects. Umoh *et al*., investigated fish smokers in the Niger Delta, reporting a higher prevalence of chronic bronchitis and reduced lung function among exposed individuals compared to controls [11]. Similarly, Obiebi and Oyibo examined charcoal workers, noting elevated PM2.5 and PM10 levels at production sites, alongside increased respiratory symptoms like chronic cough and wheeze [12]. Adewole *et al*., found higher rates of chest tightness and lower FEV1 among “Mai suyas” (meat roasters) in Northwest Nigeria, highlighting the occupational risks of open-fire cooking [13]. In a broader context, Obaseki *et al*., reported that solid fuel use was associated with increased respiratory symptoms and worse quality of life among Nigerian adults, though lung function differences were less consistent [14]. Beyond Nigeria, global studies have further elucidated pollutant-specific effects. For instance, Kurmi et al.’s meta-analysis linked biomass smoke exposure to COPD and chronic bronchitis, with PM2.5 and CO as key contributors [15]. Torres-Duque *et al*., detailed the mechanisms by which PM2.5 penetrates alveoli, triggering inflammation and reducing FEV1 and FVC [16]. In China, Elbarbary *et al*., found that PM2.5 and NO2 exposure was associated with significant reductions in FEV1 and FEV1/FVC ratio among older adults, while Hou *et al*., reported NO2-related declines in FVC and inspiratory capacity [17, 18]. Yoon *et al*., in South Korea linked VOC metabolites (e.g., hippuric acid from toluene) to reduced FEV1 and FEV1/FVC in elderly populations [19]. These studies highlight the variability in pollutant effects, influenced by exposure duration, fuel type, combustion conditions, and population susceptibility, yet few have focused on food vendors in Northeast Nigeria. The purpose of this study is to investigate the association between biofuel pollutants (PM2.5, CO, CO2, NO2, VOCs) and respiratory health outcomes, specifically the prevalence of respiratory symptoms (e.g., dyspnoea, cough, phlegm, wheeze) and ventilatory function parameters (PEF, FEV1, FVC, FEV1/FVC), among commercial food vendors in Maiduguri, Nigeria, compared to a control group of non-exposed residents. The scope encompasses a community-based cross-sectional study with a comparative design, utilizing standardized tools such as the Modified Medical Research Council (MRC) questionnaire for symptom assessment, spirometry, and air quality monitoring to quantify pollutant levels. The study targets adult vendors using wood or charcoal. This research addresses a critical gap in the literature, as no such studies have been conducted in Northeast Nigeria, and also help to mitigate the respiratory risks of biofuel smoke exposure.

MATERIALS AND METHODS

Study Design and Setting:

This community-based cross-sectional study with a comparative design was conducted in Maiduguri, Borno State, Northeast Nigeria (11°51'N, 13°5'E), a city in the Sahel region with a subtropical steppe climate (average temperature: 27.4°C; rainy season: June–September). The study compared commercial food vendors exposed to biomass fuels (firewood/charcoal) with age-, sex-, and height-matched controls.

Study Population:

The study population included food vendors in Maiduguri who fry, roast, or smoke local delicacies (e.g., bean cakes, fish, suya, yam) using biomass fuels in open or partially enclosed settings. Controls were Maiduguri residents without occupational exposure to biomass smoke or other respiratory hazards.

Inclusion Criteria (Vendors):

- Aged >18 years, consenting to participate.
- Primarily using biomass fuels (wood/charcoal) for cooking.
- Worked as a food vendor for ≥1 year, ≥5 days per week.
- No prior diagnosis of chronic respiratory conditions (e.g., asthma, COPD).

Inclusion Criteria (Controls):

- Aged >18 years, consenting to participate.
- No history of work as food vendors or exposure to biomass smoke/dust/fumes.
- No prior diagnosis of chronic respiratory conditions.
- Resident in Maiduguri.

Exclusion Criteria (Both Groups):

- Aged <18 years or refusal to consent.

- Diagnosed chronic respiratory diseases (e.g., asthma, COPD, tuberculosis).
- Use of medications affecting lung function (e.g., steroids, methotrexate).
- Non-residents of Maiduguri (vendors); chest wall abnormalities (controls).
- Known contraindications for spirometry

Sample Size Determination

The sample size was calculated using Fisher's formula:
$n = z^2pq/d^2$
where (n) = sample size, z = 1.96 (95% confidence level), p = 0.867 (prevalence of respiratory symptoms from Akani *et al*.,),[20] q = 1 - p = 0.133, and d = 0.05 (margin of error). This yielded: $(1.96)^2(0.867)(1-0.867)/(0.05)^2 = 177$

With 10% attrition, the sample size was rounded to 195 vendors and 195 controls making a total of 390 across both arms of the study.

Multi stage sampling was used to recruit 195 consenting vendors and an equal number of age, sex, height and weight matched consenting controls from the 15 wards of Maiduguri. Questionnaires were administered to capture their sociodemographic characteristics and duration of exposure, followed by anthropometric measurements. A separate time was arranged for spirometry.

Data Collection

Spirometry: Ventilatory function was assessed using a portable battery-powered SP 100 spirometer (manufactured by Contec® Medical Systems Co., Ltd, China) following American Thoracic Society (ERS/ATS) guidelines [21]. Parameters measured included peak expiratory flow (PEF), forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and FEV_1/FVC ratio. Spirometry was performed between 8:00 AM and 2:00 PM to minimize diurnal variation. Obstructive ventilatory defects were defined as $FEV_1/FVC < LLN$, and restrictive defects as $FVC < LLN$ with $FEV_1/FVC \geq LLN$ [22].

Respiratory symptoms (such as dyspnoea, cough, phlegm, wheezing, blocked nose and runny nose) were evaluated using a structured, interviewer-administered Modified Medical Research Council (MRC) questionnaire. Participants were expected to answer with yes/no responses to assess prevalence. The questionnaire also collected biodata (age, sex, ethnicity, education) and occupational history.

Air quality was assessed using a portable battery powered hand held Quality Sensor to measure PM2.5, CO, CO2, NO2, and VOCs at vendor workplaces and control sites over 15-minute periods during peak cooking hours. Measurements were taken at breathing zone height to reflect personal exposure, with calibration performed per manufacturer specifications.

Anthropometric Measurements:

Height (cm) and weight (kg) were measured using a standard meter rule and a calibrated weighing scale, respectively, with participants barefoot and in light clothing based on WHO guidelines.[23] Measurements were taken twice, and the average was recorded to ensure accuracy.

Quality Control:

The spirometer was calibrated daily, and all measurements adhered to ATS acceptability and reproducibility criteria (e.g., no cough, early termination, or leaks; ≤150 mL variation between the best two FVC and FEV_1 values). Questionnaires were pre-tested on 20 participants (not included in the final sample) to ensure clarity and reliability. Research assistants were trained to standardize data collection, and all the spirometry was performed and interpreted by the principal researcher.

Statistical Analysis

Statistical analysis was performed using SPSS version 25 (IBM Corporation, Armonk, NY, USA). Continuous variables (e.g., pollutant levels, PEF, FEV1, FVC, FEV1/FVC) were summarized as means ± standard deviations (SD) or medians (interquartile range, IQR) if non-normally distributed, and compared between vendors and controls using independent t-tests or Mann-Whitney U tests. Respiratory symptom prevalence was analysed as categorical variables using frequency tables and chi-square tests. Associations between pollutant levels (PM2.5, CO, CO2, NO2, VOCs) and respiratory outcomes (symptom prevalence, ventilatory parameters) were assessed using Pearson's correlation coefficient (r) for continuous variables and logistic regression models for symptom prevalence, reporting odds ratios (ORs) with 95% confidence intervals (CIs). Statistical significance was set at $p<0.05$.

Ethical Considerations

The study was approved by the Borno State Government Ethics Committee. Written informed consent was obtained from all participants after explaining the study's purpose, procedures, risks, and benefits in English or local languages (Hausa, Kanuri). Confidentiality was maintained by assigning unique identifiers to participants, and data were stored securely on password-protected devices.

RESULTS

Out of the 390 participants who consented, twenty three (23) participants failed to avail themselves for spirometric examination. Only 367 participants (182 vendors and 185 controls) therefore had spirometry done.

Table 1, Figures 1 and 2 show a summary of the sociodemographic characteristics of the participants. The means and standard deviation of the ages of the subjects and controls were 37.18±10.17 years and 36.42±10.11 years, respectively, with a mean difference of 0.76 year. The p value was 0.303, indicating no statistically

significant differences between the two mean ages. The most common age group for both the subjects and controls was the 35-44 years group. There was no significant difference in the age distribution (p=0.358).

Table 1: Sociodemographic characteristics

Variable		Vendors N (%)	Controls N (%)	Total	X^2	P value
Age (years)	(Mean ±SD)	37.18±10.7	36.42±10.11			0.303
	Mean difference	0.76	0.76			
	Age group					
	18-24	22 (11.3)	24 (12.3)	46 (11.8)	5.498	0.358
	25-34	55 (28.2)	65 (33.3)	120 (30.8)		
	35-44	65 (33.3)	71 (36.4)	136 (34.9)		
	45-54	44 (22.6)	27 (13.8)	71 (18.2)		
	55-64	6 (3.1)	6 (3.1)	12 (3.1)		
	>65	3 (1.5)	2 (1.0)	5 (1.3)		
	Total	**195 (100.0)**	**195 (100.0)**	**390 (100.0)**		
Marital status						
Female	Married	39 (39.8)	57 (58.2)		37.08	<0.001
	Divorced	33 (33.7)	5 (5.1)			
	Single	24 (24.5)	31 (31.6)			
	Widow	2 (2.0)	5 (5.1)			
	Total	**98 (100.0)**	**98 (100.0)**			
Male	Married	71 (73.2)	53 (54.6)		8.42	<0.015
	Divorced	2 (2.1)	1 (1)			
	Single	24 (24.7	43 (44.3)			
	Total	**97 (100.0)**	**97 (100.0)**			
Education						
Female	Primary	15 (15.3)	1 (1.0)			<0.001
	Secondary	28 (28.6)	44 (44.9)			
	Tertiary	9 (9.2)	53 (54.1)			
	Quranic	46 (46.9)	0 (0)			
	Total	**98 (100)**	**98 (100)**			
Male	Primary	32 (33.0)	42 (43.3)			0.389
	Secondary	33 (34.0)	26 (26.8)			
	Tertiary	12 (12.4)	8 (8.2)			
	Quranic	20 (20.6)	21 (21.6)			
	Total	**97 (100)**	**97 (100)**			

Table 2 shows a comparative analysis of the anthropometric measurements between the vendors and the controls.

Table 2: Anthropometric measurements of participants

Weight (Kg)			
Group	**Mean ± SD**	**Mean difference**	**P value**
Vendor (n=182)	62.31±11.91	0.63	0.571
Control (n=185)	61.68±10.06		
Height (m)			
Group	**Mean ± SD**	**Mean difference**	**P value**
Vendor (n=182)	1.64±0.09	-0.07	<0.001
Control (n=185)	1.68±0.08		
BMI (Kg/m^2)			
Group	**Mean ± SD**	**Mean difference**	**P value**
Vendor (n=182)	23.27±3.91	1.52	0.022
Control (n=185)	21.74±3.06		

Table 3 presents a comparison of biofuel PM and gas levels between subjects and controls, revealing significant differences in exposure. All the pollutants (except NO_2) were substantially in the vendors than controls (p<0.001).

Table 3: Comparison of Levels of Biofuels between the Vendors and Controls

Biofuels	Levels of Biofuels* [Median (IQR)]		P-value
	Vendors (N=195)	Controls (N=195)	
PM 2.5 (μ/m^3)	162.00 (136.00 – 182.00)	14.00 (8.00 21.00)	<0.001
CO (ppm)	36.00 (25.00 – 41.00)	2.00 (1.00 – 3.00)	<0.001
NO_2 (ppb)	2.00 (1.00 – 6.00)	2.00 (1.00 – 6.00)	0.949
CO_2 (ppm)	836.00 (830 - 840)	579.50 (519.00 – 632.50)	<0.001
VOCs (ppb)	907.50 (638.50 – 1257.00)	139.00 (77.00 214.00)	<0.001

**, Mann-Whitney U Test*
IQR, Interquartile range

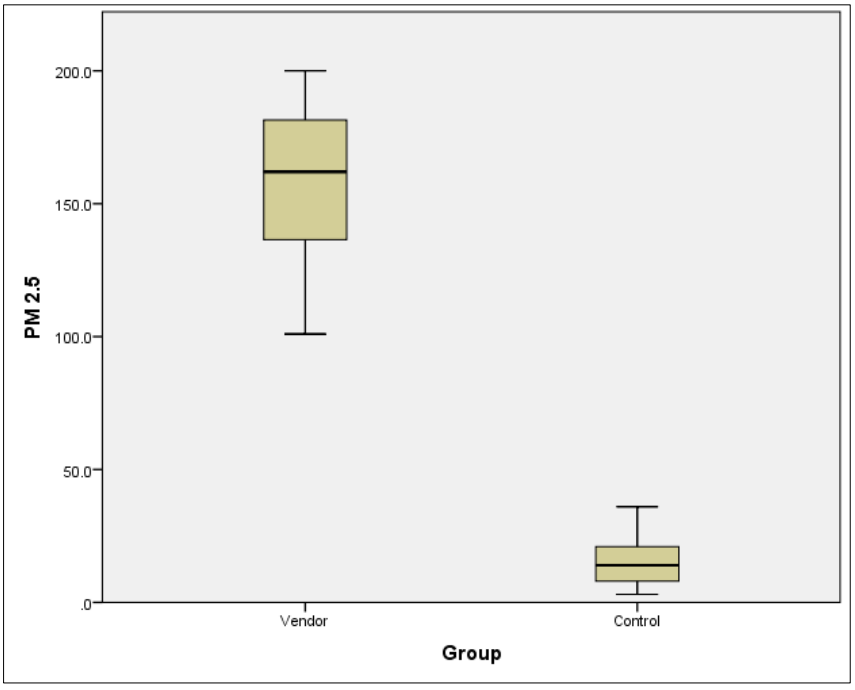

Figure 1: Box plot showing the distribution of $PM_{2.5}$ levels in vendors and controls (μ/m^3)

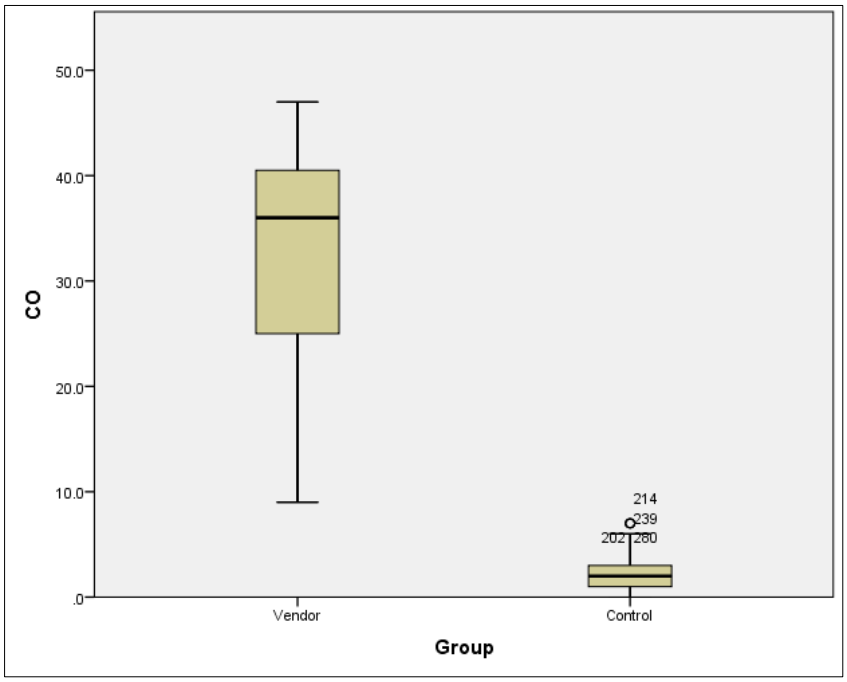

Figure 2: Box plot showing the distribution of CO levels in vendors and controls (ppm)

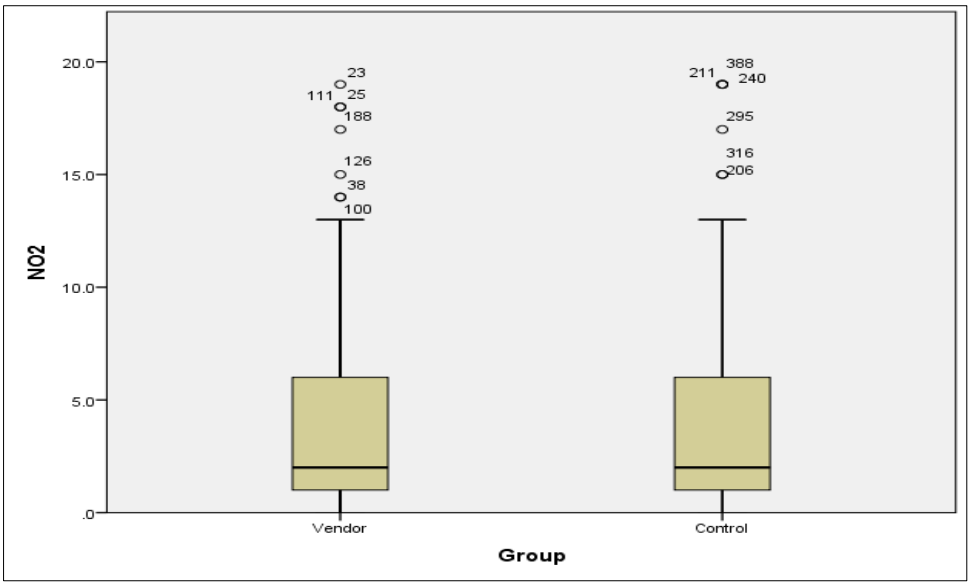

Figure 3: Box plot showing the distribution of NO_2 levels in vendors and controls (ppb)

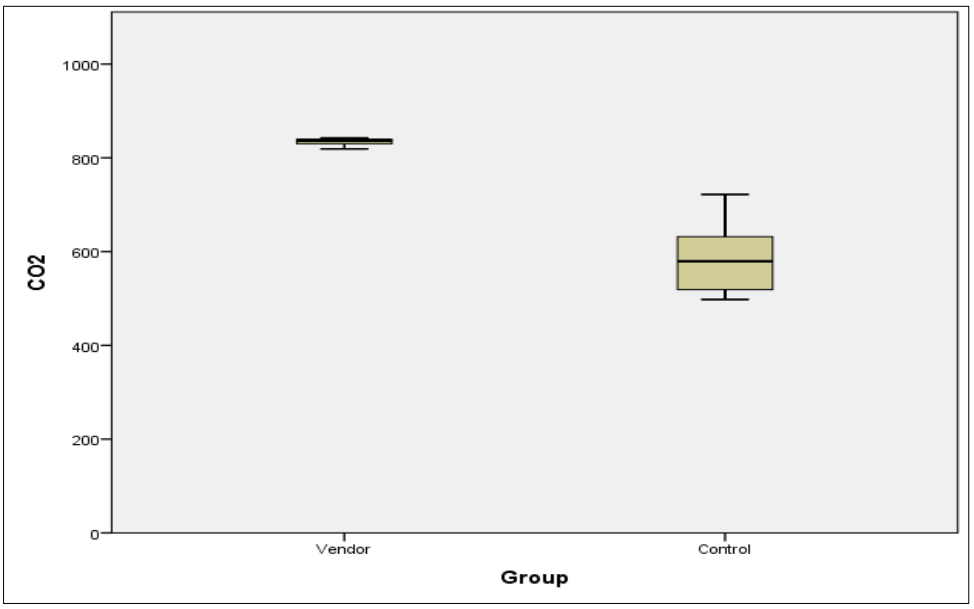

Figure 4: Box plot showing the distribution of CO_2 levels in vendors and controls (ppm)

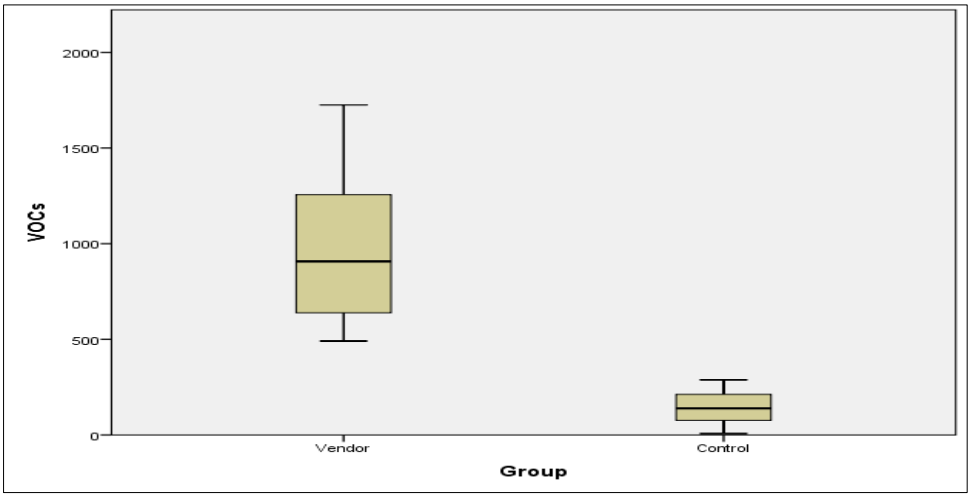

Figure 5: Box plot showing the distribution of VOCs levels in vendors and controls (ppb)

Table 4 presents an analysis of the association between various biofuel levels and respiratory symptoms. Dyspnoea shows a statistically significant association with $PM_{2.5}$ (p=0.002, OR=1.018, 95% CI: 1.01 to 1.03) and NO_2 (p=0.051, OR=1.086, 95% CI: 1.00 to 1.18). Cough demonstrates a significant association with CO levels (p=0.051, OR=1.043, 95% CI: 1.00 to 1.09).

Table 4: Association between the Level of Biofuels and Respiratory Symptoms

Symptoms*	Biofuels (Predictors)	B	P-value	OR	95% CI
Dyspnoea	PM2.5	0.018	0.002	1.018	1.01 to 1.03
	CO	0.003	0.842	1.003	0.97 to 1.04
	NO_2	0.082	0.051	1.086	1.00 to 1.18
	CO_2	-0.013	0.607	0.987	0.94 to 1.04
	VOCs	0.000	0.291	1.000	0.99 to 1.00
Wheeze	PM2.5	0.080	0.115	1.083	0.98 to 1.20
	CO	-0.139	0.608	0.870	0.51 to 1.48
	NO_2	0.015	0.879	1.015	0.84 to 1.23
	CO_2	0.006	0.320	1.006	0.99 to 1.02
	VOCs	0.003	0.564	1.003	0.99 to 1.01
Cough	PM2.5	0.010	0.155	1.010	0.99 to 1.03
	CO	0.042	0.051	1.043	1.00 to 1.09
	NO_2	-0.003	0.954	0.997	0.91 to 1.10
	CO_2	0.014	0.631	1.014	0.96 to 1.07
	VOCs	0.000	0.349	1.000	0.99 to 1.00
Phlegm	PM2.5	0.005	0.516	1.005	0.99 to 1.02
	CO	0.055	0.034	1.056	1.00 to 1.11
	NO_2	-0.006	0.914	0.994	0.89 to 1.12
	CO_2	0.061	0.091	1.063	0.99 to 1.14
	VOCs	0.000	0.801	1.000	0.99 t0 1.00
Blocked Nose	PM2.5	-0.021	0.584	0.979	0.91 to 1.06
	CO	-0.165	0.364	0.848	0.594 to 1.21
	NO_2	0.021	0.753	1.021	0.90 to 1.16
	CO_2	0.002	0.672	1.002	0.99 to 1.01
	VOCs	0.003	0.488	1.003	0.99 to 1.01
Runny Nose	PM2.5	0.011	0.213	1.011	0.99 to1.03
	CO	-0.005	0.826	0.995	0.95 to 1.04
	NO_2	0.011	0.839	1.011	0.91 to 1.13
	CO_2	0.003	0.926	1.003	0.94 to 1.08
	VOCs	0.001	0.173	1.001	1.00 to 1.00
Itchy Nose	PM2.5	-0.025	0.592	0.976	0.89 to 1.07
	CO	-0.259	0.267	0.772	0.49 to 1.22
	NO_2	0.058	0.425	1.060	0.92 to 1.22
	CO_2	-0.001	0.834	0.999	0.99 to 1.01
	VOCs	-0.006	0.230	0.994	0.99 to 1.00
Sneeze	PM2.5	0.004	0.931	1.004	0.92 to 1.10
	CO	-0.131	0.568	0.877	0.56 to 1.38
	NO_2	0.018	0.831	1.018	0.87 to 1.20
	CO_2	0.005	0.357	1.005	0.99 to 1.02
	VOCs	-0.002	0.610	0.998	0.99 to 1.01

*B, slope of the regression; CI, confidence interval; OR, odds ratio, * Logistic Regression*

Table 5 presents an analysis of the relationship between various biofuel levels and ventilatory function parameters among the vendors. $PM_{2.5}$ shows significant positive correlations with PEF (r=0.198, p=0.007), FEV_1 (r=0.160, p=0.031) and FVC (r=0.166, p=0.025). CO2 shows negative correlations with FEV_1 (r=-0.209, p=0.005) and FVC (r=-0.183, p=0.013). The others do not demonstrate any significant correlations with ventilatory function parameters.

Table 5: Relationship between the Levels of Biofuel and Ventilatory Function, N=182

Biofuel	Ventilatory Function	Correlation	P-value
PM 2.5	PEF	0.198**	0.007
	FEV1	0.160*	0.031
	FVC	0.166*	0.025
	FEV1/FVC	0.027	0.720
CO	PEF	-0.101	0.176
	FEV1	-0.037	0.616
	FVC	-0.056	0.455
	FEV1/FVC	0.042	0.574
NO_2	PEF	-0.143	0.054
	FEV1	0.041	0.578
	FVC	0.024	0.744
	FEV1/FVC	0.041	0.585
CO_2	PEF	-0.070	0.351
	FEV1	-0.209**	0.005
	FVC	-0.183*	0.013
	FEV1/FVC	-0.130	0.079
VOCs	PEF	0.088	0.236
	FEV1	0.089	0.235
	FVC	0.102	0.173
	FEV1/FVC	-0.002	0.975

**, Correlation is significant at the 0.05 level (2-tailed)*
***, Correlation is significant at the 0.01 level (2-tailed)*
****, Pearson Correlation.*

Table 6 presents the comparative analysis of oxygen saturation (SpO_2) levels between Vendor and Control groups. The Vendor group exhibited a mean SpO_2 of 97.90% (±0.790), while the Control group had a marginally higher mean of 98.04% (±0.830). The mean difference of -0.14% (95% CI: -0.32, 0.04) was not statistically significant (p = 0.415).

Table 6: Oxygen saturation (SpO_2) in vendors and controls

	Group	N	Mean ± SD (%)	Mean diff (%)	P value
Oxygen Sat (%)	Vendor	182	97.90 ± 0.790	-0.14	0.415
	Control	185	98.04 ± 0.830	-0.14	

DISCUSSION

The study's findings reveal obvious disparities in air pollutant levels between vendors and controls, with significant differences observed in all measured pollutants except nitrogen dioxide (NO_2).

Particulate matter 2.5 ($PM_{2.5}$) levels were many folds higher around the environment of the subjects, with a median of 162µg/m^3 compared to 14µg/m^3 in controls (p<0.001). This vendor exposure level is alarmingly high, exceeding the World Health Organization's recommended limits by a more than 10 times [24]. However the median levels observed fell just short of the upper limits of the WHO recommended levels in 24 hours [24]. Similar readings were observed in studies in Nigeria by Obaseki *et al*., [25], comparing respiratory symptoms and lung function parameters among users of fire wood, kerosine and LPG. The mean $PM_{2.5}$ level among the users of fire wood was 118.5±22.7µg/m^3 compared to 25.8±7.26µg/m^3 among LPG users. This mean level of $PM_{2.5}$ also exceed the WHO limits by many folds. Surprisingly the PM2.5 levels in our study was higher despite our measurements being taken outdoors rather than indoors as in their study. Similar values were also recorded in Beijing [26]. Much higher levels were recorded in a measurement of outdoor traditional wood stoves in Guatemala [27]. They observed $PM_{2.5}$ levels of 528 µg/m^3 for open fire conditions and the kitchen. This is almost 4 times the median levels recorded in this study.

Vendors in this study were exposed to median CO levels of 36ppm, which is significantly higher than the 2ppm observed in the control group (p<0.001). This exposure level among vendors substantially exceeds the World Health Organization's (WHO) recommended daily exposure limit of 3.8ppm (4mg/m^3).[89] In contrast, the median CO levels in the control group fell within the WHO's accepted limits [24]. These findings align with a study conducted in Guatemala, which reported a slightly elevated outdoor CO level of 5.9ppm near traditional wood stoves [27].

The study also revealed significantly elevated levels of both carbon dioxide (CO_2) and volatile organic

compounds (VOCs) among vendors, with median CO_2 concentrations of 836ppm versus 579ppm in controls, and median VOC levels of 907.5 versus 139 in controls, respectively (both $p<0.001$)

Notably, nitrogen dioxide (NO_2) was the only pollutant that did not show a statistically significant difference between the two groups, with both vendors and controls recording a median level of 2ppm ($p=0.949$) which is within the WHO recommended daily limit.

Dyspnoea demonstrated statistically significant positive associations with both particulate matter 2.5 ($PM_{2.5}$) and nitrogen dioxide (NO_2) concentrations. For $PM_{2.5}$, the association was highly significant ($p=0.002$) with an odds ratio of 1.018, indicating that higher levels of PM2.5 were linked to an increased likelihood of experiencing dyspnoea. A similar finding was observed in a study by Ying *et al.,* which demonstrated association between $PM_{2.5}$ and wheeze, cough and sputum. As part of The Prospective Urban and Rural Epidemiology Air Pollution study, they examined 48-hour household and personal exposure to fine particulate matter (PM2.5) and black carbon (BC) among 870 individuals using various cooking fuels across 62 communities in eight countries, followed by collection of self-reported respiratory symptoms. For each interquartile range (IQR) increase in PM2.5, there was a 25% higher likelihood of experiencing wheezing, 22% higher likelihood of coughing, and 26% higher likelihood of sputum production [28].

Ndinomholo *et al*., also demonstrated positive correlation between PM2.5 and respiratory symptoms in cross-sectional study in Namibia. A modified, standardized self-reported questionnaire was employed to gather information on participants' respiratory symptoms and conditions, family background of respiratory illnesses, job-related exposures and work history, as well as the kinds of fuel utilized for heating and cooking purposes. The study revealed a high prevalence of respiratory symptoms among the participants, with 43% reporting cough, 25% experiencing breathlessness, and 11.2% suffering from asthma. The study also found that PM exposure was a significant risk factor for episodes of coughing and phlegm production. Notably, individuals in the high PM exposure category had an increased odds ratio (OR) of 2.5 (95% confidence interval: 0.8–8.0) for experiencing episodes of phlegm and cough [29].

In this study, NO_2 levels showed a borderline significant association with dyspnoea ($p=0.051$), with an odds ratio of 1.086, suggesting that elevated NO_2 concentrations also contributed to an increased risk of breathlessness among the study participants.

The findings on NO2 in this study is similar to study in Palermo, Italy, by Cibell *et al*., They focused on indoor air quality and respiratory health among adolescents. From a larger survey of 2,150 students, researchers selected 303 participants, with an emphasis on including those with current asthma. The study involved health questionnaires, skin prick tests, spirometry, and indoor nitrogen dioxide (NO2) monitoring in participants' homes during spring and winter. The results showed that those exposed to higher indoor NO2 concentrations demonstrated significantly increased rates of current asthma ($p=0.005$), wheezing episodes in the past year ($p=<0.001$), chronic phlegm ($p=0.013$), and rhinoconjunctivitis ($p=0.008$) [30].

Also, phlegm production demonstrated a statistically significant positive association with CO levels in this study ($p=0.034$, OR=1.056), indicating that increased CO exposure was linked to a higher probability of experiencing phlegm symptoms. Cough showed a marginally significant positive association with carbon monoxide (CO) levels ($p=0.051$, OR=1.043), suggesting that higher CO exposure may slightly increase the likelihood of experiencing cough. These findings are similar to those of a study conducted in rural Ghana, in which a composite score of respiratory symptoms, including persistent cough, wheezing, and dyspnoea, showed a significant positive association with CO exposure (OR: 1.2, $p=0.03$). Individual symptom analysis revealed that wheezing had a notable correlation with CO levels (OR: 1.3, $p=0.05$), while phlegm production showed a marginally significant association (OR: 1.2, $p=0.08$) [31].

In contrast to dyspnoea, cough, and phlegm, this study found no statistically or clinically significant associations between biofuel emissions and wheezing, nasal congestion, runny nose, nasal itching, or sneezing, as evidenced by non-significant p-values and odds ratios.

The disparities in findings regarding associations between different air pollutants in biofuels and respiratory symptoms can be attributed to several factors. Variations in exposure levels and duration across studies may lead to inconsistent results, while differences in biofuel smoke composition due to fuel type, burning conditions, and local environmental influences can affect outcomes. Sample size variations and study design differences can affect statistical power and the ability to detect significant associations. Unaccounted confounding factors and temporal differences in exposure and symptom assessment may also play a role, as some effects may be acute while others are chronic. Potential synergistic effects between different pollutants in biofuel smoke might not be evident when studying individual components, and the existence of concentration thresholds could mean that certain pollutants only show significant associations above specific levels. Finally, self-reported symptoms and potential reporting bias could significantly influence the observed associations.

$PM_{2.5}$ showed significant positive correlation with PEF (r=0.198, p=0.007), FEV_1 (r=0.160, p=0.031), and FVC (r=0.166, p=0.025). In other words, an increase in the levels of $PM_{2.5}$ was associated with an increase in these parameters of lung function.

This contrasts the findings in a cohort study conducted in Taiwan, assessing the effect of long-term exposure to particulate matter on lung function and decline. $PM_{2.5}$ was associated with a reduction in lung function and increased risk for COPD. The study recruited about 285, 046 participants aged 20 years or older. The study found that for every 5 µg/m3 increase in PM2.5 concentration; FVC decreased by 1.18%, FEV1 declined by 1.46%, the maximum mid-expiratory flow (MMEF) declined by 1.65% and the FEV1/FVC ratio declined by 0.21% [32].

Another study which contrasts the finding in this study was conducted in China by Liu *et al*., The study involved 5993 participants from different provinces in China. Clusters were randomly selected from four different cities in Guangdong province. This is contrary to our study that was conducted in within one city. The much larger sample size also presents a greater statistical power. The study found that an increase of 10 mg/m3 in PM2.5 levels was associated with a 26ml decrease in FEV1, a 28ml decrease in FVC and a 0.09% decrease in the FEV1/FVC ratio [33].

Another Chinese study that contrasts the finding in this study was conducted by Elbarbary *et al*., They investigated the impact of air pollution on lung function and COPD prevalence among older Chinese adults, addressing a significant knowledge gap. The study utilized data from the WHO Study on global AGEing and adult health (SAGE) China Wave 1, which included 11,693 participants from 64 townships across China. Researchers conducted a cross-sectional analysis to explore the relationship between satellite-based air pollution exposure estimates and various lung function parameters, as well as COPD prevalence. The air pollutants examined were particulate matter with aerodynamic diameters of ≤10µm (PM10) and ≤2.5µm (PM2.5), along with nitrogen dioxide (NO2). Lung function was assessed using forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and the FEV1/FVC ratio. COPD was defined as a post-bronchodilator FEV1/FVC ratio below 70%. It was observed that, an interquartile range (IQR) increase of PM2.5 by 26.1µg/m3 was associated with a decrease in FEV1 by 71.88mL (95% CI: -92.13 to -51.64) and reduction in FEV1/FVC ratio by 2.81 (95% CI: -3.37 to -2.25). For PM10, an IQR increase of 31.2µg/m3 was linked to a decrease in FEV1 by 8.86mL (95% CI: -5.40 to 23.11) and a reduction in FEV1/FVC ratio by 1.85 (95% CI: -2.24 to -1.46) [17].

NO_2 demonstrated no significant correlation with any of ventilatory function parameters. However, negative correlation with NO_2 were observed in other studies.

For instance, Elbarbary *et al*., in a large study of 11,693 participants demonstrated that an IQR increase of 26.8µg/m3 in NO2 levels was associated with a decrease in FEV1 by 60.12mL (95% CI: -84.00 to -36.23) and a reduction in FVC by 32.33mL (95% CI: -56.35 to -8.32).

Also, Hou *et al*., demonstrated similar negative correlations in a study examining the relationship between annual concentrations of fine particulate matter (PM2.5) and nitrogen dioxide (NO2) and various measures of lung capacity among adults in Shanghai, China. The research involved 5,276 permanent residents of Shanghai aged 20 and older. Researchers estimated participants' annual exposure to PM2.5 and NO2 using sophisticated modeling techniques. The findings reveal significant negative associations between exposure to NO2 and several measures of lung function. Higher levels of NO2 was linked to lower values of FVC, inspiration capacity (IC), and vital capacity (VC). For every 10 µg/m3 increase in annual average NO2 concentration, FVC decreased by about 27 ml, IC by 65 ml, and VC by 46 ml. However, the study did not find statistically significant effects of NO2 on FEV1 [17].

CO_2 showed a negative association with FEV_1 (r=-0.209, p=0.005), FVC (r=-0.183, p=0.013).

No significant associations were found between VOCs and any of the parameters of lung function. However, studies have demonstrated a contrast to this finding.

Yoon *et al*., examined the effects of volatile organic compounds (VOCs) on lung function and oxidative stress in elderly individuals in South Korea, following 154 elderly participants over time to assess the relationship between exposure to VOCs, oxidative stress, and respiratory health. The findings revealed significant associations between urinary levels of certain VOC metabolites and reduced lung function. Specifically, hippuric acid (a metabolite of toluene) and methylhippuric acid (a metabolite of xylene) were linked to decreases in FEV1, FEV1/FVC, and forced expiratory flow at 25-75% of FVC [19].

Several previous studies have documented that exposure to pollutants from biofuel smoke leads to a deterioration of lung function [34-38].

While these measurements are presumed to be objective rather than subjective (such as symptom assessments), a number of factors could account for this discrepancy. The recorded levels of various air pollutants might not accurately represent their actual levels throughout the entire duration of vendors' activities. This discrepancy could arise because in this study measurements were taken over a brief 15-minute period,

which may not adequately reflect pollutant levels throughout the entire workday. Most of the studies cited measured air pollutant levels for much longer than 15 minutes. Consequently, these measurements may not be reliable for assessing associations. Furthermore, accurately determining the vendors' true exposure to these pollutants may prove challenging.

CONCLUSION

This study reveals that commercial food vendors in Maiduguri, Nigeria, are exposed to significantly elevated levels of biofuel pollutants—particularly PM2.5, CO, CO2, and VOCs—well above WHO guidelines. These exposures are linked to increased respiratory symptoms such as dyspnoea, cough, and phlegm, as well as alterations in lung function parameters. These findings underscore the urgent need for targeted public health interventions to reduce biofuel smoke exposure among food vendors, including promotion of cleaner cooking technologies, improved ventilation in cooking areas, and occupational health policies tailored to informal sector workers. Further longitudinal research is warranted to monitor chronic respiratory outcomes and evaluate the effectiveness of such interventions. Addressing biofuel smoke exposure in occupational settings is critical to alleviating the respiratory disease burden in Maiduguri.

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