

## Original Research Article

# Cytological Evaluations of the Respiratory Tract Epithelium of Firewood Users in Some Northern Parts of Nigeria

Sotade Gabriel<sup>1\*</sup>, Steve Oyero<sup>2</sup>, Chukwu O.O.Chukwu<sup>3</sup>, Bennett C. Nwanguma<sup>4</sup>,  
Olayoku Folasade<sup>5</sup> and Olatunji Trimiziy<sup>6</sup>

<sup>1</sup>Molecular Biology Department, Federal College of Veterinary and Medical Laboratory  
Technology Vom, Plateau State, Nigeria

<sup>2</sup>Histopathology Laboratory, Jos University Teaching Hospital Jos, Plateau State, Nigeria

<sup>3</sup>Environmental Health Department, Federal College of Veterinary and Medical Laboratory  
Technology Vom, Plateau State, Nigeria

<sup>4</sup>Biochemistry Department, University of Nigeria, Nsukka

<sup>5</sup>Chemical Pathology Department, Federal College of Veterinary and Medical Laboratory  
Technology Vom, Plateau State, Nigeria

<sup>6</sup>Histopathology Laboratory, National Veterinary Research Institute, Vom, Plateau State, Nigeria

\*Corresponding author

## A B S T R A C T

### Keywords

Smoke,  
Firewood,  
Metaplastic,  
Pulmonary  
infection,  
Respiratory  
tract

Smoke, either from the firewood or from burning of other organic fuels, has been proposed to be a major environmental risk factor for a variety of human diseases. Three hundred (300) early morning sputum samples were collected, two hundred (200) were from firewood users while the remaining one hundred (100) were from non firewood users who were used as control subjects. The 100 control subjects were all apparently healthy and normal individuals with no present or past cases of any serious pulmonary diseases. The test and the control subjects were screened using Papanicolaou staining technique, Grocott's methenamine silver technique and Perl's Prussian blue reaction for ferric ions. The test and control subjects' results were compared and it was observed that one hundred percent (100%) of the test subjects have metaplastic changes in the epithelial lining of the respiratory tract while half of them were infected with mycosis. It can be deduced from this study that wood smoke can predispose the users to pulmonary infections and metaplastic changes of the respiratory tract. All the test subjects that took part in this work have metaplastic changes while half of them were infected with mycosis.

## Introduction

Smoke inhalation is very harmful because it delivers "2 hits" to the pulmonary parenchyma. Smoke damages the lung tissue directly due to exposure of toxic compounds

in the smoke and also causes repetitive alveolar collapse and expansion to occur (Sugi *et al.*, 1990).

The sentiment that wood smoke, being a natural substance, must be benign to humans is still sometime heard. It is now well established, however, that wood, burning stoves and fireplaces as well as wild land and agricultural fires emit significant quantities of known health-damaging pollutants, including several carcinogenic compounds (e.g. polycyclic aromatic hydrocarbons, benzene, aldehydes, respirable particulate matter, carbon monoxide (CO), nitrogen dioxides (NO<sub>2</sub>), and other free radicals) (Tuthill, 1984; Koenig and Pierson, 1991; Larson and Koenig, 1994; Leonard *et al.*, 2000; Dubick *et al.*, 2002; Traynor *et al.*, 1987).

Patients who suffer severe smoke inhalation during a structure fire can develop serious pulmonary lesions that may progress into acute respiratory distress syndrome (ARDS) (Traber, 2000). Currently, the treatment for ARDS is only supportive in the form of mechanical ventilation and general critical care (Betchley *et al.*, 1997).

However, if mechanical ventilation is improperly used, it can cause ventilator induced lung injury (VILI) that exacerbates the primary injury and significantly increases mortality in ARDS patients (Boman, 2003).

The effects of wood smoke on the respiratory tract of firewood users are therefore numerous. It could cause cancer of the lungs, asphyxia (lack of oxygen), and metaplasia (Pryor, 2002).

An environment protection agency (EPA) study concludes that breathe wood smoke particles during high pollution day is equivalent to smoking 4- 16 cigarette. Some of the cancers causing chemicals found in cigarette smoke have also been found to be abundant in wood smoke (Sparrow *et al.*, 2002).

Wood smoke exposures can disrupt cellular membrane, depress immune system activity, damage the layer of cells that protects and cleanse the airways and disrupt enzyme levels (Zelikoff, 2000).

In Northern Nigeria, majority of the household use firewood as means of cooking.

The main aim of this study is for the possible changes in the lining epithelium of the respiratory tract of firewood users and to determine the extent of damage on the epithelial lining of firewood users. Another is to compare the cytological changes in the epithelial lining of the respiratory tract of firewood users and those of non users. Final aim is to educate the users on safety measures when using firewood. Also to educate the populace on the health hazards associated with careless use of firewood.

## **Materials and Methods**

Three hundred (300) subjects participated in this study with age range from 18–70 years irrespective of age, sex, or nationality. 200 were firewood users while the remaining 100 were non firewood users who were used as controls.

The 100 control subjects were all apparently healthy and normal individuals with no present or past cases of any serious pulmonary disease(s).

## **Sputum sample collection**

Early morning sputum sample used for this study was collected from all these subjects. They were instructed to gaggle mouth and buccal cavity with water and then take about four deep breaths followed by a few short cough, then inhale and cough out forcefully. They then expectorated sputum into a clean and sterile container.

**Results and Discussion**

From the macroscopic distribution of sputum appearance of test and control subjects, thirty two subjects (16%) from the test subjects produced black mucoid sputum against 0 (0%) of the control subjects. Fifty six (28%) of the test subjects produced mucoid sputum while ten subjects (10%) produced mucoid sputum among the control subjects.

All the 200 test subjects that participated in this study have metaplastic change which represents 100%. 104 (52%) out of the 200 test subjects have the samples positive for mycotic infections. The manifestation of mycotic infections among the test subjects may be as a result of continuous inhalation

of wood smoke which may have provided a conducive environment for fungi to grow. Beck and Brain in 1998 stated that besides the fuel quality, the impact of indoor air quality depends directly on ventilation and air mixing of the space. There was an experimental evidence to show that reduction in ventilation increased exposure to wood smoke, which consequently increased susceptibility of the lungs to different types of infections.

Eight (8%) of the control subjects were positive for mycoses while none of them had metaplastic changes. This agrees with the work by Sallsten *et al.* (2006) which states that 10% of individuals who were not exposed to wood smoke had pulmonary tract infections.

**Table.1** Shows the macroscopic distribution of sputum appearance of test and control subjects

Macroscopy	Test subjects		Control subjects	
	No. of cases	Percentage (%)	No. of cases	Percentage (%)
Black mucoid	32	16	0	0
Mucoid	56	28	10	10
Creamy	44	22	8	8
Brownish	24	12	4	4
Foamy	36	18	0	0
Watery	8	4	78	78
<b>TOTAL</b>	<b>200</b>	<b>100</b>	<b>100</b>	<b>100</b>

**Table.2** The cytological findings using Grocott’s modification of Gomori’s Methenamine silver method of test and control subjects

Findings	Test subjects		Control subjects	
	No. of cases	Percentage (%)	No. of cases	Percentage (%)
Fungi hypae	88	42	18	18
Spores	44	22	6	6
Abnormal				
Squamous cells	200	100	-	-
Ferruginous bodies	104	52	8	8
Dust cells	200	100	12	12
<b>TOTAL</b>	<b>200</b>	<b>100</b>	<b>100</b>	<b>100</b>

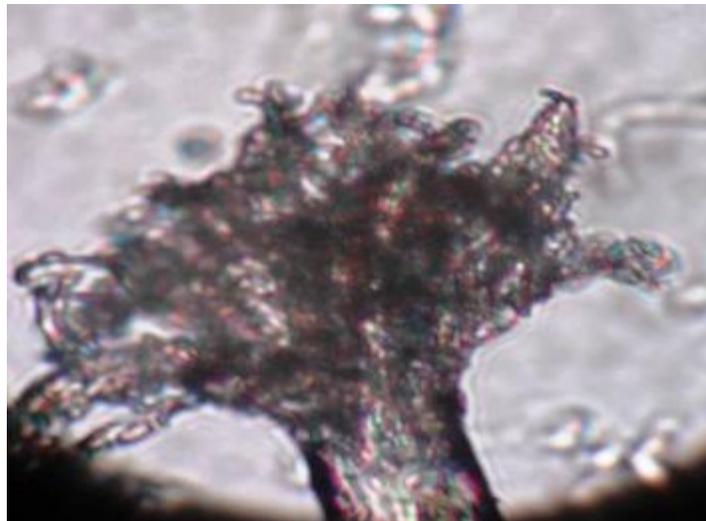
**Table.3** Cytological findings using the Papanicolaou staining technique of test and control subjects

Findings	Test subjects		Control subjects	
	No. of cases	Percentage (%)	No. of cases	Percentage (%)
Fungi hypae	72	36	12	12
Spores	60	30	6	6
Abnormal Squamous cells	200	100	-	-
Ferruginous bodies	104	52	-	-
Dust cells	200	100	-	-
Bacteria	96	48	-	-
Parasite	-	-	-	-
Curschmann's spiral	-	-	-	-
Inflammatory cells	116	58	-	-
<b>TOTAL</b>	<b>200</b>	<b>100</b>	<b>100</b>	<b>100</b>

**Table.4** Cytological findings using the Perl's Prussian blue technique of test and control subjects

Findings	Test subjects		Control subjects	
	No. of cases	Percentage (%)	No. of cases	Percentage (%)
Haemosiderin (Ferric salt)	32	16	6	6
Nuclei	200	100	100	100
<b>TOTAL</b>	<b>200</b>	<b>100</b>	<b>100</b>	<b>100</b>

**Figure.1** *Aspergillus fumigatus* (Grocott's Methenamine Silver tech., X40)



**Figure.2** *Aspergillus fumigatus* (Grocott's Methenamine Silver tech., X40)



This study also reveals numerous abnormal squamous cells which indicate metaplastic changes in the epithelial lining of the respiratory tract as a result of continuous inhalation of wood smoke among the villagers using Papanicolaou staining technique. Bacteria, inflammatory cells, ferruginous bodies and dust cells may be as a result of carbon content in wood smoke. The control subjects were negative for abnormal squamous cells, bacteria, parasite, dust cells, ferruginous bodies, curschmann's spiral and inflammatory cells.

Perl's Prussian blue technique of test and control subjects revealed that 32 (16%) of the test subjects were positive for haemosiderin (ferric salt) pigment while only 6 (6%) of the control subjects have this pigment, this may be as a result of ferruginous bodies or asbestos bodies which are composed of various substances including iron that are encrusted upon a thin needlelike fiber (Johnston *et al.*, 2002).

As was observed in this study, the use of Papanicolaou staining technique, Grocott's methenamine silver method and Perl's Prussian blue technique should be

encouraged when carrying out investigation on sputum cytology.

In conclusion, wood smoke can predispose the users to pulmonary infections, metaplastic changes of the respiratory tract and can also be infected with mycosis (*Aspergillus fumigatus*).

### **Recommendation**

We recommend the adoption of the use of improved categories of stoves to reduce the impact of smoke on people's health and the environment. Government should initiate a deliberate policy that would discourage the felling of trees for fire wood, and ensure that rural dwellers had sustainable access to kerosene and other environment friendly sources of energy, as parts of efforts to stop the use of fire wood.

### **Acknowledgment**

Special thanks to my Parents, Evangelist & Mrs. Sotade E.O, my wife, Mrs. Sotade B.D, my son and my daughter, and also to my brothers and sisters.

My sincere gratitude also goes to Olatunji Tope, Olayoku Folashade, Olawuyi Nathaniel, Opata Magdalene and the entire staff of Histopathology, Chemical Pathology and Bacteriology Laboratory (Federal College of Veterinary & Medical Laboratory technology) Vom for their moral and spiritual supports.

## Reference

- Becks, B.D., Brian, J.D. 1998. "Prediction of pulmonary toxicity of respirable combustion products from residential wood and coal stoves". Proceedings of the residential wood and coal combustion special conference. Air Pollution Control Association, Pittsburg.
- Betchley, C., Koenig, J.Q., van Belle, G., Checkoway, H., Reinhardt, T. 1997. Pulmonary function and respiratory symptoms in forest fire fighters. *Am. J. Ind. Med.*, 31: 503–509.
- Boman, B.C., Forsberg, A.B., Jarvholm, B.G. 2003. Adverse health effects from ambient air pollution in relation to residential wood combustion in modern society. *J. Work Environ. Health*, 29(4): 251–260.
- Dubick, M.A., Carden, S.C., Jordan, B.S., Langlais, P.C., Mozingo, D.W. 2002. Indices of antioxidant status in rats subjected to wood smoke inhalation and/or thermal injury. *Toxicology*, 176(1–2): 145–157.
- Johnston, F.H., Kavanagh, A. M., Bowman, D.M., Scott, R.K. 2002. Exposure to bushfire smoke and asthma: An ecological study. *Med. J. Aust.*, 176(11): 535–538.
- Koenig, J.Q., Pierson, W.E. 1991. Air pollutants and the respiratory system: Toxicology and pharmacologic interventions. *J. Toxicol. Clin. Toxicol.*, 29(3): 401–411.
- Larson, T.V., Koenig, J.Q. 1994. Wood smoke: Emissions and noncancer respiratory effects. *Annu. Rev. Public Health*, 15:133–156.
- Leonard, S.S., Wang, S., Shi, X., Jordan, B.S., Castranova, V., Dubick, M.A. 2000. Wood smoke particles generate free radicals and cause lipid peroxidation, DNA damage, NFkappaB activation and TNF-alpha release in macrophages. *Toxicology*, 150(1–3): 147–157.
- Pryor, W.A. 2002. Biological effects of cigarette smoke, wood smoke, and the smoke from plastics: The use of electron spin resonance. *Free Radical Biol. Med.*, 13(6): 659–676.
- Sallsten, G., Gustafson, P., Johansson, L., Johannesson, S., Molnar, P., Strandberg, B., Tullin, C., Barregard, L. 2006. Experimental woodsmoke exposure in humans. *Inhal. Toxicol.*, 18(11): 855–864.
- Sparrow, D., Bosse, R., Rosner, B., Weiss, S. 2002. The effect of occupational exposure on pulmonary function: A longitudinal evaluation of fire fighters and nonfire fighters. *Am. Rev. Respir. Dis.*, 128: 319–322.
- Sugi, K., Theissen, J.L., Traber, L. D., Herndon, D.N., Traber, D.L. 1990. Impact of carbon monoxide on cardiopulmonary dysfunction after smoke inhalation injury. *J. Circ. Res.*, 66: 69.
- Traber, D.L. 2000. Postgraduate course: Respiratory injury. Part II: A synopsis of respiratory function. *J. Burn Care Rehab.*, 5: 191–195.
- Traynor, G.W., Apte, M.G., Carruthers, A.R., Dillworth, J.F., Grimsrud, D.T., Gundel, L.A. 1987. Indoor air-pollution due to emissions from wood-burning stoves. *Environ. Sci. Technol.*, 21(7): 691–697.
- Tuthill, R.W. 1984. Woodstoves, formaldehyde, and respiratory disease. *Am. J. Epidemiol.*, 120(6): 952–955.
- Zelikoff, J.T. 2000. Woodsmoke, kerosene heater emission, and diesel exhaust. In: Cohen, M.D., Zelikoff, J.T., Schlesinger, R.B. (Eds). *Pulmonary immunotoxicology*, Vol. 9. Kluwer Academic., Boston. Pp. 369–387.