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# Cardiorespiratory Toxicity of Biomass Pollutants

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**Abstract** Traditional biomass alone accounts for 8.5% of global energy consumption and exclusively covers the energy needs of nearly 3 billion people and 1.2 billion of whom do not yet have access to electricity. The smoke resulting from the combustion of this biomass contains various gases having harmful effects on health. This work aims to evaluate the toxic effects of carbon monoxide, methane and hydrogen sulfide on biochemical and cardiorespiratory parameters of Wistar rats. The complete blood count, C-reactive protein, lipid profile and histology of the lungs are evaluated after subchronic exposure to biomass smoke. The results showed a decrease in red blood cells ( $p = 0.004$ ), an increase in blood platelets, an increase in C - reactive protein ( $p = 0.003$ ) and triglycerides ( $p = 0.0041$ ) and a decrease in HDL ( $p = 0, 0215$ ) after 28 days of exposure. Exposure to smoke from biomass is therefore a source of alteration of cardiorespiratory parameters.

**Keywords:** cardiorespiratory parameters, biomass, exposure, toxicity

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## 1. Introduction

Pollutants threaten the atmosphere, subsoil and oceans, as well as the species that live there [1]. Their determination of their toxic effects is necessary for ecosystems's protection. The specific health risks presented by these pollutants require a better understanding of their action mechanisms and call on various scientific disciplines. Recent and converging data from air pollution in literature suggest the presence of an effect of ambient air pollution on the risk of cardiorespiratory events [2].

During the latest estimates, published in World Health Organization [3] indicated that more than 7 million premature deaths in 2012 could be attributed to exposure to air pollution, including about 2 million 6 hundred thousand to pollution from outside. That is 1 in 8 deaths in the world. Majority of deaths from air pollution (51%) were mainly related to cardiovascular and respiratory diseases. Short-term links have been demonstrated between exposure to air pollution, particularly particulate matter, and the occurrence of ischemic heart disease. Other recent epidemiological studies have also noted a close relationship between recent exposure to particulate air pollution, especially pollution emitted by

road traffic, and the occurrence of myocardial infarction [4,5,6,7,8].

However, in Benin as in other low-income countries, the high cost of gas and energy means that many households, both rural and urban, use wood for cooking food. These fuels and cooking methods produce high levels of air pollution in homes, which involves a multitude of pollutants harmful to health [7,8], including fine particles that penetrate deep into the lungs and the blood. Several other studies have demonstrated the effect of biomass pollution on respiratory health [7,9]. Exposure to biomass smoke is responsible for respiratory worsening such as rhinitis, chronic bronchitis, obstructive ventilatory disorders and asthma symptoms [10].

This study aims to evaluate the toxic effects of carbon monoxide, methane and hydrogen sulfide on the lipid profile, inflammatory, biochemical, hematological parameters and pulmonary histology of animals.

## 2. Materials and methods

### 2.1. Ethics Statement

The Wistar rats used in this study were handled according to the Institutional animal safety guidelines

(Animal facility, Faculty of Health Science, University of Abomey-Calavi, Benin). The experiments were performed according to the Institutional Animal Ethics No. 084 MS/DC/SG/DFRS/CNPERS/SA (University of Abomey Calavi, Benin).

## 2.2. Animal Specimen, Growth and Feeding Conditions

Ten weeks old Wistar rats (both male and female) with an average weight of  $185.55 \pm 6$  g were kept in living conditions as recommended by the breeders (Sixty Wistar rats (60) aged 10-week-old male and female nulliparous Wistar strain with an average weight of  $185.55 \pm 6$  and divided into 06 batches of 10 rats were used. They were supplied by the Research Unit in Effort Physiology of the National Institute of Youth, Physical Education and Sport (INJEPS). University of Abomey Calavi, Benin). The rats were kept in sets of six per

feeding device in standard wire mesh cages with stainless steel tray floor, in a room illuminated at 12 h light, 12 h dark at 25–30°C with relative humidity of 70-80 %. Rats were fed with diet composed of 53 % crushed maize, 19 % fish meal, 20 % wheat bran, 5 % groundnut oil, 1.5 % vitamin complex (Olivitasol), and 1.5 % NaCl. The chemical analysis of the diet was 16.1 % crude protein, 12.9 % crude fiber and 2.6 % crude fat. The diet and drinking water were provided ad libitum.

## 2.3. Gas Detection and Variation

The animals are divided into a control batch and a batch exposed to the fumes from biomass combustion under actual fish smoking conditions. Exposures are carried out for 28 day. The animals are placed in a closed cylindrical jar made of transparent glass for inhalation of the smoke from biomass. The closed cylindrical jar is connected to the fireplace used for the fish smoking activity by a pipe.

The concentration of gases inside cylindrical jar is taken before and after exposure of rats using a SENKO Portable Multi Gas Detector and Analyser. The gas analyser indicates on the screen the concentration of 4 gases (oxygen O<sub>2</sub>, carbon monoxide CO, hydrogen sulphide H<sub>2</sub>S, and methane CH<sub>4</sub>) simultaneously.

## 2.4. Determination of Blood Parameters

The sample is taken on the 7th, 14th, 28th and then on

the 90th day after exposure according to guidelines 433 [11] and 413 [12] of the revised OECD standard, respectively. Blood parameters measured are white blood cells, red blood cells, hematocrit, hemoglobin, platelets and lymphocytes mean blood cell volume, mean blood cell hemoglobin concentration and mean blood cell hemoglobin content.

## 2.5. Determination of Biochemical Parameters

The determination of triglycerides, total cholesterol and HDL cholesterol are carried out in accordance with the prescriptions of the reagents of the kits of the Spanish group SPINREACT S.A.

## 2.6. Dissection

The animals are anesthetized by 0.5 ml of 2% lidocaine intraperitoneally. The abdominal cavity is incised, allowing observation of the abdominal viscera. The lungs are removed and opened with a scalpel blade for histological study.

## 2.7. Statistical Analysis

The statistical analyses of the results were carried out using Graph Pad prism Version 8.0.2 software. Comparisons of mean values were performed using parametric tests, including paired t-series tests after performing the Anova test on repeated measurements. The significance level is set at  $p < 0.05$ .

## 3. Results

### 3.1. Cell Count, Hemoglobin Content and Hematocrit in Rats

The results of the blood count of the rats after exposure are compared with the results of the control rats (Table 1 and Table 2). Red blood cells dropped from the 7th day after exposure. The drop is significant in male rats on the 7th day ( $p = 0.022$ ). In contrast, white blood cells increased significantly ( $p = 0.034$ ) on the 7th day after exposure. Blood platelets or thrombocytes significantly increased on day 14 in both sexes. The increase was more significant in females ( $p = 0.0014$ ).

Table 1. Cell count, hemoglobin content and hematocrit of male rats

	Témoins	Acute exposure		Subchronic exposure	
		7 <sup>ème</sup> jour	14 <sup>ème</sup> jour	28 <sup>ème</sup> jour	90 <sup>ème</sup> jour
RB	8.55 ± 0.85	5.73 ± 0.089**	7.96 ± 0.95	5.02 ± 0.014**	7.52 ± 1.03
WBC	8.9 ± 0.92	11.1 ± 1.24**	6.8 ± 0.72	10.5 ± 0.84**	8.8 ± 0.031
Platelets	481 ± 36.145	526 ± 20.54**	489 ± 24.13	510 ± 20.124**	432 ± 27.54
HLH	16.9 ± 1.35	12.5 ± 0.245**	15.3 ± 1.203	11.8 ± 0.023**	15.8 ± 1.57
H	52.8 ± 1.652	26 ± 0.697**	46 ± 0.142	38.4 ± 0.246**	46 ± 2.33
Lym	65 ± 2.144	75 ± 0.254**	68 ± 2.256	72 ± 1.201**	67 ± 2.72
MCV	62 ± 0.245	56.8 ± 0.324**	59.8 ± 0.231	54.8 ± 0.038**	61.3 ± 1.28
AGHC	32 ± 0.327	31.7 ± 0.216	33.2 ± 0.524	30.3 ± 1.003	34.3 ± 0.94
AGHC	19.7 ± 0.426	16.9 ± 0.048	19.2 ± 0.364	15.2 ± 0.161	21 ± 0.85

RB: Red blood cells- WBC : white blood cells- Platelets- HLH : hemoglobin level- H: hematocrit (%) – MBV: mean blood volume-MCV : mean corpuscular volume, AGHC: average globular hemoglobin concentration, AGHC: average globular hemoglobin content, \*:  $p < 0.05$ ; \*\*:  $p < 0.001$ ; \*\*\*:  $p < 0.0001$ .

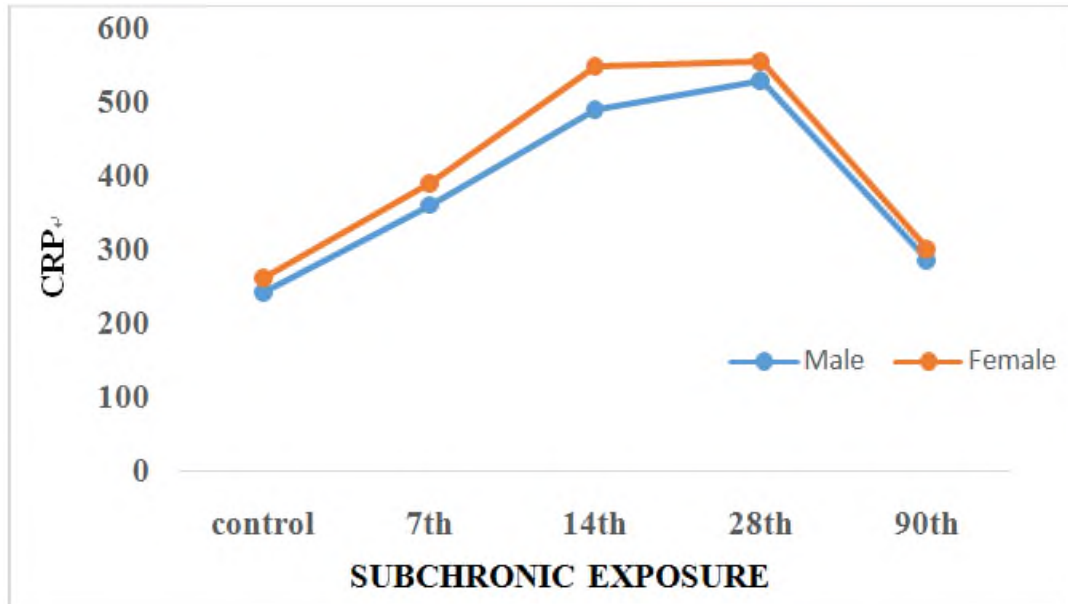
**Table 2. Cell count, hemoglobin content and hematocrit of female rats**

	control	Acute exposure		Subchronic exposure	
		7th	14th	28th	90th
<b>RB</b>	7.91 ± 0.057	6.12 ± 0.059*	7.16 ± 0.083	5.51 ± 0.257**	6.84 ± 0.37
<b>WBC</b>	5.6 ± 0.81	11.8 ± 1.10***	6.1 ± 0.021	9.49 ± 0.029***	5,9 ± 0.064
<b>Platelets</b>	483 ± 24.57	545 ± 30,33***	495 ± 34.29	665.5 ± 18.25***	497 ± 5.61
<b>HLH</b>	16.8 ± 1.56	13.4 ± 0.214**	15,9 ± 0.028	11.45 ± 0,14***	15 ± 1.03
<b>H</b>	50 ± 1.258	41.5 ± 1.563	46 ± 1.251	38,25 ± 1.76	47.5 ± 1.86
<b>Lym</b>	64 ± 1.962	74 ± 1.259***	65 ± 0.027	80 ± 1.258***	70 ± 1.54
<b>MCV</b>	63.3 ± 1,210	59.5 ± 0.341	60,5 ± 1.204	57.75 ± 0,15	63.6 ± 1.51
<b>AGHC</b>	33.6 ± 0.282	31.2 ± 0.402	32.3 ± 0.871	30.1 ± 0.053	34.4 ± 0.92
<b>AGHC</b>	21.2 ± 0.304	19.5 ± 0.209	19.5 ± 0.524	20.55 ± 0.28	21.9 ± 0.065

**RB:**Red blood cells- **WBC :** white blood cells- **Platelets- HLH :** hemoglobin level- **H:** hematocrit (%) – **MBV:** mean blood volume-**MBH:** mean blood hemoglobin **CMBLH:** concentration mean blood level hemoglobin (pg / red blood cell), \*: p < 0.05; \*\*: p < 0.001; \*\*\*: p < 0.0001.

### 3.2. C-Reactive-Protein (CRP)

The results show that CRP increased significantly from day 7 to day 28 after exposure (p= 0.003) in exposed animals before gradually falling on day 90 after exposure.



**Figure 1.** Evolution of CRP

### 3.3. Lipid Profile

In both male and female rats, a significant increase (p = 0.0041) in triglycerides was observed from day 7. In contrast, HDL-cholesterol decreased from the 14th day after exposure (p = 0.0126) in male rats and (p = 0.0251) in female rats.

**Table 3. Lipid profile of male rats**

	control	Acute exposure		Subchronic exposure	
		7th	14th	28th	90th
<b>Triglycérides</b>	0.9 ± 0.012	<b>1.55 ± 0.004**</b>	1.09 ± 0.013	<b>1.72 ± 0.029*</b>	0.76 ± 0.032
<b>Cholestérol Total</b>	1.59 ± 0.062	1.65 ± 0.021**	1.62 ± 0.009	1.69 ± 0.021**	1.52 ± 0.017
<b>HDL</b>	0.51 ± 0.005	<b>0.37 ± 0.001**</b>	0.45 ± 0.021	0.34 ± 0.018**	0.48 ± 0.002

HDL: high density cholesterol \*: p < 0,05 ; \*\*: p < 0,001.

**Table 4. Lipid profile of female rats**

	Témoins	Acute exposure		Subchronic exposure	
		7 <sup>ème</sup> jour	14 <sup>ème</sup> jour	28 <sup>ème</sup> jour	90 <sup>ème</sup> jour
<b>Triglycérides</b>	0.81 ± 0.008	1.14 ± 0.001**	1.07 ± 0.035	1.11 ± 0.215**	0.93 ± 0.021
<b>Cholestérol Total</b>	1.54 ± 0.268	1.77 ± 0.021**	1.58 ± 0.024	1.63 ± 0.005**	1.55 ± 0.004
<b>HDL</b>	0.52 ± 0.006	<b>0.42 ± 0.031*</b>	0.47 ± 0.002	0.37 ± 0.041**	0.48 ± 0.001

HDL: high density cholesterol \*: p < 0,05 ; \*\*: p < 0,001.

### 3.4. Histology of the Lungs

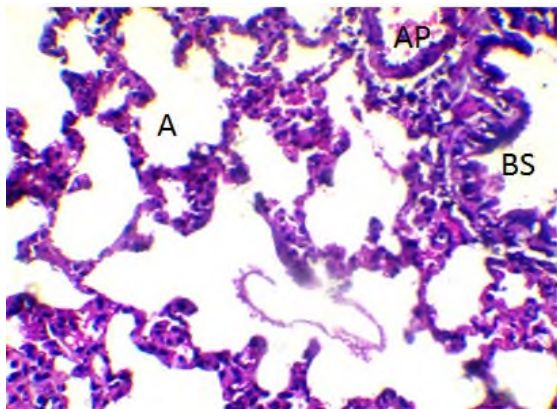


Figure 2a. Control male rat lung

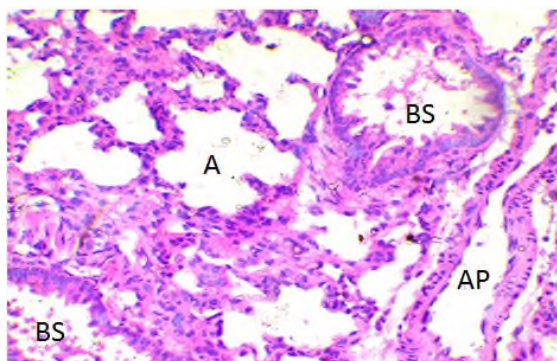


Figure 2b. Control female rat lung

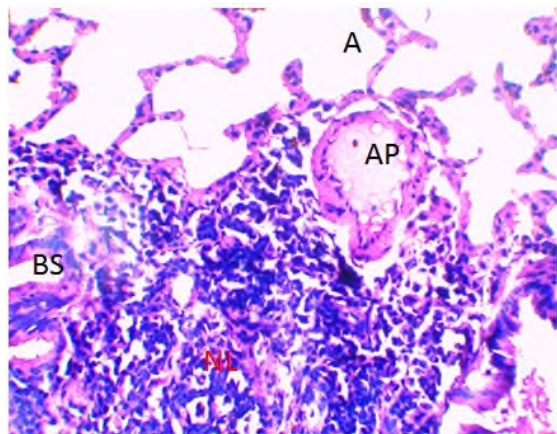


Figure 2c. Lung of exposed male rat

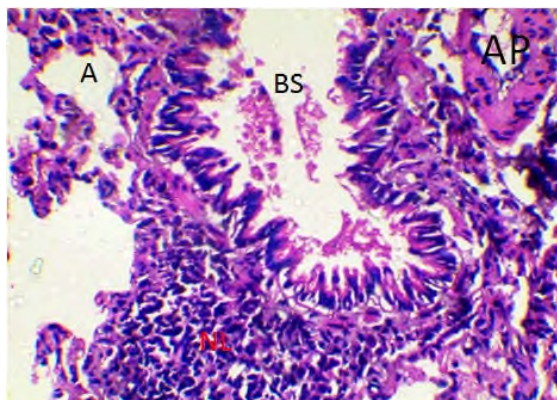


Figure 2d. Lung of exposed female rat

In control male or female rats, the lung parenchyma is typical with segmental bronchi (BS), pulmonary arteries (PA) and characteristic alveoli (A). After subchronic exposure, in both male and female rats, there is inflammatory infiltration which evolves into true lymphoid nodules (NL).

## 4. Discussion

Biochemical parameters are of primary importance in the assessment of many pathologies. They allow the evaluation of the possible toxic effect of an agent on the physiological functions of the organism [13]. Environmental and physiological factors are known to affect many blood parameters. Hematological assessment in animals is of vital interest in the diagnosis of many diseases [14].

Analysis of the data in Tables I and II showed variation in blood parameters. There was a significant decrease ( $p = 0.022$ ) in red blood cells associated with a significant increase ( $p = 0.034$ ) in white blood cells and blood platelet count ( $p = 0.0014$ ) in female rats. These changes are thought to be due to exposures to harmful pollutants from biomass burning smoke. Indeed, this smoke contains methane, hydrogen sulfide and a significant amount of carbon monoxide [15]. The latter binds to hemoglobin to form carboxyhemoglobin (HbCO) which is not suitable for respiration. These results are similar to an Algerian study [16,17]. The significant drop in red blood cells associated with an increase in white blood cells from day 7 onwards is characteristic of an infection responsible for inflammation [18]. This justifies the high level of trombocytes. Also, a significant increase ( $p = 0.003$ ) in CRP was observed on day 14. Indeed, associations between many markers of inflammation and short and long term pollution exposure have been demonstrated [19]. Exposure to biomass burning leads to airway inflammation and the effect is greater for high particle number concentrations [20]. CRP increases very rapidly in response to infection and tissue damage. In addition, the indicators of chronic inflammation observed in the histological section of the lungs could justify the increase in CRP concentration. According to several studies, there is a strong correlation between increased environmental particulate matter and detrimental effects on pulmonary and cardiovascular health [21,22]. Pulmonary effects include increased frequency of asthma exacerbations and increased lung cancer deaths. Pulmonary inflammation caused by particulate matter could lead to a systemic increase in blood clotting [23,24]. The significant increase in triglycerides and the significant decrease in HDL levels have as direct consequences, the occurrence of complications related to hypercholesterolemia [25,26,27,28]. In fact, according to these authors, a high plasma concentration of very low density lipoprotein (VLDL) can explain at least in part a decrease in HDL-c in situations of metabolic syndrome.

Indeed, the increase of VLDL circulating stimulates in an exaggerated way the activity of the enzyme Cholesteryl ester transfer protein (CETP) which carries out exchanges of triglycerides and cholesterol between lipoproteins rich in triglycerides (VLDL, chylomicrons) and lipoproteins rich in (LDL, IDL, HDL).

This involved the increase of HDL with triglycerides and their depletion in cholesterol. In addition, the clearance of these HDL rich in triglyceride is increased.

These results are explained by the presence of the identified gases in the biomass smoke and are supported by a German study that showed that quitting smoking increases HDL cholesterol by about 10% [29]. Other authors [30] have also justified that changes in HDL levels could occur even after brief exposure to air pollution.

## 5. Conclusion

Ultimately, air pollution is a major health problem. The results showed a variation of physiological parameters compared to controls. These variations would be responsible for the occurrence of cardio-respiratory diseases in women engaged in the activity of fish smoking.

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