American Journal of Medical Sciences and Medicine, 2021, Vol. 9, No. 3, 70-74
Available online at http://pubs.sciepub.com/ajmsm/9/3/2
Published by Science and Education Publishing
DOI:10.12691/ajmsm-9-3-2

Cardiorespiratory Toxicity of Biomass Pollutants

Judith F. Ahounou Aïkpe[1,2,*], Jean-Bénoît M. Godonou[1], Maxim Senou[3,4], Credo R. Koukpoliyi[1], Armel Hounkonnou[1], Huguette B Akakpo[1,2], Joachim D. Gbenou[2], Pierre H. Dansou[1]

[1]Research Unit in Exercise Physiology (URPEF), University of Abomey Calavi, 01 BP 169 Porto-Novo, Bénin
[2]Laboratory of Pharmacognosy and Essential Oils (LAPHE), Faculty of Health Sciences,
Faculty of Sciences and Techniques, University of Abomey Calavi. 01BP 918 Cotonou, Benin
[3]Experimental and Clinic Biology Laboratory, National School of Applied Biosciences and Biotechnology, National University of Science, Technology, Engineering and Mathematics (UNSTIM), Dassa-Zoumé, R. Benin
[4]Research Laboratory in Applied Biology, Polytechnic School ofAbomey-Calavi,
University of Abomey-Calavi, Cotonou, R. Benin
*Corresponding author: judifam@yahoo.fr

Received May 27, 2021; Revised July 03, 2021; Accepted July 11, 2021

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*

Cite This Article: Judith F. Ahounou Aïkpe, Jean-Bénoît M. Godonou, Maxim Senou, Credo R. Koukpoliyi, Armel Hounkonnou, Huguette B Akakpo, Joachim D. Gbenou, and Pierre H. Dansou, "Cardiorespiratory Toxicity of Biomass Pollutants." *American Journal of Medical Sciences and Medicine*, vol. 9, no. 3 (2021): 70-74. doi: 10.12691/ajmsm-9-3-2.

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 ± 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 ± 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_2, carbon monoxide CO, hydrogen sulphide H_2S, and methane CH_4) 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

		Acute exposure		Subchronic exposure	
	Témoins	**7ème jour**	**14ème jour**	**28ème jour**	**90ème 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

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

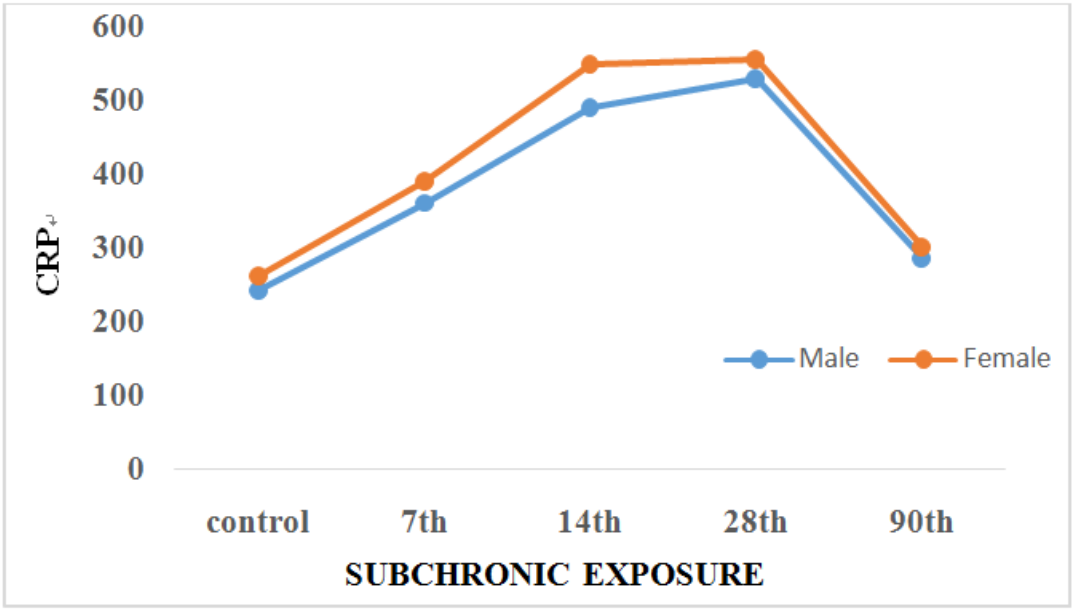

Figure 1. Evolution of CRP

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

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

		Acute exposure		Subchronic exposure	
	Témoins	**7ème jour**	**14ème jour**	**28ème jour**	**90ème jour**
Triglycérides	0.81 ± 0.008	1.14 ± 0.001**	1.07 ± 0.035	1.11 ± 0.215**	0.93 ± 0.021
Cholestérol Total	1.54 ± 0.268	1.77 ± 0.021**	1.58 ± 0.024	1.63 ± 0.005**	1.55 ± 0.004
HDL	0.52 ± 0.006	**0.42 ± 0.031***	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

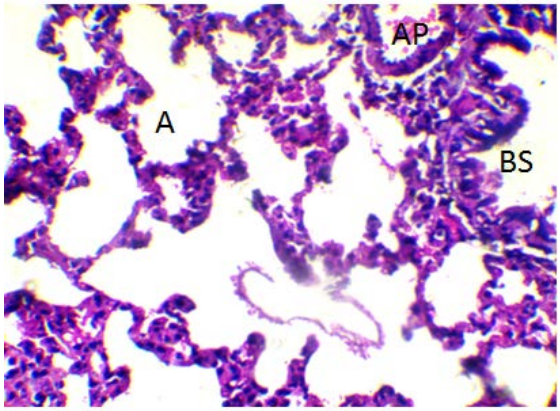

Figure 2a. Control male rat lung

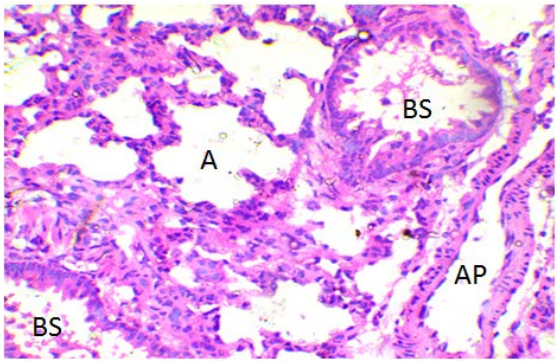

Figure 2b. Control female rat lung

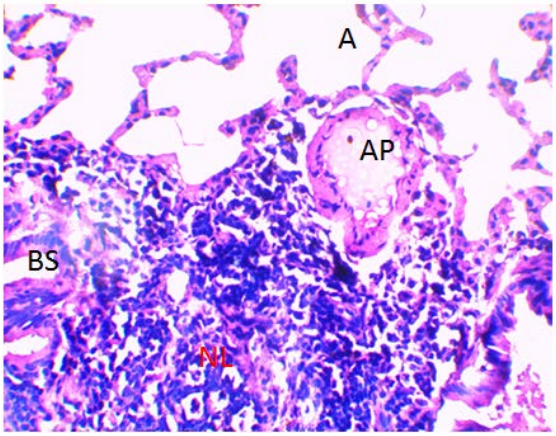

Figure 2c. Lung of exposed male rat

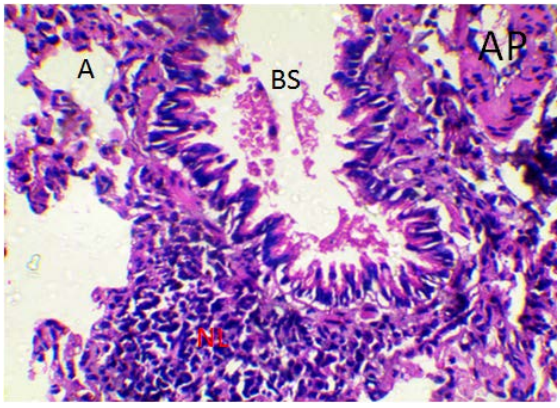

Figure 2d. Lung of exposed female rat

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 significative 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.

References

[1] Boukerche S., Aouacheri W., Saka S. Toxicological effects of nitrate: biological study in human and animal. Ann Biol Clin, 65 (4) : 385-91. 2007.

[2] Zeller M., Royer C., Besancenot J.P., Cottin Y. Air pollution and cardiovascular disease. La Lettre du Cardiologue. (404): 14-9. 2007.

[3] Who.int [Internet]. Pregny-Chambésy: *Organisation Mondiale de la Santé*. Aide-mémoire n°292.c1948-04 Février 2016; [Consulté le 22 Août 2016]. Disponible : http://ww.who.int/whr/2016/fr/index.html.

[4] Peters A. Particulate matter and heart disease: Evidence from epidemiological studies. Toxicol Appl Pharmacol. 2005; 207(2 suppl): 477-82.

[5] Lanki T., Pekkanen J., Aalto P. et al. Associations of traffi crelated air pollutants with hospitalisation for first acute myocardial infarction: the HEAPSS study. Occup Environ Med; 63(12): 844-51. 2006.

[6] Zanobetti A., Schwartz J. Air pollution and emergency admissions in Boston, MA. J Epidemiol Community Health. 60: 890-5. 2006.

[7] Kavouras I.G. et Chalbot M.C.G. Réponse inflammatoire, symptômes respiratoires et dégradation de la fonction respiratoire suite à l'exposition à la combustion de biomasse. Une perspective globale. Anses. Bulletin de veille scientifique. Santé / Environnement / Travail. (18): 23-6. 2012.

[8] Ahounou Aïkpe F.J., Godonou M.J.B, Hounmondji E., Akakpo B.H., Gbenou D.J. and Dansou H.P. Effect of firewood smoking on the respiratory parameters of fish smokers. The Pharma Innovation Journal9(3): 697-702. . 2020.

[9] Marano F., Aubier M., Brochard P., De Blay F., Marthan R., Nemery B., et al. Impact des particules atmosphériques sur la santé : aspects toxicologiques. Environnement, Risques et Santé. 3(2): 241-55. 2004.

[10] Agodokpessi G., Ade G., Hinson V., Ade S., Okoumassou C.X., Fayomi B., et al. Prévalence des troubles respiratoires chez des femmes exerçant sur un site artisanal de fumage de poisson à Cotonou au Bénin. Mali Médical. Tome XXVI n°4: 34-8. 2011.

[11] OECD (2018), Ligne directrice de l'ocde pour les essais de produits chimiques. Toxicité aiguë. Document 413. ENV Publications. Series on Testing and Assessment, Paris. 2018.

[12] OECD (2018), Ligne directrice de l'ocde pour les essais de produits chimiques. Toxicité (subchronique) par inhalation: étude sur 90 jours. Document 413. ENV Publications. Series on Testing and Assessment, Paris. 2018.

[13] Smaoui M., Ghurbel F., Buujelbene M., Makni-Ayadi F., El FEKI A. Impact de l'exposition chronique aux gaz d'échappement d'origine automobile sur certains biomarqueurs touchant la fonction hormonale sexuelle mâle, la fonction rénale et l'hémogramme chez le rat. Pollution Atmosphérique. (167): 439-49. 2000.

[14] Diaby V., Yapo A.F., Adon A.M., Yapi H.F., Djama A.J., Dosso M. 2016. Biotoxicité hématologique du sulfate de cadmium chez les rats *Wistar*. *Int. J. Biol. Chem. Sci.*, 10(4): 1765-1772. 2016.

[15] Godonou J-B.M., Ahounou Aikpe J.F.., Koukpoliyi C. R., Hounkonnou A.., Akakpo H. B., Kotin L., Gbenou D. J., Dansou H. P. Toxicity of biomass pollutants in Wistar rats. IJMSIR- January - Vol – 6, Issue - 1, P. No. 235-241. 2021.

[16] Tadjine A., Djebar H., Courtois A. Toxicité des poussières rejetées par le complexe sidérurgique d'Annaba sur quelques paramètres hématologiques du lapin *Europeus*. Environnement, Risques & Santé. 7 (3): 209-15. 2008.

[17] Lefranc A., Larrieu S. Pollution atmosphérique et risque cardiovasculaire. Correspondances en Risque CardioVasculaire. 2008. 6 (1): 19-23.

[18] Mathieu M., Guimezanes A. InflammatIon et maladies: clé de compréhension 2011-2012. 72p.

[19] Ravindra K., Sokhi R., VanGrieken R. Atmospheric polyclyclic aromatic hydrocarbons: Source attribution, emission factors and regulation. Atmospheric Environment. 42: 2895-921. 2008.

[20] Rutter M.K., Meigs J.B., Sullivan L.M. et al. C-reactive protein, the metabolic syndrome, and prediction of cardiovascular events in the Framingham offspring study. Circulation. 110:380-5. 2004.

[21] Pope III C.A., Burnett R.T., Thun M.J., *et al.* Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA.* 287: 1132-41. 2002.

[22] Delvin R.B., Ghio A.J., Kehrl H., Sanders G., Cascio W. Elderly humans exposed to concentrated air pollution particles have decreased heart rate variability. *Eur Respir J. Suppl* 40: 76-80. 2003.

[23] Harrison R.M., Yin J. Particulate matter in the atmosphere: Which particle properties are important for its effects on health? *Sci Total Environ* 249: 85-101. 2000.

[24] Hajat S., Anderson H.R., Atkinson R.W., Haines A. Effects of air pollution on general practitioner consultations for upper respiratory diseases in London. *Occup Environ Med.* 59: 294-9. 2002.

[25] Chagnon A. Contribution de certaines anomalies biologiques au diagnostic du paludisme. Med Mal Infect. 1999; 29 Suppl 3: 302-6.

[26] Montonen J., Boeing H., Fritsche A., Schleicher E., Joost H.G., Schulze M.B. et al. Consumption of red meat and whole-grain bread in relation to biomarkers of obesity, inflammation, glucose metabolism and oxidative stress. Eur J Nutr. 52(1): 337-45. Feb. 2013.

[27] Sahach V.F., Korkach I.U.P., Kotsiuruba A.V., Prysiazhna O.D. The inhibition of oxidative and nitrosative stresses by ecdysterone as the mechanisms of its cardio- and vasoprotective action in experimental diabetes type I. Fiziol Zh. 54(5): 4654. 2008.

[28] Boris Hansel, Alain Carrié, Éric Bruckert. Les hypo- et les hyper-HDLémies. Métabolismes Hormones Diabètes et Nutrition. XI (4): 167-74. 2007.

[29] Walter F.R., Markus H. HDL bas-haut risque, HDL haut-faible risque ? 2008: 8(14): 24-62.

[30] Masson D., Yvan-Charvet L. L'augmentation du HDL-cholestérol Un traitement d'avenir dans le domaine de l'athérosclérose? Médecine/sciences 24: 341-74. 2008.

© The Author(s) 2021. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (http://creativecommons.org/licenses/by/4.0/).