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Persistent exposure to wood smoke is associated with variations in biochemical and hematological indices among regular wood burners in the Cape Coast metropolis, Ghana

Emmanuel K. Dadzie, Richard K.D. Ephraim, Justice Afrifa, Richard Quaicoe, Benjamin S. Bediako, Benedict I. Duku, Townshed Adjetey, Isaac Dadzie*

Department of Medical Laboratory Science, School of Allied Health Sciences, University of Cape Coast, Ghana

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ABSTRACT

Background: Acute and chronic exposure to wood smoke is associated with adverse health effects. This study investigated the potential effect of persistent exposure to woodsmoke on hematological and biochemical indices among regular wood burners in the Cape Coast Metropolis, Ghana.

Methodology: A total of 101 consenting participants were recruited into the study. Serum lipids (total cholesterol, high density lipoprotein, triglyceride, low density lipoprotein, very low-density lipoprotein and non-HDL), renal function (urea, creatinine), liver enzymes (AST and ALT) were measured using an automated analyzer. The full blood count of participants was also measured with an FBC automated analyzer. Demographic and lifestyle data of the participants were obtained with the help of questionnaires.

Results: Individuals exposed to wood smoke were older (47.21±1.583), smoked fish [36(47.4%)] and were predominantly females. Female preponderance [75(98.7%), $P=0.046$], low haemoglobin (HGB) [OR= 6.553 (95%CI=1.431 to 30.01), $P=0.0094$], low MCV [OR=12.43(95%CI=0.7133 to 216.5), $P=0.018$], low MCH [OR=4.145(95%CI=1.284 to 13.38), $P=0.0151$], low MCHC [OR=9.844(95%CI=3.029 to 31.99), $P<0.0001$], low granulocyte [OR=12.88(95%CI=4.382 to 37.84), $P<0.0001$] and high lymphocyte [OR=21.86(95%CI=6.991 to 68.34), $P<0.0001$] were significantly associated with exposure to wood smoke. High cholesterol [OR=20.44(95%CI=2.610 to 160.2), $P=0.0002$], triglyceride [OR=17.60(95%CI=1.022 to 303.2), $P=0.0052$], non-HDL cholesterol [OR=22.15(95%CI=5.490 to 89.38), $P<0.0001$] and low HDL cholesterol [OR=96(95%CI=12.00 to 767.9), $P<0.0001$] were significantly associated with exposure to wood smoke and are at increased risk of developing coronary heart disease (CHD) [OR=474.7(95%CI=25.75 to 8750), $P<0.0001$]. Age and duration of exposure significantly correlated with cholesterol ($r=0.27$, $P=0.02$; $r=0.25$, $P=0.03$), LDL cholesterol ($r=0.31$, $P=0.01$; $r=0.28$, $P=0.01$) and non-HDL ($r=0.31$, $P=0.01$; $r=0.24$, $P=0.03$). AST levels among individuals exposed to wood smoke were also elevated [OR=69.83(95%CI=4.097 to 1190), $P<0.0001$]).

* Corresponding author.
E-mail address: idadzie@ucc.edu.gh (I. Dadzie).

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Conclusion: Wood smoke exposure is associated with variations in some biochemical and hematological indices among study participants.

Introduction

Wood smoke constitutes an array of solid, liquid, and gases that change with time and temperature and interacts with other pollutants, water vapour and surfaces. Burning wood for residential heating is increasingly associated with higher cost for utilities, the availability of wood as a renewable resource, and the promotion of wood as a greenhouse gas neutral energy source [1–3]. Approximately, one-third of the world's population and most rural households in developing countries rely heavily on unprocessed biomass fuels for cooking and heating. There is much concern about the systemic effects of air pollution on human health [4] and wood smoke is a major contributing factor. Wood burning as a major source of fuel has been in existent for a long time, however unlike coal, the fibrous nature of wood makes it difficult in the size reduction process [5] According to the Ghana Energy Commission, wood fuels contribute over 60% of the total energy consumed in Ghana. Moreover, in Ghana, about 2.5% of the women population are involved in commercial fish smoking [6], most of whom use wood for fuel in preparation of both domestic and commercial local foods such as kenkey and gari.

Much like tobacco and cigarette smoke, wood smoke contains toxic pollutants including carbon monoxide (CO), respirable particulate matter, nitrogen dioxide, benzene and other free radicals that can cause cancer and other health hazards [3]. The soluble fractions of wood smoke is able to cross to the extra-pulmonary circulations thereby increasing the chance to harm extra-pulmonary organs including the kidney, liver and heart [7,8]. The World Health Organization (WHO) estimates that more deaths (i.e. 4.3 million deaths per annum) are caused from smoke-induced diseases than deaths caused by malaria and tuberculosis, thus making it one of leading causes of deaths worldwide [9]. As many as 396,000 deaths in sub-Sahara Africa, as a result of indoor smoke were reported in 2002 [10].

Due to the generally poor educational background of rural folks engaged in commercial wood burning for cooking and other activities and limited use of personal protective equipment and efficient occupational safety methods, exposure to smoke are prevalent among such people. Nevertheless, few studies have tried to assess possible clinical implications of wood smoke exposure among these workers. There have been reports on the possible decline of the respiratory system function among people exposed to wood smoke [3]. Carbon monoxide, one of the principal gaseous pollutants in wood smoke competitively bind to haemoglobin, inducing hypoxia and necrosis as tissues become less oxygenated. Also wood smoke is known to induce alterations in phagocyte-mediated oxidative stress response and antioxidant status [3]. In bridging the gap, we sought to investigate the possible association of haemato-biochemical markers with wood smoke exposure among commercial wood burners in the Cape Coast Metropolis. To the best of our knowledge this study serves as the first of its kind among such group of workers in the west African region and hence provides background data for further studies.

Methodology

Study site/ setting

This study was undertaken in the Cape Coast Metropolis in the Central Region of Ghana. Cape Coast is a coastal area with a population of around 169,894 with the majority engaged in fishing activities.

Study population

A total of 101 consenting participants were recruited into the study. Simple convenience sampling was employed. Of these, 76 (75.25%) were exposed to wood smoke (commercial wood burners) whereas the remaining 25 (24.75%) were non-exposed. The exposed group referred to individuals who use wood as source of fuel in preparing and cooking of food (frying gari, preparing kenkey and smoking fish) on commercial basis whiles the unexposed group referred to individuals involved in the distribution and selling of these foods.

Exclusion/ inclusion criteria

Individuals who were directly and frequently exposed to wood smoke were recruited into the study. Cigarette and tobacco smokers, alcoholics and participants with known complications such as renal, hepatic, and cardiovascular diseases were excluded. Those on any medication were also excluded from the study. These were first verified after checking their medical history, and with the help of questionnaires and qualified medical personnel.

Table 1
Demographic distribution of the study participants stratified into cases and control.

Variable	Total ($n=101$)	Case ($n=76$)	Control ($n=25$)	P-value
Age (years)	46.4 ± 1.426	47.21 ± 1.583	43.92 ± 3.175	0.321[t]
20–30	17(16.8)	9(11.8)	8(32.0)	0.143[c]
31–40	18(17.8)	15(19.7)	3(16.0)	
41–50	25(24.8)	21(27.6)	4(16.0)	
51–60	25(24.8)	20(26.3)	5(20.0)	
61 and above	16(15.5)	11(14.5)	5(20.0)	
Gender				
Male	4(4.0)	1(1.3)	3(12.00)	**0.046[c]**
Female	97(96.0)	75(98.7)	22(88.00)	
Marital Status				
Single	44(43.6)	31(40.8)	13(52.0)	0.359[c]
Married	57(56.4)	45(59.2)	12(48.0)	
Level of education				
None	32(31.7)	25(32.9)	7(28.0)	**0.036[c]**
Basic	57(56.4)	46(60.5)	11(44.0)	
Senior high school	8(7.9)	3(3.9)	5(20.0)	
Tertiary	4(4.0)	2(2.6)	2(8.0)	
Occupation				
Fish	46(45.5)	36(47.4)	10(40.0)	0.535[c]
Gari	31(30.7)	24(31.6)	7(28.0)	
Kenkey	24(23.8)	16(21.1)	8(32.0)	

Values are presented as frequency (percentages), Mean ± SD; [t]student sample t-test; [c]Chi-square test.

Ethical consideration

Verbal and written consent was sought from the participants before enrolment into the study.

Sample collection and analysis

After an overnight fast, venous blood samples (5 ml) were taken into gel separator and EDTA tubes. Blood collected into the EDTA tube was run in an FBC analyzer. The sample in the gel separator tube was allowed to clot and spun at 2200 rpm for 15 min and analysed for the lipid profile [total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDC-c), high density lipoprotein cholesterol (HDL-c), non HDL-c, liver function: aspartate aminotransferase (AST), alanine aminotransferase (AST) and renal function (urea and creatinine)] by automation using the clinical data selectra junior chemistry analyzer from Elitech group.

Statistical analysis

Data collected was entered into Microsoft Excel Spreadsheet 2016, validated and analysed using Graph Pad Prism Version 7.01 for Windows (Graph Pad Software, San Diego, CA, USA). Independent Sample *t*-test was used to determine the significance of the differences in two independent variables of continuous data. Chi-square test was performed to test difference between categorical variables. Values are presented as frequency (percentages), mean ± standard deviation. Statistically significant level was put at $P < 0.05$.

Results

Table 1 shows the socio demographic distribution of the study participants stratified into cases (exposed) and control (non-exposed). The mean age of the study participants was 46.4 ± 1.426 with the majority being in the age group 41–60 [50(49.6%)], females [97(96%)], married [57(56.4%)], having basic education [57(56.4%)] and smoking fish [46 (45.5%)]. Individuals exposed to wood smoke were older (47.21±1.583), married [45(59.2%)], smoked fish [36(47.4%)] and had statistically significant basic educational level [46(60.5%), $P=0.036$] with female preponderance [75(98.7%), $P=0.046$].

Serum urea, creatinine and ALT levels showed no significant difference between the exposed and non-exposed groups. However, AST levels were significantly elevated among the exposed group as compared to the non-exposed group ($P<0.0001$) (Fig. 1).

The hematological profile indicated that low hemoglobin [OR= 6.553 (95%CI=1.431 to 30.01), $P=0.0094$], low MCV [OR=12.43(95%CI=0.7133 to 216.5), $P=0.018$], low MCH [OR=4.145(95%CI=1.284 to 13.38), $P=0.0151$], low MCHC [OR=9.844(95%CI=3.029 to 31.99), $P<0.0001$], low GRAN [OR=12.88(95%CI=4.382 to 37.84), $P<0.0001$] and high LYM [OR=21.86(95%CI=6.991 to 68.34), $P<0.0001$] were significantly associated with exposure to wood smoke (Table 2).

Table 3 shows the multivariate logistic regression of the lipid profile of the study participants. The lipid profile indicated high cholesterol [OR=20.44(95%CI=2.610 to 160.2), $P=0.0002$], triglyceride [OR=17.60(95%CI=1.022 to 303.2), $P=0.0052$],

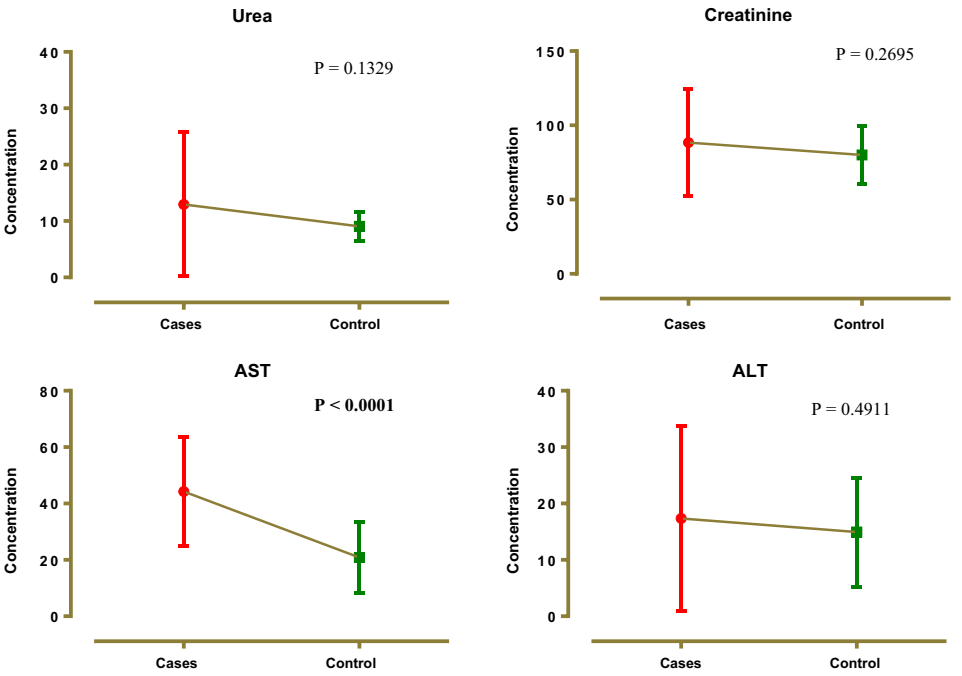

Fig. 1. Kidney and liver markers of the study participants.

Table 2
Multivariate logistic regression of the haematological profile of the study participants.

Variable	Cases ($n = 76$)	Control ($n = 25$)	P-value	OR (95% CI)	P-value
HB	11.39 ± 0.1446	12.00 ± 0.2079	**0.0329**		
Low	28(36.84)	2(8.00)	**0.0199**	6.553 (1.431 to 30.01)	**0.0094**
Normal	47(61.84)	22(88.00)		*	
High	1(1.32)	1(4.00)		0.4681(0.02795 to 7.840)	0.5461
WBC	5.157 ± 0.1533	5.712 ± 0.2933	0.1015		
Low	15(19.74)	1(4.00)	0.1403	6(0.7501 to 48.00)	0.0658
Normal	60(78.95)	24(96.00)		*	
High	1(1.32)	0(0.00)		1.215(0.04779 to 30.89)	1
PLT	231.7 ± 7.424	240.0 ± 10.98	0.5657		
Low	2(2.63)	0(0.00)	0.2071	1.96(1.960)	1
Normal	62(81.58)	24(96.00)		*	
High	12(15.79)	1(4.00)		4.645(0.5721 to 37.71)	0.1747
HCT	35.86 ± 0.4506	37.18 ± 0.7938	0.1501		
Low	37(48.68)	9(36.00)	0.1374	1.581(0.6170 to 4.052)	0.3594
Normal	39(51.32)	15(60.00)		*	
High	0(0.00)	1(4.00)		2.747(0.1338 to 56.38)	0.5586
MCV	85.59 ± 0.8370	87.22 ± 0.8878	0.2953		
Low	14(18.42)	0(0.00)	**0.0347**	12.43(0.7133 to 216.5)	**0.018**
Normal	59(77.63)	25(100.00)		*	
High	3(3.95)	0(0.00)		3(0.1494 to 60.26)	0.554
MCH	27.05 ± 0.4421	27.40 ± 0.3342	0.6577		
Low	30(39.47)	4(16.00)	**0.0089**	4.145(1.284 to 13.38)	**0.0151**
Normal	38(50.00)	21(84.00)		*	
High	8(10.53)	0(0.00)		9.494(0.5217 to 172.8)	**0.0495**
MCHC	31.66 ± 0.3597	32.73 ± 0.2760	0.1029		
Low	45(59.21)	4(16.00)	**< 0.0001**	9.844(3.029 to 31.99)	**< 0.0001**
Normal	24(31.58)	21(84.00)		*	
High	7(9.21)	0(0.00)		13.16(0.7089 to 244.4)	**0.0331**
LYM	50.21 ± 0.9972	38.25 ± 0.8381	**< 0.0001**		
Low	0(0.00)	0(0.00)	**< 0.0001**		
Normal	8(10.53)	18(72.00)		*	
High	68(89.47)	7(28.00)		21.86(6.991 to 68.34)	**< 0.0001**
GRAN	41.47 ± 0.9825	53.64 ± 1.122	**< 0.0001**		
Low	61(80.26)	6(24.00)	**< 0.0001**	12.88(4.382 to 37.84)	**< 0.0001**
Normal	15(19.74)	19(76.00)		*	
High	0(0.00)	0(0.00)			

*Values are presented as frequency (percentages), Mean ± SD; [t]student sample t-test; [c]Chi-square test; *reference group.*

Table 3
Multivariate logistic regression of the lipid profile of the study participants.

Variable	Cases ($n=76$)	Control ($n=25$)	P- value	OR (95% CI)	P-value
Cholesterol	5.014 ± 0.1418	4.549 ± 0.1557	0.0801		
Low	8(10.5)	1(4.0)	**0.0004**	5.111(0.5988 to 43.62)	0.1031
Normal	36(47.4)	23(92.0)		*	
High	32(42.1)	1(4.0)		20.44(2.610 to 160.2)	**0.0002**
HDL-C	0.9449 ± 0.09506	1.820 ± 0.1325	**0.0001**		
Low	60(78.9)	1(4.0)	**0.0001**	96(12.00 to 767.9)	**< 0.0001**
Normal	15(19.7)	24(96.0)		*	
High	1(1.3)	0(0.0)		4.742(0.1813 to 124.0)	0.2148
TG	1.447 ± 0.1008	1.080 ± 0.08445	**0.0476**		
Low	1(1.3)	0(0.0)	**0.0165**	13.06(0.05327 to 34.41)	0.5052
Normal	56(73.3)	25(100)		*	
High	19(25.0)	0(0.0)		17.60(1.022 to 303.2)	**0.0052**
LDL-C	3.508 ± 0.1222	3.457 ± 0.1332	0.8222		
Low	15(19.7)	0(0.0)	**0.0098**	14.5(0.8341 to 252.2)	**0.011**
Normal	54(71.1)	25(100.0)		*	
High	7(9.2)	0(0.0)		7.018(0.3855 to 127.8)	0.0772
NON–HDL	4.152 ± 0.1394	3.590 ± 0.1226	**0.0289**		
Low	22(28.9)	5(20.0)	**0.0001**	6.8(1.983 to 23.32)	**0.0014**
Normal	11(14.5)	17(68.0)		*	
High	43(56.6)	3(12.0)		22.15(5.490 to 89.38)	**< 0.0001**
Coronary Risk	8.924 ± 0.4151	3.678 ± 0.1014	**0.0001**		
Low	1(1.3)	0(0.0)	**0.0001**	11.77(0.4275 to 324.0)	0.0549
Normal	6(7.9)	25(100)		*	
High	69(90.8)	0(0.0)		474.7(25.75 to 8750)	**< 0.0001**

*Values are presented as frequency (percentages), Mean ± SD; [t]student sample t-test; [c]Chi-square test; *reference group.*

Table 4
Hematological, lipid, liver and kidney profile of the exposed wood smoke participants stratified according to their duration of exposure.

Variable	<5years	5–10years	>10years	P-value
HB	11.36±1.180	11.54±1.532	11.34±1.178	0.8492
WBC	5.133±2.290	5.258±1.168	5.121±1.196	0.9313
PLT	209.8 ± 32.31	253.9 ± 73.95	227±64.02	0.1732
HCT	33.52±3.890	36.87±4.473	35.9 ± 3.594	0.1067
MCV	86.82±6.979	84.96±6.763	85.61±7.662	0.8238
MCH	29.24±4.117	25.76±2.688	27.15±4.056	0.0779
MCHC	33.82±3.140	30.5 ± 2.207	31.71±3.272	**0.0296**
LYM	50.43±7.919	49.47±8.291	50.45±9.127	0.9151
GRAN	41.03±7.187	42.28±8.009	41.24±9.129	0.8946
Cholesterol	4.521±0.9284	4.927±1.388	5.142±1.220	0.3659
HDL-C	0.8256±0.2622	0.8332±0.3802	1.011±1.008	0.6623
TG	1.53±0.9329	1.388±0.7085	1.454±0.9433	0.9215
LDL-C	3.039±0.8687	3.321±1.174	3.67±1.035	0.1795
Non-HDL	3.839±1.408	4.002±1.265	4.271±1.169	0.5171
Coronary Risk	8.486±3.850	8.489±2.495	9.178±3.976	0.7304
Urea	9.933±2.201	11.71±5.689	14.01±15.60	0.6086
Creatinine	95.4 ± 42.42	96.47±50.72	83.84±26.49	0.3578
AST	38±22.58	40.61±15.13	46.85±20.08	0.2931
ALT	20.59±17.33	13.81±9.853	18.12±18.28	0.5181

non-HDL cholesterol [OR=22.15(95%CI=5.490 to 89.38), $P<0.0001$] and low HDL cholesterol [OR=96(95%CI=12.00 to 767.9), $P<0.0001$] were significantly associated with exposure to wood smoke and are at increased risk of developing coronary heart disease [OR=474.7(95%CI=25.75 to 8750), $P<0.0001$].

Table 4 highlights the haematological, lipid, liver and kidney profile of the exposed wood smoke participants stratified according to their duration of exposure (years). The haematological profile indicated that low haemoglobin (11.34±1.178), white blood cells (5.121±1.196) and high lymphocyte counts (50.45±9.127) were associated with exposure of more than ten years. Exposure within five to ten years resulted in low MCV (84.96±6.763), MCH (25.76±2.688) and MCHC (30.5 ± 2.207).

The lipid profile indicated that participants with exposure of more than ten years have high cholesterol (5.142±1.220), LDL cholesterol (3.67±1.035), non-HDL (4.271±1.169) and are at increased risk of developing coronary heart disease (9.178±3.976). Exposure for less than five years resulted in low HDL cholesterol (0.8256±0.2622).

Increased serum urea (14.01±15.60) was associated with exposure exceeding ten years whereas increased serum creatinine (96.47±50.72) was associated with exposure within five to ten years. Duration of exposure exceeding ten years resulted

in increased AST levels (46.85±20.08) whiles increased ALT was associated with participants with less than five years exposure.

Discussion

The study sought to investigate the potential effect of persistent exposure to wood smoke on hematological and biochemical indices among regular wood burners in the Cape Coast Metropolis, Ghana. Our findings showed individuals exposed to wood smoke had basic educational level with majority being female. The little or no education from our study confirms a report by the United Nations Millennium Programme which states that most individuals who use wood as domestic fuel in Africa are rural dwellers with little education [11]. Similar findings have been reported across the globe in Shanghai [12], United States [13] and Europe [14]. The female preponderance reported in our study was similar to earlier studies which reported the effects of biomass smoke to be prevalent among women [15].

Our study showed that low HGB, MCV, MCH, MCHC, low granulocyte and high lymphocyte were significantly associated with exposure to wood smoke. Also, low HGB, WBC and the high lymphocyte counts were associated with exposure for more than ten years. The iron rich protein which oxygenates the tissues of the body has a higher affinity for carbon monoxide, a component of wood smoke, than oxygen. Carbon monoxide poisoning also affects myoglobin and mitochondrial cytochrome oxidase [16]. Carboxyhaemoglobin impacts adversely as vital organs such as the brain, heart and nervous system are deprived of oxygen supply. Consistent with our findings is a study by Rehfuess and Organization [17], who found that children who live in households cooking with traditional biomass fuels had low HGB levels [18].

Lymphocytes form part of a complex network of cells known as immune cells, which defend the body against foreign bodies such as bacteria, viruses and cancer cells. The high level of lymphocytes may typically be an indication of lymphocytosis which is associated with chronic infections, blood cancers and autoimmune diseases. Smoke inhalation has been found to stimulate the bone marrow to eject immature polymorphonuclear cells into circulation [3]. As revealed from our study, the low granulocyte levels may due to chronic exposure as the low white blood cells were associated with exposure exceeding ten years, buttressing a study which indicated that wood smoke exposure induces certain chronic disease states such as chronic obstructive pulmonary disease [19]. Also, the presence of the granulocytopaenia and the less affected lymphocyte production is an indication of aplastic anaemia.

Benzene, one of the pollutants emitted from wood smoke can be metabolized in the liver to a series of phenolic and open-ring structures, including hydroquinones, which can inhibit the maturation and amplification of bone marrow stem and blast cells. In addition, the metabolites of benzene alter the function of stromal cells in the bone marrow so that they cannot adequately support the growth and differentiation of hematopoietic cells [20]. In contrast to our findings it has been observed that smoke exposure related effects in mice and rats included increases in blood platelets and circulating WBC count [21].

High TC triglyceride, non-HDL cholesterol and low HDL cholesterol were significantly associated with exposure to wood smoke and are at increased risk of developing coronary heart disease. Moreover, significant low HDL cholesterol upon exposure to air pollution has been reported by Bell et al. [22] and in a study in Taiwan [23]. Particulate matter released from wood smoke can modify LDL cholesterol by accelerating its oxidation. Increased oxidized LDL cholesterol is thus a risk factor for cardiovascular diseases [4]. Also, particulate matter has been revealed to alter the structure and function of HDL cholesterol through oxidative stress and inflammation, producing a reduced dysfunctional HDL cholesterol with a decreased anti-inflammatory ability [24, 25] and hence culminating in coronary heart diseases. The change in lipid profile over time can thus be attributed to the fact that, those with longer years of exposure to wood smoke are likely to be exposed to more particulate matter, as has been reported by other studies [26,27].

Serum urea and creatinine levels were insignificantly higher among the exposed than the non-exposed. The elevated serum urea and serum creatinine were associated with daily exposure. Increased serum urea was associated with exposure exceeding ten years whereas increased serum creatinine was associated with exposure within five to ten years. Health-damaging pollutants such as nitrogen dioxide and sulphur dioxide released from wood smoke render hemoglobin useless for oxygen transport by causing its conversion to methaemoglobin or sulfhaemoglobin [28]. These compounds among others trigger vasoconstriction of arterioles [29] and hence inducing hypoxia. It has been proposed that chronic hypoxia is the final common pathway that leads to the development of end-stage renal failure [30]. Our study correlates with that of Kales et al. [31] which established no significant difference in urea and creatinine among people engaged in fire smoke activities.

The liver profile revealed statistically significant high AST levels among individuals exposed to wood smoke. ALT levels were insignificantly higher among the cases. Duration of exposure exceeding ten years resulted in increased AST levels whiles increased ALT was associated with participants with less than five years exposure. Serum aminotransferases (AST and ALT) are enzymes that act as sensitive indicators of hepatocellular damage. A studies by Kales et al., [31] and Al Malki [32] showed no significant variation in AST levels in response to fire smoke exposure. AST is raised in acute liver damage in response to liver injury, but it is also present in red cells, cardiac and skeletal muscle. It may be raised in response to shock and during exercise and therefore not specific to the liver [33]. In contrast to our study, Al Malki [32] revealed a statistically significant increase in ALT levels. Furthermore, Reed et al., [21] indicated that exposure related effects included decreases in serum ALT and changes in the liver cell. Even though our study provides some level of insight into the hematological and biochemical dynamics of people who are persistently exposed to wood smoke, our findings may be limited by the small number of samples size. Thus, we recommend a larger scale study which could bring more stability to the conclusions.

Conclusion

Exposure to wood smoke is associated with significant alterations in the haematological profile (low haemoglobin, low white blood cell, low granulocyte and high lymphocyte), and lipid profile (high cholesterol, high LDL cholesterol, high Non-HDL cholesterol, low HDL cholesterol and high risk of developing coronary heart diseases) with no apparent differences in the renal and liver function. Exposure to wood smoke exceeding ten years resulted in significant alterations in the lipid profile and serum urea levels.

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Conflict of Interest

The authors declare that there are no financial or non-financial competing interest

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