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Renal and Pulmonary Functions Associated with Polycyclic Aromatic Hydrocarbon Exposure Among Residents of a Gas Flaring Area in Bayelsa State, Nigeria

[1]Thomas, C.C., *[2]Eworo, R.E., [2]Nsonwu-Anyanwu, A.C. and [2]Usoro, C.A.O

[1]Federal Medical Centre, Yenagoa, Bayelsa State, Nigeria

[2]Department of Clinical Chemistry & Immunology, Faculty of Medical Laboratory Science, University of Calabar, Nigeria.

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ABSTRACT

Polycyclic aromatic hydrocarbons (PAHs), a major constituent of gas flaring, has been associated with adverse public health conditions such as cancer and renal dysfunctions. This study assessed PAH metabolites, renal function parameters (urea, creatinine, electrolytes, cystatin C, eGFR), and peak expiratory flow rate (PEFR) in 160 residents of a gas flaring area (GFA, n=80) and non-gas flaring area (NGFA, n=80). The PAH-metabolites were determined by GC-MS method; urea and creatinine by colorimetry; sodium, potassium, chloride, bicarbonate by ISE; cystatin C (CTC) by ELISA; estimated glomerular filtration rate (eGFR) computed and peak expiratory flow rate (PEFR) using peak flow meter. Data were analyzed using Student's t-test, ANOVA and Pearson's correlation at $p<0.05$. The GFA residents had significantly higher levels of urea, creatinine, sodium, chloride, potassium, cystatin C, 1-hydroxypyrene, 2-hydroxynaphthalene, 3-hydroxyphenanthrene, and 9-hydroxyfluorene, and significantly lower bicarbonate and eGFR compared to NGFA residents ($p<0.05$). No significant differences were observed in PEFR or 3-hydroxybenzo[a]pyrene. Age was strongly negatively correlated with eGFR in GFA residents ($r=-0.770$, $p<0.001$). These findings suggest that gas flaring exposure is associated with PAH accumulation and renal impairment, potentially increasing the risk of chronic kidney disease in affected populations.

Keywords: Gas Flaring, Polycyclic Aromatic Hydrocarbon, Renal-Pulmonary Functions

1.0 Introduction

Gas flaring and crude oil contamination in the Niger Delta region have become serious public health issues that are associated with detrimental consequences on health (Ephraim-Emmanuel *et al.,* 2022). Gas flaring describes the process of release of associated gases from the operations of refineries, oil wells, or hydro-carbon plants, this could be to dispose these gases or as a safety measure to lessen pressure (Faboya *et al.,* 2023). In areas with limited infrastructure, this gas is burned off either at the top of a large stack or from a pit in the ground, often characterized with the release of gases and heat (Giwa *et al.,* 2019). Devastating environmental effects include greenhouse effect, increase in temperature or thermal gradient, public health issues and changes in the ecosystem (Dung *et al*., 2008; Efe 2010;

Corresponding Author: **Raymond Ekong Eworo**
Department of Clinical Chemistry & Immunology, University of Calabar,
PMB 1115, Calabar, Cross River State, Nigeria.
Email: raymondeworo@yahoo.com; raymondeworo@gmail.com Phone.: +2348069363545

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Gobo *et al.,* 2010; Ejelonu *et al.,* 2011; Anomohanran *et al*., 2012).

Gas flaring is a primary source of greenhouse gases, volatile organic chemicals (VOCs), particulate matter (PM), black carbon and polycyclic aromatic hydrocarbons (PAHs) (Ephraim-Emmanuel *et al.,* 2022), that pollutes the air, soil and water. Polycyclic aromatic hydrocarbons are generated primarily during the incomplete combustion of organic materials (e.g. coal, oil, petrol). Many PAHs and their epoxides are highly toxic, mutagenic and/or carcinogenic to microorganisms as well as to higher forms of life including humans (Giwa *et al.,* 2019; Faboya *et al.,* 2023). Sixteen priority PAHs and 7 other polycyclic aromatic hydrocarbons have been characterized by the US Environmental Protection Agency as potentially carcinogenic (USEPA 2010; Thamaraiselvan *et al.,* 2015).

Exposure to polycyclic aromatic hydrocarbons have been shown to have adverse health effects on humans because of their potential for multiple organ and system toxicity, teratogenicity and carcinogenicity (Thamaraiselvan *et al.,* 2015). Human exposure routes include inhalation of smoke (tobacco, wood, coal, auto exhaust, etc.), eating foods cooked at high temperature or contaminated by smoke and through the therapeutic use of coal-tar. Polycyclic Aromatic Hydrocarbons are highly lipid soluble and thus are readily absorbed from the gastrointestinal tract of mammals (Ephraim-Emmanuel *et al.,* 2022). After absorption, PAHs are metabolically activated by specific enzymes, in two steps biotransformation pathway, (1) Phase I metabolizing enzymes such as cytochromes P450 (2) conjugation by Phase II metabolizing proteins to form glucuronides, increasing their water solubility (Gobo *et al.,* 2010). Urinary hydroxyl polycyclic aromatic hydrocarbons (OH-PAHs), have been used as biomarkers of exposure to PAHs, with 1-hydroxypyreneas being the most commonly used indicator (Jacob & Seidel 2002; Jacob *et al.,* 2015).

Multiple organ dysfunction including nephrotoxicity, pulmonary toxicity and adverse cardiovascular effects have also been associated with environmental exposure to PAHs (Gobo *et al.,* 2010; Sun *et al.,* 2021). Oxidative tissue damage mediated by reactive oxygen species (ROS) has been implicated as one of the pathological mechanisms of PAHs associated multiple organ toxicities (Moorthy *et al*., 2015; Nsonwu-Anyanwu *et al.,* 2018;). Perturbations in renal function parameters with significantly higher urea, creatinine, sodium and potassium have been reported in residents of a gas flaring area of Delta state, Nigeria compared to the controls (Abia *et al.,* 2019).

Environmental pollution with PAHs has become a serious public health issue plaguing the ecosystem with debilitating effects on human socio-economic developments mainly in most developing countries including Nigeria (Anomohanran *et al.,* 2012; Giwa *et al.,* 2019; Ephraim-Emmanuel *et al.,* 2022; Faboya *et al.,* 2023). Failure of the Nigerian environmental regulations in monitoring the unwholesome activities and poor environmental management practices by the oil industries have contributed to environmental contamination with PAHs (Gobo *et al.,* 2010; Giwa *et al.,* 2019). Residents of Gbarain in Yenagoa Local Government Area (LGA) of Bayelsa state witness daily non-stop flaring of gases from Shell Petroleum Development Company (SPDC). Dearth of scientific data on renal-pulmonary functions associated with PAHs exposure among residents of gas flaring area of southern Nigeria, informed this work. The biomarkers of PAH exposure, nephrotoxicity and pulmonary function associated with PAH exposure from gas flaring activities and oil exploration were determined in residents of a gas flaring area. We hypothesized that GFA residents would have higher PAH metabolites and worse renal and pulmonary function compared to NGFA residents.

2.0 Materials and Method

Study Design and location

This comparative cross-sectional study was carried out in Bayelsa State, Nigeria in Gbarain

area of Yenagoa Local Government Area (gas flaring area) which served as test area and Calabar municipal in Cross River State, Nigeria (non- gas flaring area) that served as control area between June 2021 to December 2021. Gbarain area in Yenagoa LGA of Bayelsa State witnesses daily non-stop flaring from Shell Petroleum Development Company (SPDC), while Calabar municipal in Cross River State has neither crude oil nor any gas flaring station.

Study location

Gbarain area is an Ijaw town in Ekpetiama Kingdom of Yenagoa LGA of Bayelsa State. It is located on the outskirts of Yenagoa, the state capital. Shell Petroleum Development Company has been operating in the area for many years. Calabar is the capital city of Cross River State. It occupies an area of 406 km^2 and has a population of 371,022 as at the 2006 census, and is made up of twenty-two wards.

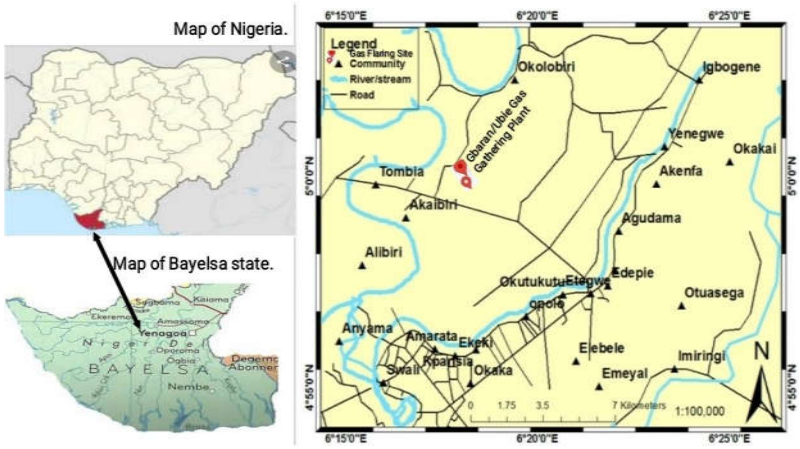

Figure 1: Map of Bayelsa state showing local government areas and location of the research area.

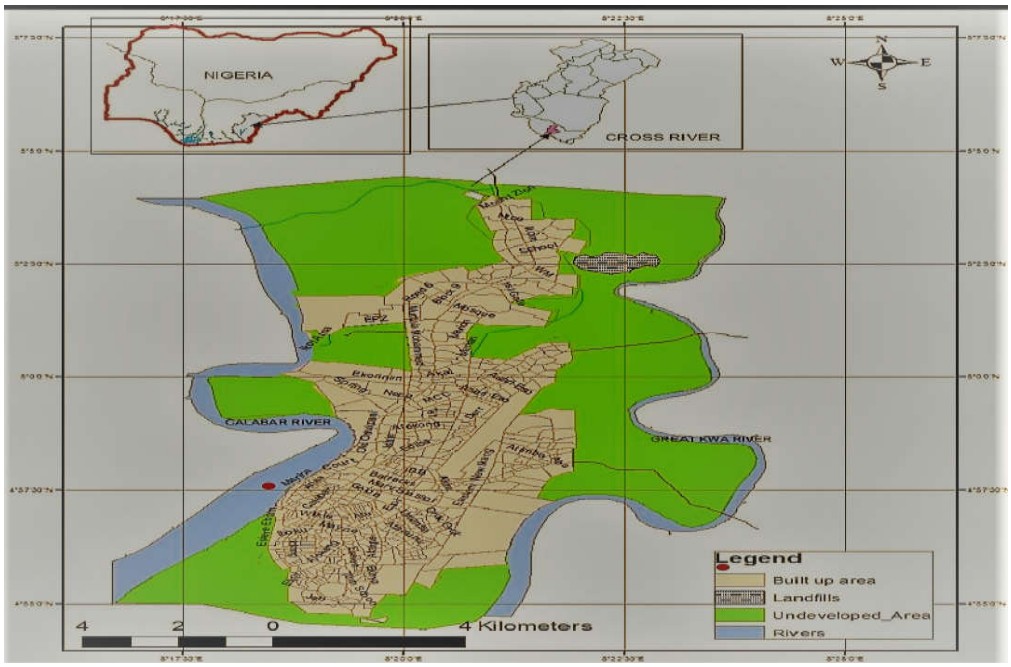

Figure 2: Map of Cross River state showing location of the research area.

The distance between the two sites is 338 kilometers, and other potential PAH sources in both areas are traffic exhaust, industrial emissions from coal gasification and incineration, domestic wood, cigarette smoke, char-boiled foods etc.

Selection of subjects

The participants in this study were made up of eighty (80) apparently healthy volunteers (males and females) aged 20 years and above who have stayed between five to twenty years or above in Gbarain, Bayelsa State (gas flaring area) as test subjects were randomly selected and eighty (80) apparently healthy age-matched volunteers who have stayed between five to twenty years or above in Calabar municipal in Cross River state, Nigeria (non- gas flaring area) as controls. Socio-demographic data were collected using well-structured questionnaires aimed at determining age, exposure risks, educational levels, work history, socio-economic status, years of residency in the communities, sources of water consumption, family and medical history.

Inclusion and exclusion criteria

Residents of Gbarain and Calabar Municipal, aged 20 years and above, who have lived in these areas for more than five years, with no history of recent blood transfusion and with no clear diagnosis of other known diseases, who were dependent on community boreholes and surface water sources as the major source of water for domestic use, that gave their consent. Those who have not lived in these areas up to five years, individuals with history of renal and pulmonary dysfunctions, history of alcohol abuse, metabolic disorders, chronic illness and chronic medications, and smokers were excluded from the study.

Sample size calculation

The sample size was calculated using the formula (Araoye, 2004).

$$N = \frac{Z^2P(1-P)}{d^2}$$

Where,

N = sample size,

Z = at the confidence level of 95%, Z value is 1.96.

P = expected prevalence. Prevalence for lung cancer is 3% (Awodele *et al.,* 2011).

and d = precision of 5%, d = 0.05),

$$N = \frac{1.96^2 \times 0.03 \times 0.97}{0.05^2}$$

N = 45.

Considering 10% attrition ratio,

N = 80.

Sample Collection

Whole blood samples (5 ml) were collected randomly after an overnight fast from all subjects of the study under aseptic conditions and dispensed into plain anticoagulant-free sample containers and labeled appropriately. The blood samples were allowed to clot and retract, and thereafter centrifuged at 1000 rpm for 10 min at room temperature to obtain serum which was then stored at -20 °C for analysis of renal function indices.

Urine collection and storage

Twenty (20) millilitres of early morning spot urine samples were collected randomly from all subjects of the study for the measurements of creatinine and PAH metabolites in sterile universal bottles and preserved by freezing at -20°C before analysis. Both blood and urine samples were collected during the day, transported in a cold chain from site of collection and stored in the laboratory at -20 °C before analysis.

Extraction of urine samples

Using liquid-liquid extraction (LLE) as described in APHA, (1998), 20ml of urine samples were extracted using a separatory funnel with 1ml sulfuric acid and 10ml portions of DCM, dried with anhydrous sodium sulfate and concentrate to about 2ml. The samples extracts were cleaned up in a chromatographic column (10 mm i.d x 20 cm)

packed with the slurry, prepared from 10g activated anhydrous Na2SO4. The samples were then eluted, concentrated and solvent exchanged to n-hexane. The concentrates were analytically determined with Gas Chromatography/Mass Spectrometry (GC/MS) instrument, using Certified Reference Materials (CRMs) as the Quality Control material to validate the whole analytical process.

Laboratory analysis

PAHs-metabolites [1-hydroxy pyrene (1-OHP), 2-hydroxy naphthalene (2-OHNAPH), 3-hydroxy phenanthrene (3-OHPHEN), 9-hydroxy flourene (9-OHFLU) and 3-hydroxy benzo{a}pyrene (3-OHBaP)] were determined by GC-MS method. Serum urea, creatinine and urinary creatinine were estimated using autoanalyzer (Selectra Pro S chemistry analyzer) with ELITech diagnostic reagent kits procured from ELITech Clinical Systems France, while the electrolytes (bicarbonate, chloride, sodium and potassium) were estimated using ion selective electrodes (ISE) (Schmidt *et al*., 2015; Zawada *et al*., 2009). Cystatin-C was estimated using ELISA kit procured from Iceberg Biotechnology Shanghai, China (Shlipak *et al*., 2022). Estimated glomerular filtration rate (eGFR) was calculated using Modified for Diet in Renal Disease (MDRD) equation (Mula-Abed *et al.,* 2012).), and peak expiratory flow rate (PEFR) performed using peak flow meter.

Statistical analysis

Results were presented as Mean±SD and data were analyzed using the statistical package for social sciences (SPSS version 22.0, IBM, USA). Data generated was test for normality by the Shapiro-Wilk test and Box and Whisker plots. Analysis of variance (ANOVA), followed with Turkey's HSD post hoc test and Pearson's correlation were used to determine significance of variations within and among group means and for comparison of multiple groups means and relationship between variables respectively. A probability value $p \leq 0.05$ was considered statistically significant.

3.0 Results

The comparison of renal function parameters [urea, creatinine, sodium, chloride, potassium, bicarbonate, estimated glomerular filtration rate (eGFR) and Cystatin-C], pulmonary function index [peak expiratory flow rate (PEFR)], and PAHs- hydroxyl metabolites [1-hydroxy pyrene (1-OHP), 2-hydroxy naphthalene (2-OHNAPH), 3-hydroxy phenanthrene (3-OHPHEN), 9-hydroxy flourene (9-OHFLU) and 3-hydroxy benzo{a}pyrene (3-OHBaP)] of GFA and NGFA residents was depicted in Table 1. Urea, creatinine, sodium, chloride, potassium, CTC, 1-hydroxypyrene, 2-hydroxynaphthalene, 3-hydroxyphenanthrene and 9-hydroxyflourene levels were significantly higher while bicarbonate and eGFR levels were significantly lower in GFA residents when compared with NGFA residents ($p<0.05$). No significant differences were observed in PEFR and 3-hydroxybenzo{a}pyrene level of the two groups ($p>0.05$).

The renal function, pulmonary function and PAHs-hydroxyl metabolites of GFA residents based on age periods of human life advances was shown in Table 2. Significant variations were observed in urea and eGFR among the studied groups ($p<0.05$), while no significant variations were observed in creatinine, electrolytes, CTC, PEFR and PAH hydroxyl metabolites with age among residents of GFA ($p>0.05$).

Figure 3 showed the correlation of eGFR against age in GFA residents. A significant strong negative correlation ($r = -0.770$, $p = 0.000$) was observed between age and eGFR in residents of GFA.

Table 1: Renal function, pulmonary function and PAHs-hydroxyl metabolites levels of residents in gas flaring area and control area.

Parameters	GFA n=80	NGFA n=80	Calt. T	p-value
Renal function				
Urea (mmol/l)	4.84±0.41	3.05±0.11	37.183	<0.001*
Creatinine (µmol/l)	112.51±12.29	85.50±1.13	19.576	<0.001*
Sodium (mmol/l)	141.66±2.00	135.50±1.06	24.438	<0.001*
Chloride (mmol/l)	105.39±3.65	95.88±1.66	21.206	<0.001*
Potassium (mmol/l)	4.63±0.17	3.37±0.22	40.276	<0.001*
Bicarbonate (mmol/l)	21.59±1.59	26.44±1.16	-22.148	<0.001*
CTC (ng/ml)	972.38±84.33	636.25±79.50	25.940	<0.001*
eGFR (ml/min)	67.79±5.93	75.00±5.78	-7.787	<0.001*
Pulmonary function				
PEFR (l/min)	450.00±14.50	452.88±12.24	-1.355	0.177
PAHs-OH Metabolites				
2-OHNaph (µmol/molCr)	0.33±0.34	0.14±0.12	4.642	<0.001*
9-OHFlou (µmol/molCr)	0.72±0.56	0.33±0.31	5.418	<0.001*
3-OHPhen (µmol/molCr)	2.59±20.10	0.80±0.53	7.414	<0.001*
1-OHP (µmol/molCr)	1.45±1.13	0.61±0.35	6.335	<0.001*
3-OHBaP (µmol/molCr)	0.66±0.44	0.56±0.45	1.436	0.153

Mean ± SD; * = significant at $p < 0.05$; CTC = Cystatin C; eGFR = Estimated Glomerular Filtration Rate; PEFR = Peak Expiratory Flow Rate; 2-OHNaph = 2-Hydroxy Naphthalene; 9-OHFlou = 9-Hydroxy Flourene; 3-OHPhen = 3-Hydroxy Phenanthrene; 1-OHP = 1-Hydroxy Pyrene; 3-OHBaP = 3-Hydroxy Benzo{a}Pyrene.

Table 2: Effect of age on renal function, pulmonary function and PAHs-hydroxyl metabolites in GFA residents

Parameters	Young Age (20-44 years) n= 25	Middle Age (45-60 years) n= 32	Elderly Age (> 60 years) n= 23	F-ratio	p-value
Renal function					
Urea (mmol/l)	5.09±0.26[a]	4.83±0.39	4.57±0.42	12.744	<0.001*
Creatinine (µmol/l)	115.84±11.93[b]	113.13±12.19	108.04±11.99	2.575	0.083
Sodium (mmol/l)	141.72±1.86	141.66±1.91	141.61±2.31	0.018	0.982
Chloride (mmol/l)	106.36±3.72	105.09±3.49	104.74±3.74	1.366	0.261
Potassium (mmol/l)	4.59±0.17	4.67±0.19	4.64±0.16	1.291	0.281
Bicarbonate (mmol/l)	21.12±1.48	21.75±1.80	21.87±1.32	1.638	0.201
CTC (ng/ml)	958.80±75.29	984.38±90.27	970.43±86.31	0.648	0.526
eGFR (ml/min)	72.44±3.70[c]	67.97±5.08	62.48±4.58	28.811	<0.001*
Pulmonary function					
PEFR (l/min)	451.60±15.46	450.31±11.77	447.83±17.04	0.412	0.664
PAHs-OH Metabolites					
2-OHNaph (µmol/molCr)	0.28±0.32	0.33±0.36	0.37±0.35	0.377	0.688
9-OHFlou (µmol/molCr)	0.83±0.56	0.65±0.55	0.70±0.59	0.770	0.467
3-OHPhen (µmol/molCr)	2.54±2.16	2.99±2.00	2.09±2.13	1.265	0.288
1-OHP (µmol/molCr)	1.52±1.21	1.41±1.11	1.44±1.13	0.072	0.931
3-OHBaP (µmol/molCr)	0.73±0.45	0.65±0.43	0.61±0.44	0.448	0.641

Mean ± SD; * = significant at $p < 0.05$; CTC = Cystatin C; eGFR = Estimated Glomerular Filtration Rate; PEFR = Peak Expiratory Flow Rate; 2-OHNaph/molCr = 2-HydroxyNaphthalene per mol creatinine; 9-OHFlou/molCr = 9-HydroxyFlourene per mol creatinine; 3-OHPhen/molCr = 3-HydroxyPhenanthrene per mol creatinine; 1-OHP/molCr = 1-HydroxyPyrene per mol creatinine; 3-OHBaP/molCr = 3-HydroxyBenzo{a}Pyrene per mol creatinine. a=significantly higher in 20-44yrs than in 45-60yrs and >60yrs, b=significantly higher in 45-60 yrs than in >60yrs, c=significantly higher in 20-44 yrs than in 45-60 yrs and >60yrs, significantly higher in 45-60 than in >60yrs

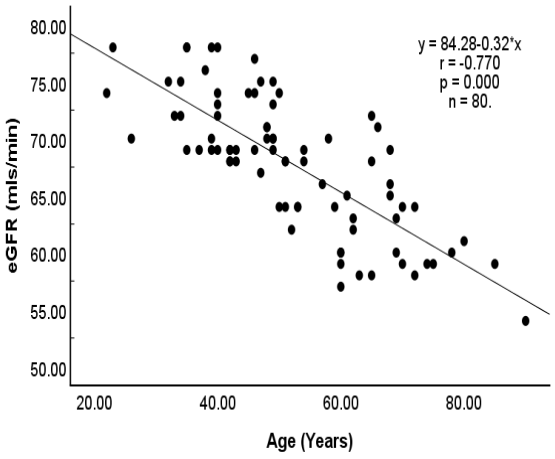

Figure 3: Correlation plot of eGFR against Age in GFA residents.

Discussion

Higher levels of renal function indices (urea, creatinine, sodium, chloride, potassium, cystatin-C) and PAHs-metabolites (1-hydroxypyrene, 2-hydroxynaphthalene, 3-hydroxyphenanthrene, 9-hydroxyflourene) observed in residents of GFA when compared with residents of NGFA could be attributed to chronic exposure to PAHs from gas flaring activities in the area, causing nephrotoxicity with associated high risk of renal dysfunction, this has been reported in previous studies (Gobo *et al.,* 2010; Sun *et al.,* 2021), which demonstrated both glomerular and tubular toxicity following chronic exposure to PAHs.

Higher levels of sodium observed in GFA residents compared to NGFA residents could be as a result of dehydration and profuse sweating, emanating from exposure to high temperature caused by gas flares, as gas flaring results in increased ambient temperature (Anomohanran 2012). It has been reported that about 45.8 billion kW of heat are discharged into the atmosphere of the Niger-Delta, due to the flaring of about 1.8 billion cubic feet of gas every day (Giwa *et al.,* 2019; Dung *et al.,* 2008), therefore, increased ambient temperature can cause persistent insensitive loss of body fluid, thus leading to chronic dehydration among residents of gas flared environment which can lead to reduced blood volume, rise in blood viscosity and blood pressure. Dehydration is further worsened by the poor water quality in the region as there is shortage of potable water to balance the effect of fluid loss from the body (Gobo *et al.,* 2010; Omoniyi and Ubale, 2015). This finding is in tandem with the report that chronic and persistent dehydration emanating from heat generated as a result of gas flaring led to reduced glomerular filtration rate and elevated levels of serum sodium ion level in gas flaring residents (Egwurugwu *et al.,* 2013).

High creatinine level could be as a result of renal failure, prostatic diseases, cancer of the bladder, drugs such as salicylate, acetate from patients with diabetic ketoacidosis (Odo *et al.,* 2019). Higher levels of creatinine observed in GFA residents compared to NGFA residents in this study agrees with the report of Egwurugwu *et al.,* 2013), who noted that an increase in creatinine level among individuals exposed to the toxic effect of gas flares in Imo state was as a result of chronic and persistent dehydration emanating from heat leading to reduced renal perfusion and reduced glomerular filtration rate.

There has been no reported case linking cystatin-C with gas flaring effect but, (Anderson, *et al.,* 2019), reported that cystatin-C increased in the serum of workers exposed to industrial chemicals, while (Ferguson *et al.,* 2015), noted that the concentration of cystatin-C is not affected by age, gender, race, protein intake and muscle mass unlike urea and creatinine. The higher cystatin-C levels of GFA residents in this study could be attributed to the toxic effect of gas flared constituents. These constituents serve as secondary metabolites that are generated during oxidation of benzene and benzene-like compounds by cytochrome P450 enzymes which are well known electrophiles that readily react with peptides, proteins, DNA and forms protein adducts, that have been identified in parallel with PAHs exposure (Nsonwu-Anyanwu *et al.,* 2018).

The significantly higher estimated glomerular filtration rate and peak expiratory flow rate of NGFA residents compared to those of the GFA residents may be as a result of their non-exposure to high temperature from gas flaring because, at high temperature, organic compounds are partially cracked to smaller unstable fragments (pyrolysis), mostly radicals that recombine to give relatively stable PAHs (pyrosynthesis) (Anomohanran, 2012), of which exposure to PAHs, heat stress and related dehydration can result in acute or chronic kidney failure (Anderson *et al.,* 2019; Ferguson *et al.,* 2015; Mula-Abed *et al.,* 2012; Odo *et al.,* 2019).

Significant variations observed in urea and eGFR among the studied groups ($p<0.05$) may be attributed to differences in the dose of PAHs the residents are exposed to, their genetic makeups and individual peculiarities. This is because, levels of PAHs and PAHs-metabolites have been shown to be influenced by racial, genetic, and individual peculiarities that influence the metabolism and polymorphism in metabolizing enzymes (Jacob and Seidel, 2002; Jacob *et al.,* 2015). Significantly higher urea, eGFR and creatinine levels in residents with younger age compared to those older ($p = 0.007$, $p = 0.000$, $p = 0.010$, $p = 0.028$), may be due to non-adaptiveness to higher exposure concentration of the toxicant in the residents with lower exposure duration years, which may have induced decreased activity of varied fractions of the antioxidant system not to neutralize the increased reactive oxygen species, in order to restore redox equilibrium between pro-oxidants and antioxidants (Moorthy *et al.,* 2015).

The significant strong negative correlation ($r = -0.770$, $p = 0.000$) observed between age and eGFR in residents of GFA may be due to progressive decreases in glomerular filtration rate and renal blood flow posed by advancement in age. Aging is characterized by progressive tubular dysfunction, decrease in sodium reabsorption, potassium excretion and urine concentrating capacity which potentially contributes to an increased susceptibility to acute kidney injury (Egwurugwu *et al.,* 2013; Odo *et al.,* 2019; Chen *et al.,* 2019). The findings of this study are in agreement with the study that reported a negative correlation between eGFR and age ($r = -0.386$, $p< 0.001$) on the influence of age on the effect of reduced renal function on outcomes in patients with coronary artery disease (Chen *et al*., 2019).

The significantly higher levels of PAH metabolites (1-hydroxypyrene, 2-hydroxynaphthalene, 3-hydroxyphenanthrene and 9-hydroxyflourene) observed in GFA residents when compared with NGFA residents may be due to exposure of the residents to PAHs accruing from gas flares. Increased exposure to PAHs entails higher systemic and tissue levels of PAHs leading to increased PAHs levels being presented for metabolism in the hepatocytes and consequently higher urinary excretion of PAHs-metabolites. Reactive PAHs-metabolites have been shown to cause oxidative stress and oxidative DNA damage in vital organs (Jacob and Seidel, 2002; Jacob *et al.,* 2015; Moorthy *et al.,* 2015). Positive association between total PAHs, 2-hydroxynaphthalene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 9-hydroxyfluorene and the risk of kidney stones after adjusting for confounding factors has been reported in National Health and Nutrition Examination Survey (NHANES) dose-response curves (Sun *et al.,* 2021).

Limitations of the study

Non estimation of other novel acute renal injury markers like kidney injury molecule-1, Interleukin-18 (IL-18), Neutrophil gelatinase-associated lipocalin (NGAL) etc.

Longitudinal studies with environmental PAH measurements, assessment of hydration status, and adjustment for confounders are needed to establish causality

Conclusion

This study demonstrates that residents of a gas flaring area in Bayelsa State, Nigeria, have significantly higher urinary PAH metabolites and evidence of renal function impairment compared

to residents of a non-gas-flaring area. These findings suggest that chronic exposure to PAHs from gas flaring may contribute to renal dysfunction. Longitudinal studies with environmental PAH measurements, assessment of hydration status, and adjustment for confounders are needed to establish causality. In the meantime, public health interventions including provision of clean water, regular renal function screening, and efforts to reduce gas flaring should be prioritized.

Conflict of interest

Authors declare no conflict of interest with regards to publication of this work.

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