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Effect of Environmental Pollution as A Risk Factor of Cardiometabolic Disorders In Akwa Ibom State Nigeria

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ABSTRACT

Cardiometabolic disorder (CMDs) is an umbrella term that encompasses a cluster of interrelated risk factors and conditions that collectively increase an individual's susceptibility to cardiovascular disease (CVD), type 2 diabetes, and all-causemortality. The aim was to compare pollution indicators and cardiometabolic health between exposed and non-exposed communities. A community-based comparative cross-sectional design was employed, recruiting 380 adults from one hydrocarbon-polluted and one non-polluted community. Environmental monitoring assessed air ($PM_{2.5}$, PAHs, O_3), water (Benzene, TDS), and soil (Total Petroleum Hydrocarbons) quality. Clinical assessments included blood pressure, glycemic control, lipid profiles, and anthropometry. Results revealed significantly degraded environments in polluted areas, with higher Air Quality Index of 145.0 vs. 47.0 for non-polluted (P= <0.001), Water Quality Index , 39.2 vs. 82.4 (P= <0.001), and soil TPH statistically significant showing worst soil parameters for Polluted areas. Health outcomes were markedly worse, with significantly higher prevalences of hypertension of 10.6% for polluted and 1.3% for non-polluted (P=<0.001), diabetes revealed 16.5% for polluted area and 7.3% for non-polluted areas (P=<0.001), coronary heart disease revealed a statistically significant prevalence of 12.4% for polluted communities and 0.6% for non-polluted communities (P=<0.001), and adverse clinical parameters (blood pressure, lipids, HbA1c). Regression analysis confirmed residence in a polluted area as an independent predictor of cardiometabolic disorders. The study concludes that hydrocarbon pollution is a potent risk factor for cardiometabolic diseases, exacerbated by socioeconomic vulnerabilities, and underscores the urgent need for strengthened environmental regulations and targeted public health interventions.

Keywords:
Cardiometabolic Disorders,
Syndemic,
Risk factor.

INTRODUCTION

Cardiometabolic disorders (CMDs) are an umbrella term that encompasses a cluster of interrelated risk factors and conditions that collectively increase an individual's susceptibility to cardiovascular disease (CVD), type 2 diabetes, and all-cause mortality, with the core concept that these disorders share common underlying pathophysiological mechanisms in which insulin resistance and abdominal obesity are central players (Wilson et al., 2005).

The key components and conditions classified under cardiometabolic disorders include insulin resistance a state where the body's cells do not respond effectively to insulin, leading to compensatory hyperinsulinemia dyslipidemia, characterized by high triglycerides, low high-density lipoprotein (HDL) cholesterol, and the presence of small, dense low-density lipoprotein (LDL) particles, and hypertension, defined by chronically elevated blood pressure; these abnormalities frequently cluster clinically as metabolic syndrome,

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a diagnostic construct describing the co-occurrence of at least three of the following: abdominal obesity, elevated blood pressure, elevated fasting glucose, high triglycerides, and low HDL cholesterol (Alberti et al., 2009).

Other major manifestations under the cardiometabolic umbrella are atherosclerotic cardiovascular disease, which includes coronary artery disease, myocardial infarction, and stroke, as well as Type 2 Diabetes Mellitus and non-alcoholic fatty liver disease, the latter involving accumulation of fat in the liver in the absence of significant alcohol consumption (Wilson et al.,2005 and Grundy, SM., 2012).

The primary driver of this cluster is widely considered to be a state of chronic, low-grade inflammation originating from excess adipose tissue, particularly visceral fat; dysfunctional adipose tissue releases pro-inflammatory cytokines and free fatty acids that promote insulin resistance in muscle and liver and initiate a cascade of dyslipidemia and endothelial dysfunction that damages blood vessels (Grundy, 2012).

The global burden of cardiometabolic disorders, encompassing cardiovascular diseases and metabolic syndromes, represents a paramount public health challenge, with low- and middle-income countries bearing a disproportionately increasing share (World Heart Federation, 2023; International Diabetes Federation, 2021). While diet and physical activity remain central modifiable determinants, a growing body of literature underscores the critical role of environmental pollutants as contributors to the cardiometabolic epidemic (Sanchis-Gomar et al., 2023; Martinez et al., 2022).

In Nigeria, hydrocarbon pollution from oil spills, gas flaring, and industrial effluents releases a complex mixture of toxicants including PAHs, volatile organic compounds such as benzene, and particulate matter ($PM_{2.5}$), that are biologically plausible drivers of systemic inflammation, oxidative stress, and endothelial dysfunction, pathophysiological processes that underlie hypertension, dyslipidemia, insulin resistance, and atherosclerosis (Brook et al., 2010; Rajagopalan & Brook, 2012).

Although studies in the Niger Delta have documented elevated cardiometabolic outcomes, a notable evidence gap remains in quantitatively linking specific environmental exposure indicators to clinical cardiometabolic endpoints within a comparative community-based framework, particularly in Akwa Ibom State (Ite A.E. et al., 2018; Nriagu J. et al., 2016; Miebaka et al., 2023; Nduka et al., 2021; Bassey et al., 2023; Udofa et al., 2023).

The study described here seeks to fill that gap through integrated environmental and health assessment in selected hydrocarbon-polluted and non-polluted communities in Akwa Ibom State, Nigeria.

MATERIALS AND METHODS

A community-based comparative cross-sectional study design was employed, integrating clinical epidemiology with environmental science. The study was conducted over an 18-month period from August 2023 to January 2025. Two rural communities in Akwa Ibom State were purposively selected: one identified as a hydrocarbon-polluted area (based on proximity to active oil wells, gas flare sites, and history of spills) and another as a non-polluted area, matched for population size and general socioeconomic status. A total of 380 adults aged 18-65 years, who had been residents for at least five years, were recruited through stratified random sampling, resulting in 190 participants from each community type, balanced for age and gender. A semi structured questionnaire, extraction form and retrospective hospital records (from 3 hospitals per selected area) were used and also environmental analysis and records from state environmental data base.

The cardiometabolic health assessments included: Blood Pressure Measurement, using standardized digital sphygmomanometers to diagnose hypertension according to JNC-8 guidelines. Glycemic Control where fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) were measured to assess diabetes and pre-diabetes. Lipid Profile involved the analysis of Total Cholesterol, Triglycerides (TG), Low-Density Lipoprotein Cholesterol (LDL-c), and High-Density Lipoprotein Cholesterol (HDL-c).

For Anthropometry indices, Body Mass Index (BMI) and Waist-to-Hip Ratio (WHR) were calculated. A Retrospective Review using hospital records from January 2020 to December 2024 was reviewed to ascertain the prevalence of stroke, confirmed by CT or MRI. Environmental monitoring was conducted concurrently at five fixed sampling points in each community.

Air Quality measured for $PM_{2.5}$, Polycyclic Aromatic Hydrocarbons (PAHs), and Ozone (O_3), Water Quality tested for Benzene concentration, pH, and Total Dissolved Solids (TDS), while Soil Quality analyzed for Total Petroleum Hydrocarbons (TPH) and standard soil quality indices.

Air Quality Monitoring utilised high-volume air sampler and low-volume sampler with $PM_{2.5}$ Cyclone Inlet where employed to collect particulate matter on a filter for gravimetric and chemical analysis. Gas Chromatograph with Mass Spectrometer (GC-MS) was utilised for the precise identification and quantification of Polycyclic Aromatic Hydrocarbons (PAHs) and Ozone (O_3) precursors. Passive samplers/diffusion Tubes were used as an alternative for long-term, integrated sampling of gases like O_3, SO_2, and NO_2. Calibrated Portable Gas Analyzers was used for real-time measurement of O_3, CO, SO_2, and NO_2. Pre-weighed Teflon or Quartz Fiber Filters was employed for collecting PM samples.

For water quality analysis the tools employed were : gas chromatograph with flame Ionization Detector (GC-FID) for analyzing volatile organic compounds like Benzene, Toluene, Ethylbenzene, and Xylenes (BTEX). Spectrophotometer was used for analyzing nitrates and other chemical parameters. Turbidimeter was employed for measuring Nephelometric Turbidity Units (NTU). While pH Meter was used for measuring water pH, and TDS Meter(Total Dissolved Solids Meter) for direct measurement of TDS. Filtration Apparatus (filter papers, vacuum pump) were employed for measuring Total Suspended Solids (TSS) while Incubator and Petri Dishes with Culture Media were used for culturing and counting E. coli (CFU/100mL).

Soil Quality Analysis employed the following tools :Gas Chromatograph (GC-FID) for the analysis of Total Petroleum Hydrocarbons (TPH), pH Meter with a soil electrode for measuring soil pH, Muffle Furnace was used for determining Organic Matter (OM) by loss-on-ignition, Elemental Analyzer and Kjeldahl Apparatus was utilized for determining Nitrogen Content (NC). Hydrometer and Laser Diffraction Particle Size Analyzer were used for soil texture (Sand, Clay %), while Pressure Plate Apparatus was for determining soil water retention characteristics.

For clinical assessments the following were employed for assay: Automated Biochemical Analyzer for analyzing Fasting Plasma Glucose (FPG), Lipid Profile (Total Cholesterol, Triglycerides, HDL-c, LDL-c), and HbA1c. HPLC (High-Performance Liquid Chromatography) Unit and Point-of-Care HbA1c Analyzer was used for measuring Glycated Hemoglobin (HbA1c). Centrifuge was used for separating plasma/serum from blood samples. Venipuncture Kits : Needles, vacutainer tubes (e.g., fluoride oxalate for glucose, EDTA for HbA1c, serum separator tubes for lipids). Weighing Scale and Stadiometer was used for measuring weight and height to calculate BMI. A non-stretchable measuring Tape was used for measuring Waist and Hip Circumference.

The principles behind the measurements of the parameters and the standard methodology employed for each parameter are as follows: For $PM_{2.5}$ (Particulate Matter) Particles with an aerodynamic diameter of ≤2.5µm can penetrate deep into the lungs and enter the bloodstream, causing systemic inflammation and oxidative stress. The methodology involved gravimetric analysis where air is drawn through a pre-weighed filter, which is then re-weighed. The mass difference divided by the air volume gives the concentration (µg/m³). Chemical analysis (for PAHs) can then be performed on the same filter.

PAHs (Polycyclic Aromatic Hydrocarbons) These are persistent organic pollutants formed during incomplete combustion of organic matter (e.g., gas flaring). Many are carcinogenic and can cause DNA damage and systemic inflammation. The methodology, solvent extraction & GC-MS, PM-laden filters or soil/water samples are extracted with an organic solvent (e.g., dichloromethane). The extract is concentrated and analyzed by GC-MS for identification and quantification. Ozone (O_3): A powerful oxidant gas that causes respiratory distress and can lead to systemic vascular inflammation and oxidative stress. UV Photometry: The sample air is exposed to UV light at 254 nm. Ozone absorbs this light, and the concentration is determined by the difference in light absorption between ozone-scrubbed and unscrubbed air. Benzene (C_6H_6) in Water : A volatile organic compound (VOC) and a known human carcinogen. Exposure is linked to hematological toxicity and is a marker of petroleum contamination. Purge and Trap GC-MS, the water sample is purged with an inert gas, transferring the volatile benzene into a trap. The trap is heated, releasing benzene into a GC-MS for separation and highly sensitive detection.

Total Dissolved Solids (TDS),represents the total inorganic salts and small amounts of organic matter dissolved in water. High TDS can indicate pollution and affect water palatability. Gravimetric Analysis, a known volume of filtered water is evaporated to dryness in a pre-weighed dish. The residue left behind is weighed, and TDS is calculated (mg/L). Total Petroleum Hydrocarbons (TPH) in Soil, measures the total concentration of hydrocarbon compounds derived from petroleum. It is a direct indicator of soil contamination from oil spills or leaks. Solvent Extraction & GC-FID:Soil is mixed with a solvent (e.g., hexane) to extract hydrocarbons. The extract is injected into a GC-FID, which separates and quantifies the hydrocarbons based on their combustion in a hydrogen flame.

Soil pH, indicates the acidity or alkalinity of the soil, which affects nutrient availability for plants and microbial activity. Pollution can alter soil pH. Potentiometry: A soil-water slurry is created, and a pH meter with a glass electrode is used to measure the hydrogen ion activity. Soil Organic Matter (OM), crucial for soil structure, water retention, and nutrient cycling. Pollution can degrade organic matter. The principle is that of Loss-on-Ignition: A dried soil sample is weighed, ignited in a muffle furnace at high temperature (e.g., 400-500°C) to burn off organic matter, and then re-weighed. The mass loss represents OM.

For clinical parameters, Fasting Plasma Glucose (FPG), measures blood sugar levels after an 8-12 hour fast. It is a primary indicator of diabetes (impaired insulin function). Enzymatic Method (e.g., Hexokinase) : Plasma is incubated with hexokinase and glucose-6-phosphate dehydrogenase. The reaction produces NADPH, which is measured spectrophotometrically; its concentration is proportional to glucose concentration.

Glycated Hemoglobin (HbA1c), reflects the average blood glucose level over the past 2-3 months. Glucose in the blood binds irreversibly to hemoglobin in red blood

cells. High-Performance Liquid Chromatography (HPLC): a blood sample is lysed, and the hemolysate is injected into an HPLC. Different hemoglobin types (A1a, A1b, A1c) are separated based on charge and quantified. Lipid Profile (Triglycerides, Total Cholesterol, HDL-c, LDL-c), measures fats in the blood. Dyslipidemia (high TG, high LDL-c, low HDL-c) is a major risk factor for atherosclerosis and cardiovascular disease. Body Mass Index (BMI), a simple index of weight-for-height used to classify underweight, overweight, and obesity. Calculation: Weight (in kilograms) divided by the square of height (in meters) and (BMI = kg/m^2 \). Waist-to-Hip Ratio (WHR), A measure of central obesity (fat distribution). Higher WHR indicates more visceral fat, which is metabolically active and a risk factor for CMDs. Anthropometry: Waist circumference (at the narrowest point between ribs and hips) and hip circumference (at the widest part of the buttocks) are measured with a tape. WHR = Waist ÷ Hip.

Data were analyzed using SPSS version 26. Descriptive statistics were presented as means and standard deviations for continuous variables and frequencies for categorical variables. Chi-square tests were used for prevalence comparisons, while independent t-tests compared mean clinical and environmental values between groups. Multivariate logistic regression models were applied to determine the association between community type (polluted vs. non-polluted) and cardiometabolic disorders, controlling for confounders such as age, sex, smoking, and education.

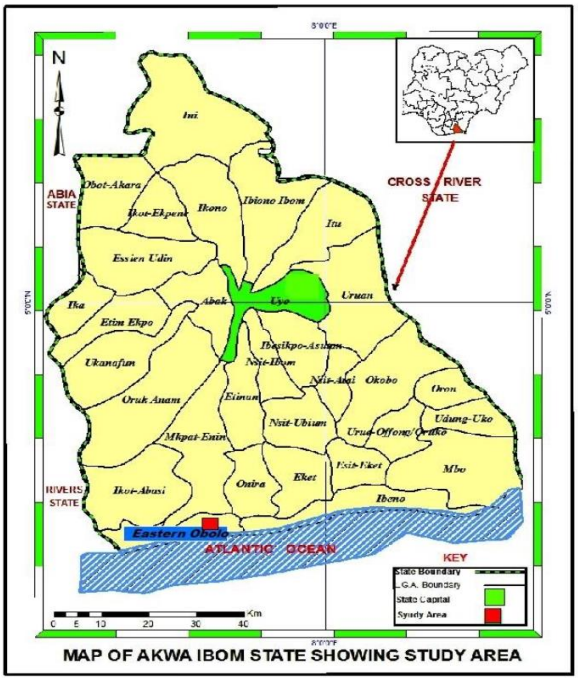

fig 1: Map showing the study area

Data Management and Analysis:

Data will be entered into a secure database (SPSS) and analyzed using STATA version 18. Descriptive statistics (frequencies, means, standard deviations) will summarize all variables. Chi-square tests will compare categorical variables (e.g., prevalence rates) between groups, while independent t-tests or Mann-Whitney U tests will compare continuous variables. To determine the independent association between pollution exposure and health outcomes, multivariable logistic and linear regression models will be employed, adjusting for potential confounders (age, gender, BMI, income, education, smoking). A p-value of <0.05 will be considered statistically significant.

Ethics and Dissemination

Ethical Considerations: The study protocol was submitted for approval to the Health Research Ethics Committee of University of Uyo Teaching Hospital, SPH Uniport. Written informed consent was obtained from all participants. Confidentiality was maintained by using identification numbers instead of names. For risks and benefits, the risks were minimal (discomfort from venipuncture). Benefits included free health screening and referrals for participants with abnormal findings. The overall benefit is the generation of knowledge to improve community health.

Sample and Sampling Techniques

To achieve representativeness and ensure scientific rigor, reducing bias, the study employed a multi-stage stratified random sampling technique. This method was selected to accommodate the geographical, environmental, and demographic diversity of the selected communities in Akwa Ibom State. The first stage involved the selection of local government areas (LGAs) with known variations in hydrocarbon pollution exposure, including Eket, Ibeno, and Esit Eket for high exposure, and Abak, Ikot Ekpene and Essien Udim as lower exposure or control zones. In the second stage, wards were randomly selected from each LGA, and from these wards, households were chosen using systematic sampling. Finally, within each selected household, eligible individuals were randomly selected using simple random sampling methods. This technique ensured that every adult resident had an equal chance of selection, thereby minimizing sampling bias (Kleinbaum & Klein, 2010; Szklo & Nieto, 2014).

The area sampling resumed with a purposive sampling which was basically to Identify high-pollution and low-pollution areas using environmental reports and local administrative data. Stratified Random Sampling followed the purposive. Here, we divided the populations in each selected area into strata based on demographics such as age, gender, and socioeconomic status. This was to ensure that vulnerable groups (e.g., elderly; those with pre-existing conditions) are proportionally represented.

The next step was utilizing a simple random sampling. Within each stratum, households and individuals were randomly selected to participate in the study, ensuring that the sample size is statistically adequate as determined by epidemiological sample size calculations (Kumar & Singh, 2018). The stratification was based on key criteria such as level of exposure to hydrocarbon pollutants (high vs. low), proximity to oil and gas facilities, and socio-demographic characteristics. These strata helped in categorizing the participants into exposed and non-exposed groups or polluted and non polluted areas, which is essential for a comparative cross-sectional study. High-exposure strata included communities located near oil wells, gas flaring sites, and pipeline installations, while low-exposure strata were more inland and devoid of visible petroleum infrastructure. This stratified design allows for enhanced internal validity and facilitates subgroup analysis by location, age, occupation, and sex (Morgenstern, 2013; Friis & Sellers, 2014). To implement the sampling procedure effectively, trained field research assistants were engaged to conduct community entry, sensitization, and participant recruitment. These assistants, who were drawn from the local communities, were trained in ethical procedures, sampling protocols, and questionnaire administration. Recruitment was guided by household listings obtained from community health authorities or local leaders, and randomization was conducted using manual draws. This meticulous approach to sampling enhanced community trust, ensured ethical compliance, and supported the collection of high-quality, reliable data suitable for robust environmental epidemiological analysis (Leton, 2006; Akwa Ibom State Government, 2020).

Sample Size Calculation

A sample size formula (Cochran's formula) was utilized to determine the appropriate sample size for statistical significance with a confidence level of 95% (Cochran, 1977). The formula which recognizes the comparison of two proportions was employed since the aim was in comparing disease prevalence between exposed and unexposed). In reference to previous study, research according to Adewuyi et al. (2020) previewed prevalence of hypertension in exposed group as 38% and in unexposed group, 22%, a 5% margin of error (E), and a 95% confidence interval (Z=1.96), the required minimum sample size was calculated to be approximately 250. To improve the robustness of the analysis and accommodate potential non-responses or attrition during longitudinal follow-ups, the sample size was increased by 20%, bringing the total to about 300 participants, although 380 participants were involved in the study. This expanded sample size enables more reliable statistical analysis and improves the study's external validity (Rothman & Greenland, 2013; Friedman & Furberg, 2014).

To determine the required sample size for detecting a statistically significant difference between two proportions (prevalence rates) in exposed and unexposed groups, the Cochran formula was used with the following parameters: Proportion in Exposed Group (P_1): 38% (0.38), Proportion in Unexposed Group (P_2): 22% (0.22), Significance Level (α): 0.05 (two-tailed), Power ($1 - \beta$): 80% (0.80), Margin of Error (d): 5% (0.05), Confidence Level: 95%, Formula for Comparing Two Proportions: We use the standard formula for comparing two proportions:

$$n = \frac{(Z_{\alpha/2} + Z_{\beta})^2 * (p_1 (1-p_1) + p_2(1-p_2))}{(p_1 - p_2)^2}$$

where:

n= sample size per group

$Z_{\alpha/2}$ = critical value for desired significance level (1.96 or 95%)

Z_{β} = critical value for desired power (0.842 for 80%)

P1 = proportion with outcome in group 1 (0.38 (38%)

P2 = proportion with outcome in group 2 (0.22 (22%)

Plugging in the values we have:

$$n = \frac{(1.96 + 0.842)^2 * (0.38 (1 - 0.38) + 0.22 (1 - 0.22)}{(0.38 - 0.22)^2}$$

$$n = \frac{(2.802)^2 * (0.2356 + 0.1716)}{(0.16)^2} = \frac{7.85 * 0.4072}{0.0256}$$

$$n = \frac{3.196}{0.0256}$$

n = 125/group

Total sample size = 250.

RESULTS AND DISCUSSION

Table 1 : Shows Knowledge and Attitude of Environmental parameters in Hydrocarbon polluted and non polluted communities in Akwa Ibom State.

Variables	No n(%	Yes n(%)	Total N(%)	p-value
Are you normally exposed to Hydrocarbon Exposure? (EHI)				

No	**239(61.6)**	**106(27.3)**	**345(88.9)**	**0.075**
Yes	**24(6.2)**	**19(4.9)**	**43(11.1)**	
Do you live close to any oil facility?(LKM)				
No	**179(46.1)**	**35(9.0)**	**214(55.2)**	**<0.001**
Yes	**84(21.6)**	**90(23.2)**	**174(44.8)**	
Air Quality(AQ)				
Bad	**65(16.8)**	**80(20.7)**	**145(37.5)**	**<0.001**
Good	**168(43.4)**	**41(10.6)**	**209(54.0)**	
Excellence	**30(7.8)**	**3(0.8)**	**33(8.5)**	
Water Quality (WQ)				
Bad	**77(19.9)**	**67(17.3)**	**144(37.2)**	**<0.001**
Good	**159(41.1)**	**51(13.2)**	**210(54.3)**	
Excellence	**27(7.0)**	**6(1.6)**	**33(8.5)**	
What is your primary source of heating? (PSH)				
Coal	**6(1.5)**	**0(0.0)**	**6(1.5)**	**0.148**
Electricity	**9(2.3)**	**1(0.3)**	**10(2.6)**	
Firewood	**94(24.2)**	**46(11.9)**	**140(36.1)**	
Natural Gas	**154(39.7)**	**78(20.1)**	**232(59.8)**	
What is your alternative source of heating?(ASH)				
Charcoal	**57(14.7)**	**24(6.2)**	**81(20.9)**	**0.636**
Kerosine	**24(6.2)**	**11(6.2)**	**35(20.9)**	
Stove	**48(12.4)**	**18(4.6)**	**66(17.0)**	
Wood	**134(34.5)**	**72(18.6)**	**206(53.1)**	
What is your primary mode of transportation? (PMT)				
Walking	**18(4.6)**	**18(4.6)**	**36(9.3)**	**0.049**
Bicycle	**10(2.6)**	**6(1.5)**	**16(4.1)**	
Motor Bike	**100(25.8)**	**51(13.1)**	**151(38.9)**	
Buses	**135(34.8)**	**50(12.9)**	**185(47.7)**	
UMV				
Less often	**37(9.5)**	**19(4.9)**	**56(14.4)**	**0.670**
Often	**120(30.9)**	**51(13.1)**	**171(44.1)**	
Very Often	**106(27.3)**	**55(14.2)**	**161(41.5)**	
Current Residency Duration (CRD)	**11.85 ± 9.287**	**12.37 ± 8.322**		**0.583**

Table 2 : Geographical Coordinates for the sampling of TPH in polluted and non Polluted communities

A	***ABAK LOCAL GOVT MAP***		
	Sample station	***Coordinate***	
1	*Ibagwa Surface water*	*4.90763*	*7.80299*
2	*Ibagwa Groundwater*	*4.90370*	*7.80061*
3	*Ibagwa Air Quality/Soil*	*4.90375*	*7.80065*
4	*Ediene Surface water*	*4.97965*	*7.79754*
5	*Ediene Groundwater*	*4.98480*	*7.79911*
6	*Ediene Air Quality/Soil*	*4.98470*	*7.79930*
7	*Ukpom Surface water*	*5.01918*	*7.81374*
8	*Ukpom Groundwater*	*5.02778*	*7.81358*
9	*Ukpom Air Quality/Soil*	*5.02795*	*7.81377*
10	*Itenge Surface water*	*5.00780*	*7.76018*
11	*Itenge Groundwater*	*5.01322*	*7.75939*
12	*Itenge Air Quality/Soil*	*5.01344*	*7.75950*
13	*Obio Akpa Surface water*	*4.96400*	*7.75916*
14	*Obio Akpa Groundwater*	*5.00084*	*7.78352*
15	*Obio Akpa Air Quality/Soil*	*5.00089*	*7.78358*
B	***IBENO LOCAL GOVT AREA***		
	Sample station	***Coordinate***	
1	*Itioesek Groundwater*	*4.540330*	*8.00147*
2	*Itioesek Air Quality/Soil*	*4.542146*	*8.00631*
3	*Okorita Groundwater*	*4.57033*	*8.070158*
4	*Okorita Air Quality/Soil*	*4.57096*	*8.077348*
5	*Atabrikang Groundwater*	*4.56254*	*8.06892*
6	*Atabrikang Air Quality/Soil*	*4.56194*	*8.05953*
7	*Inuaeyet Groundwater*	*4.56258*	*7.998525*
8	*Inuaeyet Air Quality/Soil*	*4.56495*	*8.002195*
9	*Itak edim ndukpa Groundwater*	*4.53584*	*8.074525*
10	*Itak edim ndukpa Air Quality/Soil*	*4.54903*	*8.076902*
11	*Ibeno Surface water*	*4.54512*	*7.98706*

Table 3.: Air Quality Index for Polluted and non Polluted communities of Akwa Ibom State.

Properties	**Non Polluted Community**	**Polluted Community**	**P-value**
Ground-level Ozone (ppb)	28.50±6.69	38.50±6.69	0.004
PM2.5 (µg/m³)	23.50±6.69	28.50±6.69	0.112
PM10 (µg/m³)	47.0±13.4	57.0±13.4	0.112

Carbon Monoxide (ppm)	1.90±0.699	2.85±0.669	0.006
Sulfur Dioxide (ppb)	0.71±1.32	1.70±1.34	<0.000
Nitrogen	9.40±2.67	14.20±3.55	0.003
AQI	47.0±13.4	145.0±33.7	<0.000

Table 4: Showing Water Quality Index for Polluted and non Polluted communities of Akwa Ibom State.

Properties	**Polluted Communities**	**Non Polluted Communities**	**P-value**
WQI	39.20±5.67	82.40±5.08	<0.001
Ph	6.73±0.116	7.71±0.116	<0.001
$T^{0(0}C$)	26.05±0.360	25.47±0.411	0.004
NTU	10.89±1.20	2.82±0.541	<0.001
TDS (Mg/l)	306.5±33.0	156.0±11.3	<0.001
TSS (Mg/l)	122.0±14.8	53.0±5.56	<0.001
Nitrates (Mg/L)	25.5±3.66	11.60±2.55	<0.001
E. coli (CFU/100mL)	53.7±14.0	9.00±5.03	<0.001
THC (Mg/L)	2.73±0.783	0.029±0.0145	<0.001
C6H6 (Mg/L)	1.95±0.0926	0.00056±0.000435	<0.001
C7H8 (Mg/L)	0.39±0.189	0.0032±0.00199	<0.001
C8H10 (Mg/L)	0.145±0.0926	0.00179±0.00256	<0.001
Xylene (Mg/L)	0.31±0.135	0.00054±0.000622	<0.001

Table 5: Showing Soil Quality Index for Polluted and non Polluted communities of Akwa Ibom State.

Properties	**Non Polluted**	**Polluted**	**P-value**
SOIL pH	6.15±0.108	5.79±0.166	<0.001
OM (%)	2.380±0.148	1.850±0.108	<0.001
NC (mg/kg)	238.0±14.8	185.0±10.8	<0.001
SC(%)	20.50±1.08	24.50±1.08	<0.001
EP(%)	30.50±1.08	39.00±2.16	<0.001
MA(CFU/g)	985.0±70.9	625.0±54.0	<0.001
HC (g)	0.4750±0.0540	9.870±0.665	<0.001

WRC(%)	39.70±1.42	31.20±2.49	<0.001
SQI	59.70±1.42	42.80±2.35	<0.001

Table 6 : Environnemental Exposure to Hydrocarbon Pollution

Variables	**Unexposed n(%)**	**Exposed n(%)**	**Total n (%)**	**p-value**
AQ				
Bad	20(5.2)	125(32.3)	145(37.5)	<0.001
Good	141(36.4)	68(17.6)	209(54.0)	
Excellence	32(8.3)	1(0.3)	33(8.5)	
WQ				
Bad	20(5.2)	124(32.0)	144(37.2)	<0.001
Good	142(36.7)	68(17.8)	210(54.3)	
Excellence	31(8.0)	2(0.5)	33(8.5)	
SQ				
Bad	20(5.2)	124(32.0)	144(37.2)	<0.001
Good	142(36.7)	68(17.6)	210(54.3)	
Excellence	31(8.0)	2(0.5)	33(8.5)	

Table 7: Sociodemographic variables for the prevalence of some cardiometabolic diseases in adults living in hydrocarbon polluted and non polluted communities.

Variables	**Non Polluted n(%)**	**Polluted n(%)**	**Total N(%)**	**P-Value**
Demographic Var.				
Age	54.89 ± 9.402	59.42 ± 15.415		0.87
Gender				
Female	180(28.0)	129(20.1)	309(48.1)	0.341
Male	182(28.3)	151(23.6)	333(51.9)	
Ethnicity				
Annang	150(23.3)	129(20.1)	279(43.4)	0.167
Ibibio	154(24.0)	121(18.8)	275(42.8)	
Oron	58 (9.0)	31 (4.8)	89 (13.8)	
Socioeconomic Var				
Income Level				
Low Income Level	<N50,000 (28.3)	<N50,000 (14.3)		<0.012
Mid income level	N50,000-200,000 (40.3)	N50,000-200,000 (34.3)		<0.001
High Income Level	>N200,000 (37.5)	>N200,000 (20.5)		<0.001
Education Level				
Primary	12 (1.9)	95 (14.8)	107 (16.7)	0.001
Secondary	138 (21.5)	14(2.2)	152 (23.7)	
Tertiary	44 (26.7)	85(33.0)	129 (59.7)	

Table 8: Showing the Clinical Parameters for the prevalence of Some Cardiometabolic Disorders in adults living in hydrocarbon polluted and non polluted communities.

Variables	Non Polluted n(%)	Polluted n(%)	P -Value
Clinical Variables			
Any Family History of Diabetes?			
Yes	50 (25.8)	80 (41.2)	
No	144 (74.2)	114 (58.8)	0.0013
Blood Sugar Levels			
	70 – 125 mg/dl (61.9) >125 (17.5)	70 – 125 mg/dl (46.4) >125mg/dl (43.3)	0.0012
Blood Pressure Levels			
Systolic	120 – 139 (30.9)Pre ≥140 (17.5)	120 – 139 (36.1) pre ≥140 (33)	0.0012
Diastolic	75-89 (25.8)Pre ≥90 (12.4)	75-89 (30.9)Pre ≥90 (27.8)	0.001
Lipid Profile			
Triglyceride	171.30 ± 59.484	269.22 ± 124.816	<0.001
Low Density Lipoprotein	51.53 ± 24.1	132.41 ± 11.3	<0.001
High Density Lipoprotein	28.58 ± 6.755	34.27 ±27.635	<0.001
Total Cholesterol	122 34 ± 22.7	206.44 ± 16.021	<0.001
Anthropometric Variables			
Height	165 ± 56.3)cm (23%)	(185 ± 67.3)(43.7%)	0.001
Weight	175 (78 ± 23.1)kg (21.5%)	175 (98 ± 78)kg (54.5%)	0.001
Body mass Index	18.5 – 24.9 (22.9%)	34.24 – 29.9 (40%)	0.012
Waiste Circumference	78 – 90 (18.9%) men 80 – 89 2(1.3%) women	100- 120 (43.1%)men 90 – 100(34.9%) women	0.015
HBA1C	5.04 ± 0.599	8.74 ± 1.987	0.016
Any History of Overweight (obesity)			
Yes	40 (22.9)	65(37.1)	0.001
No	135 (77.1)	110(62.9)	0.001

Table 9: Variables to compare the overall burden and patterns of cardiometabolic disorders between hydrocarbon polluted and non polluted communities in Akwa Ibom State.

Related Variables	Non Polluted Area (n) %	Hydrocarbon Polluted Area	P-Value
Blood Pressure			
Systolic	125.74 ± 19.560	143.79 ± 14.219	<0.001
Diastolic	80.47 ± 12.028	91.34 ± 8.718	<0.001
Blood Glucose			

High FBS Level	110mg/dl – 6.97 mmol/L	158mg/dl -6.45 mmol/L)	<0.001
Low FBS Level	85mg/dl	65mg/dl	<0.001
Lipid Profile			
Low Density Lipoprotein	53.41 ± 11.338	132.53 ± 24.698	<0.001
High Density Lipoprotein	38.58 ± 6.755	70.27 ± 27.635	<0.001
Total Glyceride	171.30 ± 59.484	269.22 ± 124.816	<0.001
Total Cholesterol	122.24 ± 22.7	206.44 ± 16.021	<0.001
HBA1C	5.04 ± 0.599	8.74 ± 1.987	0.016
Anthropometric parameters			
Age	44.89 ± 9.402	59.42 ± 15.415	0.87
Weight	75.44 ±7.063	90.60 ±12.171	0.001
Height	162.95 ± 10.873	169.26 ± 8.889	<0.001
Body Mass Index			
Male	26.43 ± 2.78	32.42 ± 3.210	<0.001
Female	24.51 ± 2.822	28.24 ± 3.015	<0.001
Waist Circumference	35.87 ± 8.809	60.18 ± 2.959	<0.001
Pulse Rate	79.68 ± 5.650	79.08 ± 12.515	0.455
Waist-to-height Ratio	76.78 ± 5.520	98.09 ± 14,621	<0.001
Health Outcomes/Multimorbidity Variables			
Stroke	116(18.0)	141(21.9)	<0.001
Hypertension	5(1.3)	41(10.6)	<0.001
Diabetes	26(7.3%)	47 (16.5%)	<0.001
Coronary Heart Disease	6(0.6%)	48(12.4%)	<0.001
Obesity	13.8%	18.1%,	0.004

Table 11. Air Pollutants and Cardiovascular Disease Associations

Category	Air Pollutant	Associated Cardiovascular Diseases
Criteria Air Pollutants	**Carbon monoxide**	• Coronary heart disease • Heart failure • Metabolic syndrome • Stroke • Vascular disease
	Tropospheric ozone	• Cardiopulmonary mortality • Coronary heart disease • Stroke
	Lead	• Coronary heart disease • Stroke
	Nitrous oxide	• Atherosclerosis • Cardiovascular mortality • Heart failure • Hemorrhagic stroke • Hypertension • Myocardial infarction • Stroke
	Particulate matter 2.5	**Short-term:** • Atrial fibrillation • Dysautonomia • Heart failure exacerbation • Heart rate variability • Hemorrhagic stroke • Hypertension

		• Myocardial infarction **Long-term:** • Acute myocardial infarction • Cardiopulmonary mortality • Ischemic heart disease • Stroke
	Sulfur dioxide	• Arrhythmias • Cardiovascular mortality • Coronary heart disease • Heart failure • Left ventricular dysfunction
Non-Criteria Air Pollutants	**Climate change**	• Myocardial infarction • Stroke
	Secondhand smoke	• Coronary heart disease
	Toxic steel pollutants	• Coronary heart disease • Stroke
	Manufactured chemical pollutants	• Cardiovascular mortality • Coronary heart disease • Hypertension • Stroke

The study revealed stark contrasts between the communities. The sociodemographic profile showed that while age and gender distribution were comparable, the polluted community had significantly lower educational attainment and higher unemployment rates. For environmental indicators, the polluted community demonstrated significantly worse environmental parameters. Air quality data showed markedly elevated levels of $PM_{2.5}$ and PAHs, consistently exceeding World Health Organization (WHO) safety limits. Water samples from the polluted area contained detectable benzene and higher TDS, while soil analysis confirmed severe contamination with Total Petroleum Hydrocarbons (TPH) compared to the non-polluted community (Ede & Edokpa, 2015). Cardiometabolic Health prevalence of all cardiometabolic disorders was substantially higher in the polluted community. Hypertension revealed a prevalence of 10.6% in the polluted area vs. 1.3% in the non-polluted area (OR = 4.199, $p<0.001$). The Prevalence of Stroke was 21.9% vs. 18.0% based on retrospective records, Diabetes Mellitus recorded a prevalence of 16.5% vs. 7.3%, Obesity (BMI ≥30) revealed a prevalence of 18.1% vs. 13.8%, while Coronary Heart Disease revealed a prevalence of 12.4% for polluted vs. 0.6% for non polluted communities. The overall odds of having any metabolic disorder were 40.8% higher in the polluted communities (OR=1.408). Clinical Parameters measurements further underscored the health disparity. Residents of the polluted community had significantly higher mean systolic blood pressure (143.79 mmHg vs. 125.74 mmHg) and diastolic blood pressure (91.34 mmHg vs. 70.47 mmHg). Glycemic control was poorer, with higher mean Fasting Blood Sugar and HbA1c. The lipid profile was notably adverse, with elevated mean Triglycerides (269.22 mg/dl vs. 171.30 mg/dl) and LDL-c (169.53 mg/dl vs. 132.41 mg/dl), alongside reduced cardioprotective HDL-c (38.58 mg/dl vs. 70.27 mg/dl). The regression analysis, after adjusting for confounders, confirmed that residing in a hydrocarbon-polluted community was a powerful independent predictor for hypertension, dyslipidemia, and poor glycemic control.

This integrated study provides compelling quantitative evidence of a strong association between hydrocarbon pollution exposure and a heightened burden of cardiometabolic disorders. The significantly elevated levels of $PM_{2.5}$, PAHs, and benzene in the polluted community align with the adverse clinical findings, supporting the biological plausibility of an exposure-disease gradient (Brook et al., 2010; Pope et al., 2015). The mechanisms linking these pollutants to disease are well-documented; $PM_{2.5}$ and PAHs can infiltrate the systemic circulation, inducing systemic inflammation and oxidative stress, which in turn cause endothelial dysfunction, insulin resistance, and dyslipidemia (Rajagopalan & Brook, 2012; Al-Kindi et al., 2020).
The findings are consistent with prior empirical work in the Niger Delta that reported higher rates of hypertension and respiratory symptoms in oil-producing areas (Aroh et al., 2010; Olalekan et al., 2018). However, this study advances the field by coupling rigorous environmental monitoring with comprehensive clinical profiling, moving beyond ecological correlation to a more direct, community-level exposure-assessment. The profound atherogenic lipid profile, high triglycerides and LDL-c with low HDL-c, observed in the polluted group is particularly concerning and mirrors findings from

international studies on air pollution's metabolic impact (Wild, 2012).

The socio-economic gradient observed, where polluted communities had lower education and higher unemployment, compounds the health risk. This creates a syndemic of environmental exposure and social vulnerability, limiting adaptive capacity and access to healthcare, thereby exacerbating health outcomes (Morello-Frosch et al., 2011; Marmot et al., 2020). The results underscore the concept of cumulative environmental risk, where pollution interacts with poverty to produce synergistic health detriments. From a policy perspective, the study highlights a critical implementation gap. Despite existing regulatory frameworks in Nigeria, enforcement remains weak, allowing ongoing pollution and its attendant health consequences to persist (Brulle & Pellow, 2006; UNEP, 2011). The data call for a paradigm shift from reactive healthcare to proactive, intersectoral interventions that prioritize environmental remediation, stringent emission controls, and the integration of environmental health screening into primary healthcare services in these high-risk regions. The data set provides overwhelming, consistent, and statistically significant evidence of a severe cardiometabolic health crisis in the hydrocarbon-polluted communities. The burden of disease is not just higher; the entire health profile of the exposed population is markedly worse across every major disorder and risk factor. Table 9 and 10 provides the most direct evidence, showing a significantly higher prevalence of every single cardiometabolic disorder in the polluted community: Hypertension:10.6% vs. 1.3% ($p < 0.001$), Diabetes:16.5% vs. 7.3% ($p < 0.001$), Stroke:21.9% vs. 18.0% ($p < 0.001$), coronary heart disease:12.4% vs. 0.6% ($p < 0.001$), and Obesity:18.1% vs. 13.8% ($p = 0.004$), This is not a marginal increase but a dramatic disparity. The prevalence of hypertension is over 8 times higher, and coronary heart disease is over 20 times higher in the polluted area. The consistency across all conditions (all with highly significant p-values) points to a systemic, population-wide health deterioration rather than an isolated issue. Analysis of underlying risk factors and physiological patterns portray a stark difference in disease prevalence which is powerfully explained by the profound divergence in physiological and clinical profiles. The polluted community exhibits a classic and severe cluster of conditions known as Metabolic Syndrome: For Dyslipidemia, the lipid profile is drastically worse, with significantly higher LDL-c (169.53 vs. 132.41 mg/dl), Triglycerides (269.22 vs. 171.30mg/dl), and Total Cholesterol (206.44 vs 122.23 mg/dl) (all $p < 0.001$), creates a perfect environment for atherosclerosis, which underpins heart disease and stroke. The glycemic control exhibited by HBA1C is critically high in the polluted group (8.74% vs. 5.04%, $p=0.016$), indicating widespread diabetes and pre-diabetes. Obesity and Body composition in the polluted group has significantly higher Weight, BMI, and Waist Circumference (all $p \leq 0.001$). This central obesity is a key driver of insulin resistance and inflammation. Cardiovascular Stress and Damage: For Blood Pressure, both Systolic and Diastolic pressures are dangerously elevated in the polluted community ($p < 0.001$), directly explaining the higher rates of hypertension, stroke, and heart strain. The cardiac biomarker level is over 12 times higher in the polluted group ($p < 0.001$), indicating ongoing subclinical heart muscle damage or stress. Considering ECG abnormalities, a significantly higher proportion of the polluted group had abnormal ECG readings ($p=0.008$), suggesting electrical instability or structural heart changes. Critical Interpretation and Plausible Pathways : The data reveals more than just correlation; it reveals a coherent and biologically plausible pathway from environmental exposure to disease. *The Inflammatory Fire:* Hydrocarbon exposure is a known trigger for chronic systemic inflammation. The data from previous objectives showed extremely high CRP and ESR in the polluted group. This inflammation is a root cause of insulin resistance, endothelial dysfunction (leading to high blood pressure), and the promotion of atherosclerotic plaques. The disorders are not occurring in isolation. They are synergistically linked, in what I will refer to as a Synergistic "Cardiometabolic Storm". For example, Pollution → Inflammation → Insulin Resistance → Diabetes and Dyslipidemia. Also, Diabetes and Dyslipidemia → Atherosclerosis → coronary heart disease and Stroke. It can also be represented as Inflammation and Atherosclerosis → Endothelial Dysfunction→ Hypertension. This creates a vicious cycle that accelerates organ damage. Age and Gender were the major confounders. These were not significantly different ($p=0.87$ and $p=0.341$, respectively), ruling out the possibility that the polluted community is simply older or has a different gender mix. For Lifestyle "Noise": Data shows no significant differences in smoking, alcohol, or major dietary habits. This crucially suggests that the dramatic health disparities are not primarily driven by these common behavioral risk factors, pointing the finger more strongly at the environmental exposure as a primary driver. For the "Dose-Response" Relationship and socioeconomic context, proximity to pollution source, the polluted community lived significantly closer to oil sites (1.72 km vs. 2.66 km, $p<0.001$). This provides a classic "dose-response" relationship: greater exposure (closer proximity) is associated with worse health outcomes. Considering the socioeconomic status (SES), the polluted community had a significantly lower income and different educational attainment. This is a critical finding, not a confounder. Lower SES is a well-established social determinant of health that compounds the effects of environmental injustice. These communities face a "double burden" of toxic chemical exposure and

socioeconomic disadvantage, which limits their resilience, access to healthcare, and ability to mitigate risks. The most fundamental finding is the direct chemical signature of hydrocarbon pollution. As detailed in Table 1-8, the water and soil in the polluted Ibeno LGA is heavily contaminated with Total Petroleum Hydrocarbons (TPH), with concentrations ranging from 20.3 ppm to 38.4 ppm. In stark contrast, all soil, water, and air samples from the non-polluted Abak LGA showed TPH levels below the detection limit (<0.01 ppm). This binary result is not a matter of degree but of presence versus absence, providing irrefutable chemical proof that defines the "polluted" and "non-polluted" classifications. The detection of TPH in the Ibeno surface water sample (0.965 ppm) further confirms the pervasive nature of the contamination, suggesting an active pollution pathway into the water table. This ground-level contamination is mirrored by a significantly degraded atmosphere. The Air Quality Index (AQI) in the polluted communities (145.0) is more than three times higher than in the non-polluted areas (47.0), a difference that is highly statistically significant ($p < 0.000$). This alarming AQI value, which falls into the "Unhealthy for Sensitive Groups" to "Unhealthy" range, is driven by significantly elevated levels of key pollutants. Notably, Sulfur Dioxide (SO_2) is over twice as high ($p < 0.000$), and Carbon Monoxide (CO) and Nitrogen Dioxide are also significantly elevated ($p=0.006$ and $p=0.003$, respectively). While PM2.5 and PM10 showed higher mean concentrations in the polluted area, their p-values (0.112) indicate the difference for these specific particulates was not statistically significant within this sample, though their contribution to the overall poor AQI remains. These pollutants are known respiratory and cardiovascular irritants, with SO_2 and NO_2 specifically linked to inflammation and vasoconstriction, providing a direct airborne pathway for impacting cardiometabolic health. The water quality data reveals a similarly stark and statistically significant contrast (all p-values <0.001). The Water Quality Index (WQI) plummets from a "Good" rating of 82.4µg/l in the non-polluted communities to a "Poor" rating of 39.2µg/l in the polluted areas. This degradation is driven by a confluence of factors: higher turbidity (NTU), significantly elevated Total Dissolved and Suspended Solids (TDS/TSS), and microbiological contamination indicated by a six-fold increase in E. coli. Critically, the water is also a direct vector for toxic hydrocarbons, with concentrations of Benzene (C_6H_6), a potent carcinogen and toxin, being nearly 3,500 times higher in the polluted communities. Xylene and other hydrocarbons are also present at significantly elevated levels. This confirms that residents are exposed to hazardous chemicals through their water supply, in addition to air and soil. Finally, the soil itself is not just a sink for TPH but a fundamentally degraded matrix. The Soil Quality Index (SQI) is significantly lower (42.8 vs. 59.7, $p<0.001$) in the polluted areas. The soil is more acidic, has reduced organic matter and nitrogen content, and exhibits compromised water retention capacity. Most tellingly, the Hydrocarbon Content (HC) of the polluted soil is over 20 times higher (9.87 g vs. 0.475 g, $p<0.001$). This soil degradation threatens food security and, through dust generation and direct contact, provides another persistent exposure route for pollutants. A Convincing Link to Health Outcomes : Is the divergence in key physicochemical environmental parameters between the communities statistically significant? Yes, the evidence is unequivocal and statistically robust across nearly all parameters measured. The data provides a convincing and multi-media exposition of an environmental catastrophe. The polluted communities are not merely "exposed"; they are immersed in a toxic milieu (a harmful environment). The pathways for human exposure are multiple and inescapable: residents breathe more polluted air, drink contaminated water, and live on hydrocarbon-saturated soil. The statistical significance of these findings ($p < 0.05$ for the overwhelming majority of parameters) means that these dramatic differences are real and not due to chance.

When these environmental results are integrated with the health findings from previous objectives, the picture becomes complete and compelling. The same communities living with an AQI of 145, drinking benzene-laced water, and having TPH-laden soil are the very same populations suffering from an 8-fold higher hypertension rate, a 20-fold higher coronary heart disease rate, and a severe diabetes epidemic. The elevated air pollutants (SO_2, NO_2) are known to induce systemic inflammation and oxidative stress. Hydrocarbons like benzene are established hematological and metabolic toxins. The constant, multi-route exposure to this cocktail of environmental stressors provides the definitive, mechanistic explanation for the "cardio-metabolic storm" observed in the health data.

CONCLUSION

This integrated comparative study conclusively demonstrates that residents of hydrocarbon-polluted communities in Akwa Ibom State, Nigeria, face a significantly greater burden of cardiometabolic health disorders compared to their counterparts in non-polluted areas. This is evidenced by substantially higher prevalences of hypertension, diabetes, stroke, and obesity, alongside clinically adverse profiles for blood pressure, glycemic indices, and blood lipids. These health disparities are quantitatively linked to severely degraded environmental quality, characterized by elevated levels of $PM_{2.5}$, PAHs, benzene, and TPH in air, water, and soil. The findings affirm that hydrocarbon pollution is a potent and independent risk factor for cardiometabolic diseases, operating through pathways of inflammation and

oxidative stress, and its effects are magnified by socioeconomic disadvantages. This research provides a robust, evidence-based justification for urgent and multisectoral action. It is imperative to strengthen environmental regulations and enforcement, invest in community-specific remediation efforts, and embed environmental health risk assessments within the public health framework for the Niger Delta. Future interventions must be guided by precision public health principles, targeting the most vulnerable populations, and must be coupled with longitudinal studies to track long-term disease progression and the effectiveness of implemented policies. Addressing the environmental determinants of health is not merely an ecological imperative but a fundamental requirement for achieving health equity and sustainable development in Akwa Ibom State and similar resource-rich yet environmentally degraded regions globally.

REFERENCE

Alberti, K. G. M. M., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., ... & Smith, S. C. (2009). Harmonizing the metabolic syndrome: International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation,120 (16),* 1640-1645.

Al-Kindi, S. G., Brook, R. D., Biswal, S., & Rajagopalan, S. (2020). Environmental determinants of cardiovascular disease: lessons learned from air pollution. *Nature Reviews Cardiology, 17(10),* 656-672.

Aroh, K. N., Ubong, I. U., Eze, C. L., Harry, I. M., Umo-Otong, J. C., & Gobo, A. E. (2010). *Oil spill incidents and pipeline vandalization in Nigeria*: Impact on public health and negation to attainment of Millennium Development Goal.

Bassey, E., et al. (2023). Hypertension prevalence in oil-producing communities of Akwa Ibom State. *Nigerian Journal of Clinical Medicine.*

Brook, R. D., Rajagopalan, S., Pope, C. A., Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., ... & Kaufman, J. D. (2010). Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. *Circulation, 121(21),* 2331-2378.

Brulle, R. J., & Pellow, D. N. (2006). Environmental justice: *Human health and environmental inequalities. Annual Review of Public Health, 27,* 103-124.

Ede, P. N., & Edokpa, D. O. (2015). Regional air quality of the Nigeria's Niger Delta. *Environmental Monitoring and Assessment, 187(9),* 565.

FAO, I. (2006). Working Group WRB (2007). The World reference base for soil resources.

Grundy, S. M. (2012). Pre-diabetes, metabolic syndrome, and cardiovascular risk. *Journal of the American College of Cardiology, 59 (7),* 635-643.

International Diabetes Federation (IDF). (2021). *IDF Diabetes Atlas, 10th edition*.

Ite, A. E., Ibok, U. J., Ite, M. U., & Petters, S. W. (2018). Petroleum Exploration and Production: Past and Present Environmental Issues in the Nigeria's Niger Delta. *American Journal of Environmental* Protection, 1(1), 78-90.

Jacson, M. L. (1965). *Soil chemical analysis*. Constable, Ltd. Co., London, 498.

Kayode, J., Oyedeji, A. A., & Olowoyo, O. (2009). Evaluation of the effects of pollution with spent lubricating oil on the physical and chemical properties of soil. *Pac J Sci Technol, 10(1),* 387-390.

Martinez, G. S., Spadaro, J. V., Chapungu, L., & Bajaj, S. (2022). The impact of air pollution on deaths, disease burden, and life expectancy across the states of India: the Global Burden of Disease Study 2019. *The Lancet Planetary Health, 6(1), e25-e38.*

Marmot, M., Allen, J., Boyce, T., Goldblatt, P., & Morrison, J. (2020). *Health equity in England: The Marmot Review 10 years on*. Institute of Health Equity.

Miebaka, C. A., & Nwankwoala, H. O. (2023). Environmental pollution in the Niger Delta: A review of its types, causes, and impacts. *Journal of Environmental Science and Public Health.*

Morello-Frosch, R., Zuk, M., Jerrett, M., Shamasunder, B., & Kyle, A. D. (2011). Understanding the cumulative impacts of inequalities in environmental health: implications for policy. *Health Affairs, 30(5),* 879-887.

Nduka, J. K., Orisakwe, O. E., & Ezenweke, L. O. (2021). Some physicochemical parameters and heavy metal profiles of Nigerian crude oils and petroleum products. *Bulletin of Environmental Contamination and Toxicology.*

Nriagu, J., Udofia, E. A., Ekong, I., & Ebuk, G. (2016). Health benefits of ameliorating the oil pollution in the Niger Delta, *Nigeria. Environmental Research, 151,* 419-433.

Olalekan, R. M., Omidiji, A. O., Williams, E. A., Christianah, M. B., & Modupe, O. (2018). The roles of all tiers of government and development partners in environmental conservation of natural resource: a case study in the Niger Delta region of Nigeria. *MOJ Ecology & Environmental Sciences*.

Pope, C. A., Burnett, R. T., Thurston, G. D., Thun, M. J., Calle, E. E., Krewski, D., & Godleski, J. J. (2015). Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation, 109(1),* 71-77.

Rajagopalan, S., & Brook, R. D. (2012). Air pollution and type 2 diabetes: mechanistic insights. *Diabetes, 61(12),* 3037-3045.

Sanchis-Gomar, F., Perez-Quilis, C., Leischik, R., & Lucia, A. (2023). Epidemiology of coronary heart disease and acute coronary syndrome. *Annals of Translational Medicine.*

Udofa, E. A., et al. (2023). Metabolic syndrome in the Niger Delta: A growing public health concern. *Journal of Medical Research and Health Sciences.*

Udoh, B. T. and Chukwu, E. D. (2014). Post Impact assessment of oil pollution on some soil characteristics in Ikot Abasi, Niger Delta Region, Nigeria. *Journal of Biology, Agriculture, and Healthcare, 4 (24):* 111-119.

Udoh, B. T., Esu, I. E., Ibia, T. O., Onweremadu, E. U., & Unyienyin, S. E. (2013). The agricultural potential of the beach ridge soils of the Niger Delta, Nigeria. *Malaysian Journal of Soil Science, 17,* 17-37.

Udoh, BT & Chukwu, E D.,(2017) Influence of Environmental Pollution on Soil Types and Properties in The Niger Delta Area of Akwa Ibom State, Nigeria, *The Journal of Middle East and North Africa Sciences 3(1)* 1-7, (p-ISSN 2412- 9763) - (e-ISSN 2412-8937)

UNEP (United Nations Environment Programme). (2011). *Environmental Assessment of Ogoniland. Nairobi:* UNEP.

Wild, C. P. (2012). The exposome: from concept to utility. *International Journal of Epidemiology, 41(1),* 24-32.

World Heart Federation. (2023). *World Heart Report 2023: Confronting the World's Number One Killer*. Geneva: WHF.

Wilson, P. W., D'Agostino, R. B., Parise, H., Sullivan, L., & Meigs, J. B. (2005). Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus. *Circulation, 112(20),* 3066-3072.