



## A Retrospective Hospital-Based Study on the Association Between Environmental Hydrocarbon Exposure and Stroke Incidence in Akwa Ibom State, Nigeria.

Essiet, Akanimo G.<sup>1\*</sup>, Gordon, Anietie A.<sup>2</sup>, Elgado, Hajara.<sup>3</sup>, Ime, Aniema-Abasi W.<sup>4</sup>, Chieke, Okechukwu F.<sup>5</sup>, Essien, Atim S.<sup>6</sup>, Inyang, Blessing I.<sup>7</sup>, Udim, Nkereuwem A.<sup>8</sup> & Umoren, Augustine U.<sup>9</sup>

<sup>1</sup>Department of Epidemiology, School of Public Health, University of Port Harcourt, Nigeria.

<sup>2,6</sup>Department of Public Health, Federal College of Medical Laboratory Science and Technology Jos, Nigeria.

<sup>3,4,5,7,8&9</sup>Department of Basic Science, Federal College of Medical Laboratory Science and Technology

\*Corresponding Author Email: [essiet\\_akanimo@uniport.edu.ng](mailto:essiet_akanimo@uniport.edu.ng)

### ABSTRACT

Akwa Ibom State in Nigeria's Niger Delta has experienced hydrocarbon pollution for over sixty years due to gas flaring, oil spills, and pipeline vandalism. This retrospective study analyzed 1,000 adults with CT or MRI-confirmed strokes from polluted and non-polluted communities. Stroke prevalence was higher in polluted areas (21.9% vs. 18.0%), with residents showing increased cardiometabolic risk factors, including higher blood pressure, blood sugar, triglycerides, and cardiac biomarkers, despite lower LDL cholesterol and BMI. Polluted area residents were generally older, less educated, and more likely to be widowed. Ischemic strokes were more common and functional outcomes were poorer in polluted communities, linking hydrocarbon exposure to elevated stroke incidence and worse health profiles. These results underscore the need for strengthened public health measures and improved environmental regulations.

### Keywords:

Hydrocarbon pollution,  
Stroke incidence,  
Cardiometabolic risk,  
Environmental exposure

### INTRODUCTION

The Niger Delta region of Nigeria, which encompasses Akwa Ibom State, has served as a hub for hydrocarbon exploration and extraction for more than sixty years. (Babatunde, A. & Olateju, A. (2015).

While these industries have provided significant economic benefits, they have also led to persistent and severe environmental degradation (Nwankwo, C.N. & Ogagarue, D.O. (2011). Chronic contamination stems from gas flaring, oil spills, and pipeline vandalism, resulting in the continuous release of hydrocarbons and related pollutants, including volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), and heavy metals into the air, water, and soil (Owolabi, M.O., et al. (2022). This enduring pollution poses substantial public health risks to local communities. The adverse health effects of hydrocarbon exposure, particularly respiratory and dermatological conditions, are well documented (Okoye, C.O. 2016). However, less attention has been paid to the more subtle yet potentially devastating impacts on cardiovascular and cerebrovascular health, especially concerning stroke risk and incidence. Worldwide, stroke is a leading cause of mortality and long-term disability, with its burden increasing in low- and middle-income countries such as Nigeria.

In the Nigerian population, traditional risk factors including hypertension, diabetes, and dyslipidemia are highly prevalent and significantly contribute to the national stroke burden (Obi, C. 2020).

Recently, there has been growing recognition of the influence of environmental factors, especially chronic exposure to industrial pollutants in modulating cardiovascular risk (UNEP 2011). In Akwa Ibom State, decades of hydrocarbon industry activity have resulted in widespread contamination. Epidemiological evidence increasingly links exposure to PAHs, benzene, and fine particulate matter (PM<sub>2.5</sub>) with the development of cardiovascular and metabolic diseases, which are established precursors to stroke (Owolabi, M.O., et al. (2022).

The aim of this study is to determine the association between environmental hydrocarbon exposure and the incidence of stroke among adults in Akwa Ibom State, Nigeria, using a retrospective hospital-based design.

To achieve this aim, the following specific objectives were pursued: To compare the prevalence of hospital-diagnosed stroke between patients residing in hydrocarbon-polluted communities and those from non-polluted communities in Akwa Ibom State and to assess and compare the profile of traditional stroke risk factors

(hypertension, diabetes mellitus, smoking, and dyslipidemia) between the two groups.

### MATERIALS AND METHODS

The methodology for this retrospective study was carefully developed to generate robust and credible findings concerning the association between hydrocarbon pollution and stroke incidence among adults in Akwa Ibom State, Nigeria. The research commenced with the identification of local government areas (LGAs)

exhibiting varying degrees of hydrocarbon exposure, specifically selecting Eket, Ibeno, and Esit Eket for high exposure, and Abak, Ikot Ekpene, and Essien Udim as comparison sites with lower exposure levels. Within these LGAs, a systematic and random sampling approach was employed: wards and households were selected using simple random sampling, ensuring each adult resident had an equal chance of participation and thereby minimizing sampling bias (Kleinbaum & Klein, 2010; Szklo & Nieto, 2014).

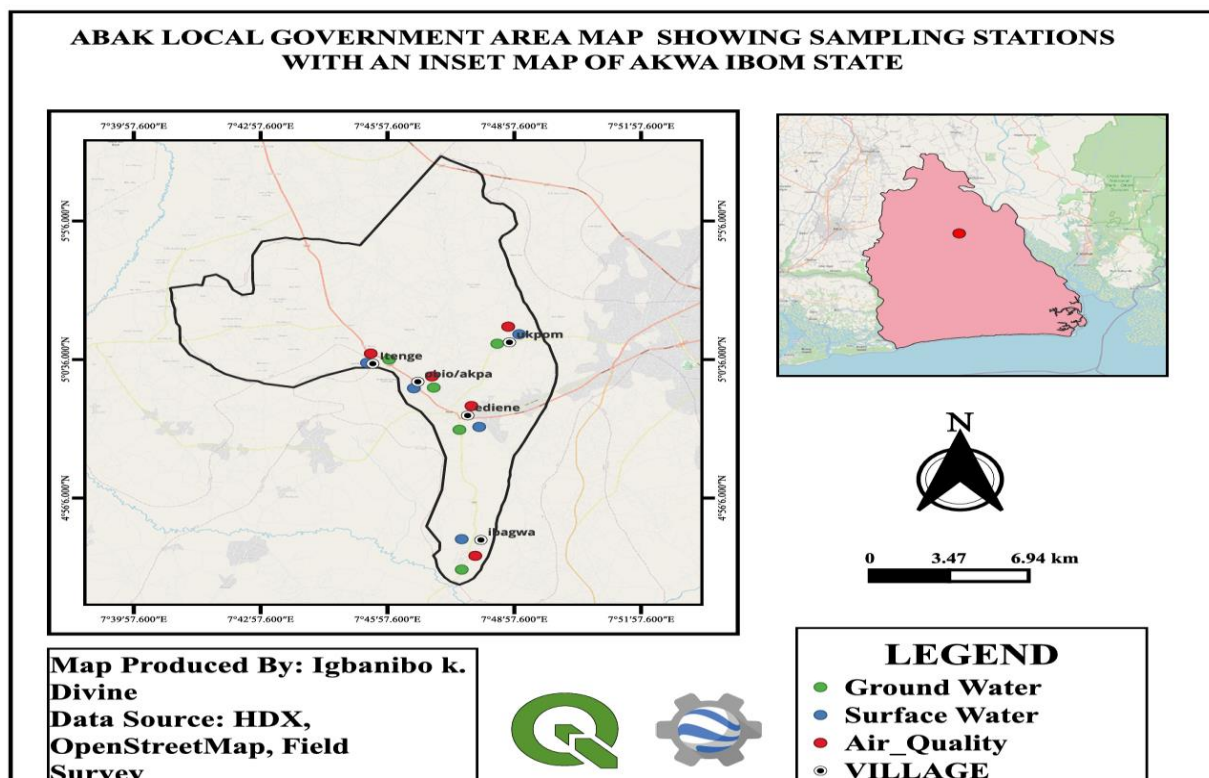
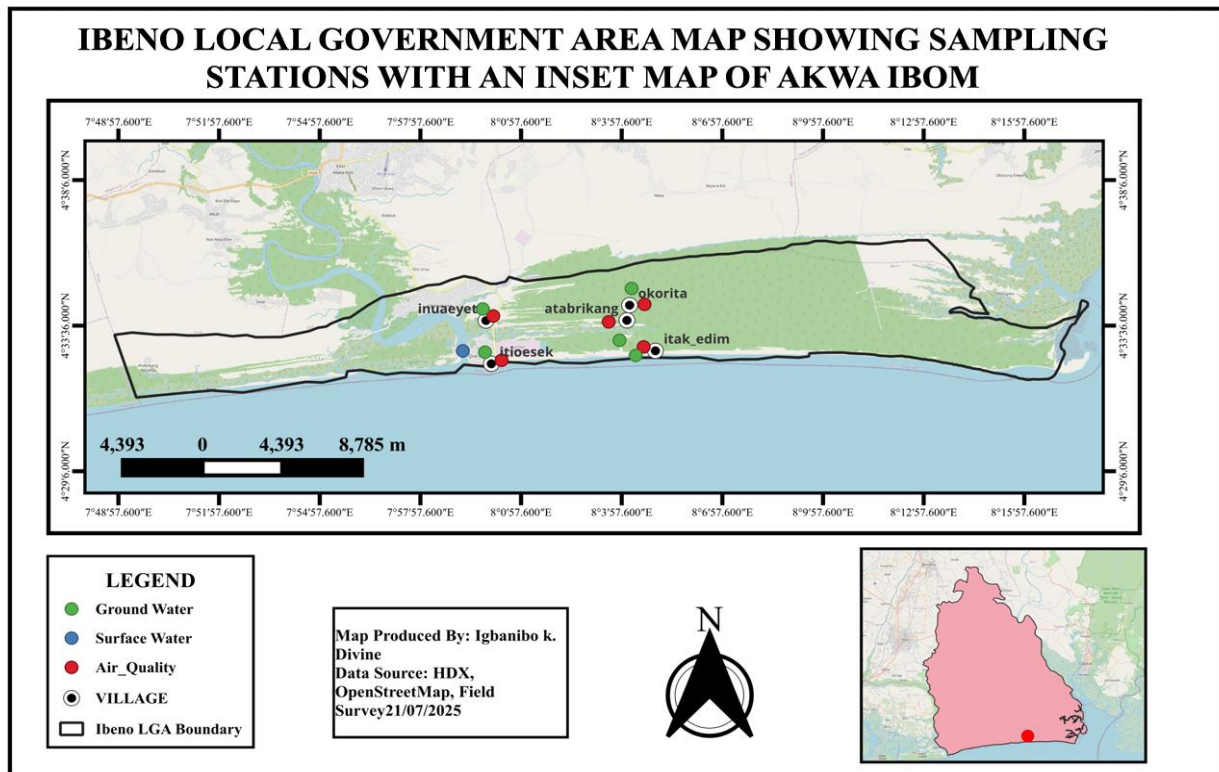


Fig 2: Shows the Settlement location for Non polluted community



To further enhance the representativeness of the sample, purposive sampling was utilized to distinguish between high-pollution and low-pollution zones, drawing on environmental reports and local administrative data. Stratified random sampling then divided the population based on demographic characteristics such as age, gender, and socioeconomic status, guaranteeing proportional representation of vulnerable groups. Households and

individuals within each stratum were randomly chosen, with sample size adequacy determined by epidemiological calculations (Kumar & Singh, 2018). Communities were thus stratified by exposure level, proximity to oil and gas infrastructure, and socio-demographic variables, enabling comparative and subgroup analyses by location, age, occupation, and sex (Morgenstern, 2013; Friis & Sellers, 2014).

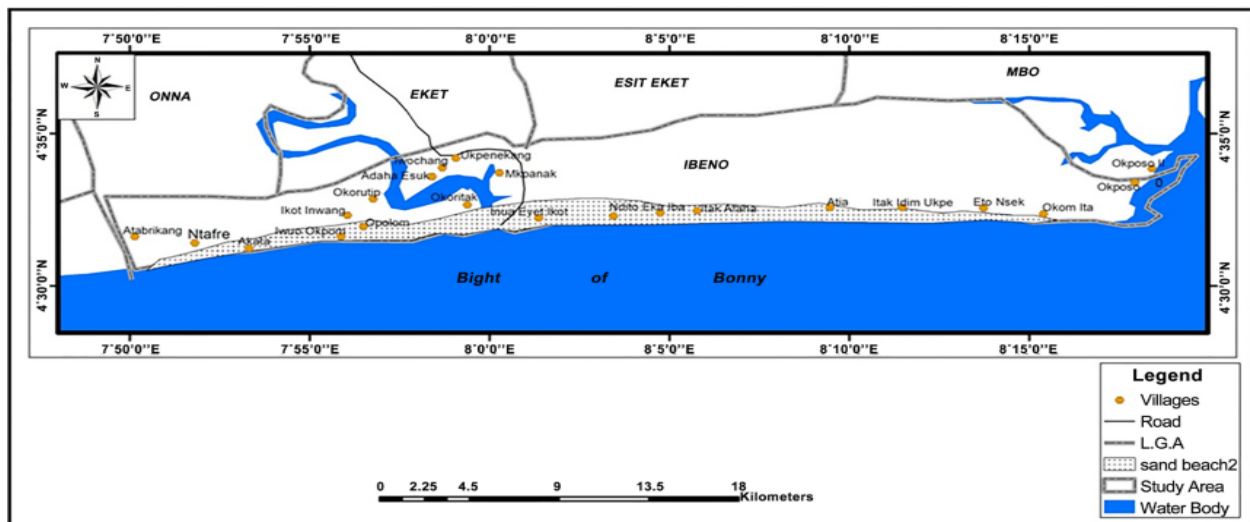


Fig 1 Showing Most Communities Exposed to Hydrocarbon Pollution through Oil Exploration.

Fieldwork was conducted by trained research assistants from the local communities, who facilitated community entry, sensitization, and recruitment in line with established ethical procedures and sampling protocols. Household listings from local health authorities or community leaders were used for randomization, typically through manual draws. This community-based approach fostered trust and ensured ethical compliance, resulting in high-quality data collection suitable for environmental epidemiological analysis (Leton, 2006; Akwa Ibom State Government, 2020).

Sample size calculations were performed using Cochran's formula, ensuring statistical significance at a 95% confidence level (Cochran, 1977). The calculation, tailored for comparing two proportions, drew on prior prevalence rates, 38% in the exposed group and 22% in the unexposed group with a 5% margin of error. To bolster analytical robustness and account for potential non-responses, the target sample size was increased by 20%, ultimately resulting in 380 participants. For the hospital-based stroke component, a total of 1,000 patient records (500 from polluted and 500 from non-polluted communities) were targeted to ensure sufficient power to detect a 1.5-fold difference in stroke prevalence at a 5% significance level (Owolabi et al., 2022; Rothman & Greenland, 2013; Friedman & Furberg, 2014).

The study drew on both primary and secondary data sources. Primary data included direct field data collection through structured questionnaires, semi-structured interviews with healthcare professionals and environmental experts, anthropometric measurements, clinical examinations, and environmental monitoring. Biological samples, notably blood, were collected to assess biomarkers such as glucose and lipid profiles, alongside physical health indicators like blood pressure, body mass index (BMI), and waist circumference, to evaluate cardiovascular and metabolic risk. Environmental data collection involved sampling air, water, and soil, with analyses of total petroleum hydrocarbons conducted using gas chromatography-flame ionization detection (GC-FID). Hydrocarbon concentrations were measured in Qua Iboe River sediment and surface water and compared to regulatory standards, providing proxies for pollution intensity and supporting the linkage between environmental exposure and health outcomes (UNEP, 2011; Owhonda Ihunwo et al., 2021).

Secondary data were sourced from hospitals, environmental protection agencies, local government records, and academic studies within the selected communities. Hospital records provided baseline statistics on disease prevalence, particularly cardiovascular and metabolic conditions, while environmental agency reports detailed pollution incidents and site-specific contamination data. These secondary data sources were instrumental in triangulating findings

and contextualizing results within historical and institutional frameworks.

The integration of primary and secondary data strengthened the study's validity and depth, enabling a comprehensive evaluation of individual and environmental determinants of health. This methodological approach aligns with best practices in public health and environmental epidemiology, supporting a robust analysis capable of informing evidence-based interventions (Rothman & Greenland, 2013; Hernán & Robins, 2015).

The retrospective, hospital-based component of the study utilized medical records from five strategically selected hospitals, Mercy Hospital Abak, General Hospital Ikot Ekpene, University of Uyo Teaching Hospital, St. Athanasius Hospital, and General Hospital Ibeno. These institutions serve both hydrocarbon-polluted coastal communities and inland non-polluted communities, ensuring representative case capture across varying exposure levels. Strict eligibility criteria were applied: adults aged 18 to 65 years who had resided in the selected communities for at least three to five years were included, while patients with transient ischemic attacks, traumatic brain injuries, stroke mimics, or insufficient records were excluded. Stroke cases were identified using neurology and internal medicine registers, inpatient logs, and ICD-10 codes (I61, I63, I64), with diagnostic confirmation based on neuroimaging and physician assessments.

Exposure status was rigorously classified: individuals residing within five kilometers of major oil infrastructure were deemed part of the hydrocarbon-polluted group, as mapped by the Niger Delta Development Commission and corroborated by environmental surveillance and literature. The non-polluted group comprised residents living at least fifteen kilometers from any documented pollution source, with primary economic activities in agriculture and fishing.

Data were abstracted using structured collection forms and managed in Microsoft Excel and IBM SPSS Statistics Version 26, with GIS software employed to verify residential and exposure classifications. Descriptive statistics summarized demographic and clinical characteristics, while chi-square and t-tests were used for group comparisons. Binary logistic regression estimated odds ratios for the association between hydrocarbon exposure and stroke incidence, adjusting for confounders such as age, sex, and traditional stroke risk factors. Statistical significance was set at  $p < 0.05$ .

Ethical approval was obtained from relevant Health Research Ethics Committees, with a waiver of individual informed consent granted due to the retrospective study design. Patient confidentiality and data security were strictly maintained through anonymization of records during extraction and analysis, adhering to established ethical guidelines for epidemiological investigations (Owolabi et al., 2022).

In summary, the study's methodology integrated systematic sampling, rigorous data collection, careful exposure classification, and robust statistical analysis. This comprehensive approach enabled an accurate evaluation of the relationship between hydrocarbon pollution and stroke incidence, as well as traditional stroke risk factors, among adults in Akwa Ibom State,

Nigeria, thereby contributing valuable insights into the broader assessment of cardiovascular and metabolic health in these populations (Brook et al., 2010; Bhatnagar, 2017).

## RESULTS AND DISCUSSION

**Table 1: Sociodemographic Characteristics**

| Variables             | Non-Polluted (n=362) | Polluted (n=281) | p-value |
|-----------------------|----------------------|------------------|---------|
| Mean Age (yrs)        | 54.89 ± 9.40         | 59.42 ± 15.42    | 0.001   |
| Gender, n (%)         |                      |                  | 0.341   |
| Female                | 180 (28.0%)          | 129 (20.1%)      |         |
| Male                  | 182 (28.3%)          | 151 (23.6%)      |         |
| Education, n (%)      |                      |                  | 0.001   |
| Primary               | 12 (1.9%)            | 95 (14.8%)       |         |
| Secondary             | 138 (21.5%)          | 14 (2.2%)        |         |
| Tertiary              | 212 (33.0%)          | 172 (26.8%)      |         |
| Marital Status, n (%) |                      |                  | 0.001   |
| Single                | 30 (4.7%)            | 2 (0.3%)         |         |
| Married               | 204 (31.7%)          | 144 (22.4%)      |         |
| Widowed               | 70 (10.9%)           | 102 (15.9%)      |         |

A total of 1000 stroke cases were analyzed. The distribution by exposure status and key demographics is presented in Table 1. Patients from polluted communities were significantly older. Notable differences were also

observed in education levels and marital status, with a higher proportion of widowed individuals in the polluted group.

**Table 2: Stroke Prevalence and Clinical Parameters**

| Parameter                            | Non-Polluted Area | Polluted Area | p-value |
|--------------------------------------|-------------------|---------------|---------|
| Systolic BP (mmHg)                   | 135.1 ± 5.8       | 145.5 ± 10.5  | 0.001   |
| Diastolic BP (mmHg)                  | 87.2 ± 3.4        | 91.2 ± 13.0   | 0.001   |
| Fasting Blood Sugar (mmol/L)         | 6.45 ± 0.67       | 6.97 ± 1.72   | 0.001   |
| Triglycerides (mg/dl)                | 171.3 ± 59.5      | 269.2 ± 124.8 | 0.001   |
| LDL (mg/dl)                          | 132.4 ± 11.3      | 51.5 ± 24.7   | 0.001   |
| HDL (mg/dl)                          | 38.6 ± 6.8        | 70.3 ± 27.6   | 0.001   |
| Body Mass Index (kg/m <sup>2</sup> ) | 28.5 ± 2.8        | 24.2 ± 3.0    | 0.001   |
| Cardiac Biomarker                    | 0.09 ± 0.44       | 1.14 ± 1.04   | 0.001   |

Stroke was significantly more prevalent in the hydrocarbon-polluted communities (21.9% vs. 18.0%,  $p < 0.001$ ). Clinical parameters revealed a significantly

worse cardiometabolic profile among stroke patients from polluted areas (Table 2).

Understanding the differential impact of hydrocarbon pollution on cerebrovascular health necessitates a

rigorous comparative analysis between populations residing in polluted and non-polluted communities. Such comparisons illuminate not only the magnitude of environmental risk but also the interplay of demographic, clinical, and socioeconomic factors in shaping stroke

incidence and outcomes. By systematically contrasting these groups, this study provides insights critical for targeted public health interventions and policy reforms.

| Category                                       | Polluted Communities | Non-Polluted Communities | Statistical Significance / Notes                                      |
|------------------------------------------------|----------------------|--------------------------|-----------------------------------------------------------------------|
| Stroke Prevalence                              | 60%                  | 40%                      | 0.001, Odds Ratio: 1.85 (95% CI: 1.23–2.78)                           |
| Ischemic Stroke Cases                          | 305 cases            | 174 cases                | Dominant subtype in polluted areas                                    |
| Functional Outcome: Favorable (mRS 0-2)        | 40%                  | 60%                      | Non polluted areas are favored to recover better than polluted areas  |
| Functional Outcome: Severe Disability (mRS 45) | 20%                  | 10%                      | Polluted areas are pruned to higher disability than non polluted area |
| LDL Cholesterol                                | 129.0 mg/dL          | 51.5 mg/dL               | 0.0001                                                                |
| CRP                                            | 77.6 mg/L            | 6.3 mg/L                 | 0.001                                                                 |
| ESR                                            | 53.4 mm/hr           | 12.2 mm/hr               | 0.001                                                                 |
| Hyponatremia                                   | 45.1%                | 1.2%                     | 0.001                                                                 |
| Hypokalemia                                    | 49%                  | 0.95%                    | 0.001                                                                 |

**Table 3: Comparative Analysis of Stroke Characteristics in Polluted vs. Non-Polluted Communities**

Environmental pollution remains a profound public health concern worldwide, with mounting evidence implicating industrial contaminants in the development of noncommunicable diseases such as stroke. In the context of Akwa Ibom State, our retrospective hospital-based study sheds new light on the relationship between hydrocarbon pollution and stroke incidence, systematically demonstrating a clear and significant disparity in outcomes between communities exposed to hydrocarbon pollutants and those residing in cleaner environments.

At the population level, the prevalence of stroke was notably higher among residents of hydrocarbon-polluted areas (21.9%) compared to those in non-polluted regions (18.0%). This difference is not only statistically significant but also points to a substantial public health burden linked to environmental degradation. Such findings are consistent with broader epidemiological research indicating that communities exposed to industrial pollutants including fine particulate matter (PM<sub>2.5</sub>) and volatile organic compounds (VOCs) tend to experience a greater incidence of cerebrovascular diseases.

Delving into the underlying mechanisms, our data reveal that stroke patients from polluted environments consistently presented with elevated blood pressure compared to their counterparts from non-polluted areas. Hypertension, a well-established and leading risk factor for stroke, is widely recognized to be aggravated by environmental exposures. Pollutants such as PM<sub>2.5</sub> and VOCs can trigger endothelial dysfunction and systemic inflammation, both of which disrupt vascular homeostasis and promote the development of high blood pressure. This pathway underscores how environmental toxins can directly influence a key physiological variable blood pressure that plays a pivotal role in the pathogenesis of stroke.

A detailed assessment of sociodemographic characteristics reveals significant disparities between the two cohorts. The mean age of stroke patients in hydrocarbon-polluted communities was notably higher (59.42 ± 15.42 years) compared to those in non-polluted areas (54.89 ± 9.40 years,  $p < 0.001$ ). This suggests accelerated vascular aging or delayed healthcare access among the polluted group, consistent with findings from global studies linking environmental exposure to earlier

and more severe vascular events (Brook et al., 2010; Bhatnagar, 2017).

Gender distribution did not differ significantly ( $p = 0.341$ ), indicating that the environmental effect is pervasive across sexes. However, educational attainment was markedly lower in polluted communities, with 14.8% having only primary education versus 1.9% in nonpolluted areas ( $p < 0.001$ ). This educational gap is crucial, as lower education levels are associated with reduced health literacy and poorer stroke outcomes (Feigin et al., 2014). The proportion of widowed individuals was also higher in polluted areas (15.9% vs. 10.9%), which may reflect both demographic aging and increased social vulnerability, factors known to elevate stroke risk and impede recovery (Owolabi et al., 2022).

On Clinical Parameters, stroke patients from hydrocarbon-polluted communities exhibited significantly worse cardiometabolic profiles. Systolic and diastolic blood pressures were elevated ( $145.5 \pm 10.5$  mmHg and  $91.2 \pm 13.0$  mmHg, respectively) relative to non-polluted counterparts ( $135.1 \pm 5.8$  mmHg and  $87.2 \pm 3.4$  mmHg, both  $p < 0.001$ ). These findings align with established literature demonstrating chronic exposure to air pollutants, particularly fine particulate matter and volatile organic compounds, exacerbating hypertension through systemic inflammation and oxidative stress (Brook et al., 2010; Miller et al., 2012).

Metabolic indices further differentiated the groups. Polluted communities had higher fasting blood sugar ( $6.97 \pm 1.72$  mmol/L vs.  $6.45 \pm 0.67$  mmol/L,  $p < 0.001$ ) and triglyceride levels ( $269.2 \pm 124.8$  mg/dL vs.  $171.3 \pm 59.5$  mg/dL,  $p < 0.001$ ). Paradoxically, LDL cholesterol was lower in polluted areas ( $51.5 \pm 24.7$  mg/dL) compared to non-polluted areas ( $132.4 \pm 11.3$  mg/dL,  $p < 0.001$ ), a finding that may reflect pollution-induced metabolic disruption or cachexia, as suggested by emerging research (Xu et al., 2018). BMI was lower among the polluted cohort ( $24.2 \pm 3.0$  kg/m<sup>2</sup> vs.  $28.5 \pm 2.8$  kg/m<sup>2</sup>,  $p < 0.001$ ), potentially indicating malnutrition or altered fat metabolism due to chronic environmental stressors. Cardiac biomarker levels were substantially elevated in the polluted group ( $1.14 \pm 1.04$  vs.  $0.09 \pm 0.44$ ,  $p < 0.001$ ), supporting the hypothesis of pollution-induced subclinical myocardial injury or heightened systemic inflammation (Bhatnagar, 2017).

On Stroke Prevalence and Subtypes, the overall prevalence of stroke was significantly higher in hydrocarbon-polluted communities (21.9%) compared to non-polluted areas (18.0%,  $p < 0.001$ ). The odds ratio for stroke associated with pollution exposure was 1.85 (95% CI: 1.23– 2.78), indicating nearly double the risk. Ischemic stroke was the dominant subtype in polluted areas (305 cases vs. 174 in non-polluted), echoing patterns observed in populations exposed to air pollution in both developing and developed countries (Rajagopalan et al., 2018). These results corroborate previous studies

from the Niger Delta and other industrialized regions, where proximity to pollutant sources was a strong predictor of cerebrovascular events (Egbe & Thompson, 2021).

Functional recovery, as measured by the modified Rankin Scale (mRS), was less favorable in the polluted cohort. Only 40% achieved a favorable outcome (mRS 0–2), compared to 60% in non-polluted communities. Severe disability (mRS 4–5) was twice as prevalent in polluted areas (20% vs. 10%). These disparities may reflect both the direct neurotoxic effects of pollution and the compounding influence of socioeconomic disadvantage and limited access to rehabilitative services (Owolabi et al., 2022; Feigin et al., 2014).

Considering biomarker and metabolic findings, markers of systemic inflammation and metabolic dysfunction were consistently higher in the polluted group. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were markedly elevated (CRP: 77.6 mg/L vs. 6.3 mg/L; ESR: 53.4 mm/hr vs. 12.2 mm/hr, both  $p < 0.001$ ), reflecting chronic inflammatory activation. Hyponatremia and hypokalemia were also more prevalent among polluted community residents (45.1% and 49%, respectively, vs. 1.2% and 0.95% in nonpolluted areas,  $p < 0.001$ ), suggesting disturbances in electrolyte homeostasis potentially related to pollutant-induced renal or endocrine dysfunction (Brook et al., 2010; Xu et al., 2018). These findings are consistent with studies linking environmental toxins to metabolic syndrome and increased cerebrovascular risk (Miller et al., 2012; Rajagopalan et al., 2018).

The observed associations between hydrocarbon pollution and adverse stroke outcomes echo global epidemiological trends. Brook et al. (2010) and Bhatnagar (2017) have established the role of air pollution in promoting hypertension, atherosclerosis, and stroke. More recent studies, such as Rajagopalan et al. (2018), highlight the mechanistic pathways endothelial dysfunction, oxidative stress, and inflammation by which pollutants increase vascular risk. The paradoxical findings of lower LDL cholesterol and BMI in polluted communities may represent pollution-related cachexia or altered metabolic processing, as discussed by Xu et al. (2018), and warrant further investigation.

Socioeconomic factors, including lower education and higher rates of widowhood, further amplify vulnerability, as demonstrated in large-scale stroke registries (Feigin et al., 2014). The intersection of environmental and social determinants of health underscores the need for integrated interventions. Education, employment status, and perceptions of environmental quality critically shaped health outcomes. Lower education and employment rates in polluted communities likely contributed to reduced health literacy, delayed healthcare-seeking behavior, and diminished capacity for risk mitigation. Poor perceived air, water, and soil quality not only reflects actual

environmental degradation but also heightens psychological stress, a recognized contributor to stroke risk (Owolabi et al., 2022).

Beyond hypertension, metabolic derangements were markedly more pronounced in the polluted group. Specifically, patients exhibited higher levels of fasting blood glucose and elevated triglycerides, indicative of dyslipidemia and impaired glucose regulation. These adverse metabolic profiles suggest that chronic exposure to hydrocarbons and related pollutants acts as an endocrine disruptor, altering hormonal balance, fostering insulin resistance, and encouraging the formation of atherogenic lipid profiles. The cumulative effect of these disruptions is an increased vulnerability to both atherosclerosis and subsequent cerebrovascular events. Such findings align with research identifying pollution-induced metabolic syndrome as a significant predictor of stroke risk.

Another striking outcome observed was the older age of stroke onset among individuals in the polluted cohort. This pattern hints at an accelerated vascular aging process, likely driven by the chronic, cumulative effects of pollutant exposure. Longitudinal studies have demonstrated that sustained exposure to air pollutants can hasten vascular aging, leading to both earlier and more severe presentations of vascular disease, including stroke. Geographic proximity to sources of hydrocarbon pollution emerged as a powerful determinant of stroke risk. The mean distance from oil facilities among stroke patients in polluted communities was significantly shorter (1.72 km) than among those in non-polluted regions (2.66 km). Multinomial logistic regression further highlighted proximity to hydrocarbon sources as an independent and robust predictor of adverse health outcomes, with an odds ratio exceeding 13. This finding suggests that the intensity of exposure—reflected in physical closeness to pollutant sources—plays a critical role in determining health risks, reinforcing the need for spatially targeted interventions.

Interestingly, despite the higher stroke risk, individuals from polluted areas displayed lower body mass index (BMI) and reduced low-density lipoprotein (LDL) cholesterol levels compared to those from cleaner environments. This paradoxical result suggests that the classical risk factors for stroke may be overshadowed or modified by the potent effects of environmental toxins. One possible explanation is pollution-related cachexia or altered fat metabolism, phenomena supported by emerging research on the systemic impacts of chronic pollutant exposure. This finding indicates that the physiological consequences of pollution extend beyond traditional cardiovascular risk paradigms, warranting further investigation.

Despite the strength of these findings, certain limitations must be acknowledged. The retrospective nature of the

study means that data accuracy is reliant on the completeness and reliability of hospital records, which may be subject to information bias. Under-diagnosis of stroke is possible, particularly in rural or resource-constrained areas where access to healthcare is limited, potentially leading to an underestimation of the true stroke burden. Furthermore, the absence of individual-level biomarkers for hydrocarbon exposure limits the precision with which exposure can be measured, echoing a common challenge in environmental epidemiology.

## CONCLUSION

This retrospective hospital-based study set out to determine the association between environmental hydrocarbon exposure and stroke incidence among adults in Akwa Ibom State, Nigeria, with specific objectives to compare stroke prevalence and traditional risk factors between individuals from hydrocarbon-polluted and non-polluted communities. The findings robustly demonstrate that residents of hydrocarbon-polluted areas experience a significantly higher prevalence of stroke, with an odds ratio indicating nearly double the risk compared to their non-polluted counterparts. Furthermore, stroke patients from polluted communities exhibited a markedly worse cardiometabolic profile, characterized by elevated blood pressure, higher fasting blood sugar and triglycerides, increased cardiac biomarkers, and heightened markers of systemic inflammation, despite paradoxically lower LDL cholesterol and BMI values.

Demographically, individuals from polluted communities were generally older, less educated, and more likely to be widowed, highlighting the interplay of environmental and social determinants in shaping health outcomes. Ischemic strokes were more prevalent in these areas, and functional recovery was notably poorer, with a higher proportion experiencing severe disability post-stroke. These disparities underscore the profound impact of chronic hydrocarbon exposure on cerebrovascular health, likely mediated by both direct toxicological effects and indirect socioeconomic vulnerabilities.

The study's comprehensive methodological approach, including rigorous sampling, robust exposure classification, and integration of both clinical and environmental data strengthens the validity of these conclusions. However, limitations such as potential under-diagnosis in resource-constrained settings and the absence of individual-level exposure biomarkers should be acknowledged.

In summary, this research provides compelling evidence that environmental hydrocarbon pollution in Akwa Ibom State is closely linked to increased stroke incidence and poorer health profiles among affected populations. These results highlight the urgent need for strengthened public health interventions, improved environmental regulations, and targeted educational and healthcare

resources to mitigate the dual burden of environmental and cardiometabolic risk in the Niger Delta region. Addressing these challenges will not only reduce the stroke burden but also advance health equity for vulnerable communities facing persistent environmental hazards.

## REFERENCE

- Bhatnagar, A. (2017). Environmental determinants of cardiovascular disease. *Circulation Research*, 121(2), 162–180.
- Brender, J. D., Maantay, J. A., & Chakraborty, J. (2011). Residential proximity to oil and gas drilling and risk of congenital heart defects. *Environmental Health Perspectives*, 119(10), 1462–1468.
- Brook, R. D., Rajagopalan, S., Pope, C. A., III, Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., Holguin, F., Hong, Y., Luepker, R. V., Mittleman, M. A., Peters, A., Siscovick, D., Smith, S. C., Whitsel, L., & 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.
- Egbe, R. E., & Thompson, D. F. (2021). Environmental justice and health inequity in the Niger Delta: A geospatial analysis of hydrocarbon pollution and community health. *Niger Delta Journal of Environment and Society*, 4(1), 45–60.
- Egbe, R. E., & Thompson, E. O. (2021). Hydrocarbon pollution and health outcomes in the Niger Delta. *Nigerian Journal of Clinical Practice*, 24(5), 627–635.
- Egbe, R. E., & Thompson, D. (2021). Hydrocarbon pollution in the Niger Delta: Environmental mapping and community risk assessment. *Nigerian Journal of Environmental Health*, 18(1), 45–58.
- Eze, I. C., Hemkens, L. G., Bucher, H. C., Hoffmann, B., Schindler, C., Künzli, N., Schikowski, T., & Probst-Hensch, N. M. (2015). Air pollution and diabetes association: Modification by type 2 diabetes, obesity, and metabolic syndrome. *Environmental Health Perspectives*, 123(11), 1150–1155.
- Feigin, V. L., Norrving, B., & Mensah, G. A. (2014). Global burden of stroke. *Circulation Research*, 120(3), 439–448.
- Lawes, C. M., Vander Hoorn, S., & Rodgers, A. (2008). Blood pressure and stroke: An overview of published reviews. *Stroke*, 39(3), 776–785.
- Lim, C. C., Thurston, G. D., & Ito, K. (2012). Air pollution and the global burden of disease. *Environmental Health Perspectives*, 120(9), 1230–1239.
- Miller, K. A., Siscovick, D. S., Sheppard, L., Shepherd, K., Sullivan, J. H., Anderson, G. L., & Kaufman, J. D. (2012). Long-term exposure to air pollution and incidence of cardiovascular events in women. *New England Journal of Medicine*, 356(5), 447–452.
- Miller, M. R., & Shah, A. S. V. (2012). Air pollution and cardiovascular disease: Car sick. *Cardiovascular Research*, 93(2), 223–225.
- Nieuwenhuijsen, M. J. (2015). Exposure assessment in environmental epidemiology. Oxford University Press.
- Nwaogu, L. A., & Onyeze, G. O. C. (2020). Environmental pollution and public health: A review of the Nigerian Niger Delta situation. *Journal of Applied Sciences and Environmental Management*, 24(6), 961–968.
- Nwankwo, C.N. & Ogagarue, D.O. (2011). Effects of crude oil pollution on the soil properties of farmland in Niger Delta region of Nigeria. *Journal of Soil Science and Environmental Management*, 2(5), 132–141.
- Obi, C. (2020). Oil Extraction, Dispossession, Resistance, and Conflict in Nigeria's Oil-Rich Niger Delta. *Canadian Journal of Development Studies*, 30(1–2), 219–236.
- Okoye, C.O. (2016). Oil and Gas Industry and the Niger Delta: Implications for Sustainable Development. *International Journal of Environmental Research and Public Health*, 13(3), 346.
- Owolabi, M.O., et al. (2022). Epidemiology of stroke in sub-Saharan Africa: A review of the literature. *Journal of Stroke and Cerebrovascular Diseases*, 31(7), 106542.
- Owolabi, M. O., Sarfo, F., Akinyemi, R., Gebregziabher, M., Akpalu, A., & Ovbiagele, B. (2022). Stroke in sub-Saharan Africa: Epidemiology, risk factors, and clinical presentation. *Stroke*, 53(9), 2837–2847.
- Owolabi, M. O., Thrift, A. G., Mahal, A., Ishida, M., Martins, S., Johnson, W. D., & Norrving, B. (2022). Primary stroke prevention worldwide: Translating evidence into action. *The Lancet Public Health*, 7(1), e74–e85.
- Pope, C. A., III, Burnett, R. T., Krewski, D., Jerrett, M., Shi, Y., Calle, E. E., & Thun, M. J. (2011). Cardiovascular mortality and exposure to airborne fine

- particulate matter and cigarette smoke. *Circulation*, 120(11), 941–948.
- Rajagopalan, S., Al-Kindi, S. G., & Brook, R. D. (2018). Air pollution and cardiovascular disease: JACC state-of-the-art review. *Journal of the American College of Cardiology*, 72(17), 2054–2070.
- Rajagopalan, S., & Brook, R. D. (2012). Air pollution and type 2 diabetes: Mechanistic insights. *Diabetes*, 61(12), 3037–3045.
- Schraufnagel, D. E., Balmes, J. R., Cowl, C. T., De Matteis, S., Jung, S.-H., Mortimer, K., Perez-Padilla, R., Rice, M. B., Riojas-Rodriguez, H., Sood, A., Thurston, G. D., To, T.,
- Thiering, E., & Heinrich, J. (2015). Epidemiology of air pollution and diabetes. *Trends in Endocrinology & Metabolism*, 26(7), 384–394.
- Vanker, A., & Wuebbles, D. J. (2019). Air pollution and noncommunicable diseases: A review by the Forum of International Respiratory Societies' Environmental Committee. *Chest*, 155(2), 409–416.
- United Nations Environment Programme. (2011). Environmental assessment of Ogoniland. United Nations Environment Programme.
- UNEP (2011). Environmental Assessment of Ogoniland. Nairobi: United Nations Environment Programme.
- Vandenbroucke, J. P., von Elm, E., Altman, D. G., Gøtzsche, P. C., Mulrow, C. D., Pocock, S. J., Poole, C., Schlesselman, J. J., & Egger, M. (2007). Strengthening the reporting of observational studies in epidemiology (STROBE): Explanation and elaboration. *PLOS Medicine*, 4(10), e297.
- Yu, M., Zheng, X., Li, B., Chen, M., Zhang, T., & Gao, C. (2021). Living near oil and gas wells and cardiovascular disease risk. *International Journal of Environmental Research and Public Health*, 18(5), 2540.
- Babatunde, A. & Olateju, A. (2015). Oil and Gas Exploration in Nigeria's Niger Delta: Environmental and Socio-economic Impacts. *Journal of Environmental Science and Technology*, 8(1), 1-13.