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The burden of hypertension in an oil and gas-polluted environment: a comparative cross-sectional study

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Background

Evidence of positive association between traffic-related air pollution and elevated blood pressure has been published widely. However, the risk of hypertension and prolonged exposure to crude oil pollution and gas flares remains unexplored.

Methods

We recruited 2,028 residents (aged 18-80) in cross-sectional survey of both oil/gas polluted and non-polluted communities in the Niger Delta region of Nigeria. Prevalence and risk of hypertension, anthropometric indices, lifestyle and socio-demographic factors, and cardiovascular comorbidities were examined and compared between the two groups. Hypertension was defined as blood pressure ≥140/90 mmHg or on anti-hypertensive medication. Both univariate and multivariate logistic regression models were used to examine factors associated with hypertension. Model fits statistics were used to assess the parsimonious model and predictive power.

Results

More than one-third of participants were hypertensive (37.4%). Half of the participants were from oil polluted areas (51%). Only 15% of participants reported family history of hypertension. In the adjusted model, participants living in oil polluted areas were almost five times as likely to have developed hypertension (adjusted odds ratio [aOR] = 4.85, 95% CI 1.84 to 12.82) compared to participants in unpolluted areas. Age modifies the association between pollution status and risk of hypertension. For every ten years increase in the age of the participants, the odds of developing hypertension increased by 108% (aOR = 2.08, 95% CI 1.77 to 2.43).

Conclusion

The results suggested that exposure to oil/gas pollution may be associated with an increased risk of hypertension. Our findings need to be further investigated in longitudinal studies.

INTRODUCTION

Residents of oil and gas host communities are exposed 24 hours per day and 7 days per week to the emissions of gas flaring and oil polluted surface and underground water[1-3]. Chronic high-level and prolonged low-level exposure to these contaminants comes with huge cost not only to humans, the flora and fauna but also to the environment comprising the air, soil and water bodies.

A meta-analysis of epidemiological studies has established strong and positive associations between exposure to environmental pollution and increased cardiovascular risk[4]. Apart from the particle size of particulate matter (PM), its trace metal (e.g. Cadmium, Arsenic, Lead, Cupper, Mercury, Manganese, Nickle, and Vanadium) components are the active oxidants that triggers inflammatory response and oxidative processes[5]. Toxicological evidence found that inflammatory dose of PM is associated with increase in plasma fibrinogen and viscosity, systemic and local inflammatory events[6], alterations in blood coagulability[7] and endothelial dysfunction [8]. Specifically, longer-term exposure to ambient air pollution and short term exposure to high PM concentration confers increased cardiovascular risk[9-12] through initiation of high blood pressure, an established determinant of atherogenesis and cardiovascular diseases (CVD), and a leading cause of death [13].

Gas flaring and oil refining may affect the sleep-wake–cycle in healthy individuals[14]. Prolonged exposure to dioxins particularly 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD), a major by-product of gas flaring and crude oil refining can cause neurological symptoms including sleep disturbances, neuralgia and severe headache[15]. Evidence found that sleep deprivation is positively associated with increased cardiovascular risk including hypertension[16,17].

Current review found that the prevalence of hypertension and other cardiovascular risk factors are significantly higher in urban cities in Nigeria compared to rural communities due to urbanisation and nutritional transition [18, 19]. This situation may be different in the Niger Delta rural communities where environmental oil and gas pollution has been increasing over time with a likely but unexplored impact on health outcomes particularly hypertension.

The objective of the present study was to estimate and compare the prevalence of hypertension in both exposed (polluted) and unexposed (non-polluted) residents in the Niger Delta region of Nigeria.

MATERIALS AND METHODS

Study setting and population

The study was carried out among residents exposed to oil and gas pollution in a dominantly oil and gas polluted community, and non-exposed residents from another community without any oil and gas exploration or related activities (Figure 1). The two communities have the same socioeconomic and cultural features in Eleme and Degema local Government area in Rivers State. However, they differ remarkably in terms of environmental pollution and exposure level largely due to oil exploration and gas flaring and allied industrial activities. The communities are about 60km apart with an estimated population of 30,580. Ebubu is a rural farming settlement with sparse social infrastructure. The community has huge functional oilfields and gas flaring sites in addition to a network of oil pipelines. It is a highly polluted community with history of oil and gas exploration activities by Shell Petroleum Development Company[1]. Usokun on the other hand is a pristine rural settlement, a non-oil and gas producing (unpolluted) community. This community constitutes the control population. It is an island, circled by Sambreiro River (an outlet of the Niger) with only one access road

connecting it to the urban areas. This rural community is inhabited by people whose predominant occupation is fishing and vegetable farming.

Study design and sample size

The study design is a community-based cross-sectional study. Participants were recruited through a door-to-door visit to randomly selected household and through invitations to attend a data collection sessions at the designated health centres. The sample size of eligible adults was calculated based on the assumed prevalence of hypertension of 18% reported elsewhere [20]. We estimated that a sample size of 2,010 was adequate for the two communities to detect the prevalence of hypertension with 90% power, 3% precision within 95% confidence level.

Data collection

The target population for the study was all men and women aged 18-80 years living continuously in the selected community for at least 10 years. The survey took place between June-September, 2014. Data collection was undertaken by research assistants recruited from the communities and trained according to standard protocols and procedures. All persons who gave informed consent (in writing and/or thumb print) were included in the study. A pre-tested study questionnaire was used to collect information on sociodemographic characteristics and lifestyle factors, as well as medical history of hypertension. Participants were asked about any previous diagnosis and current treatment for hypertension. Those with positive answers for hypertensive medications were asked to show their medication for confirmation. Anthropometric and blood pressure measurements were also taken by research assistants. Height was measured to the nearest 0.1 cm using a portable collapsible stadiometer (Leicester Height Measure SECA, Ltd, Birmingham, UK) placed on a firm level surface with

participants wearing no hat and shoes. Participants' body weight was measured to the nearest 0.1 kg while they were dressed in light clothing without footwear using calibrated digital weighing scale (SECA 877, GmbH, Hamburg, Germany). BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). Blood pressure was measured using an automated and validated[21,22] upper-arm digital blood pressure monitor (Omron M6 HEM-7001-E, Birmingham, UK) with an appropriately sized cuff. Three measures were taken two to three minutes apart, after the participant had been sitting for five minutes with their arm supported. The mean of the last two measurements was used in the analysis.

Outcome variable

We defined hypertension as systolic or diastolic BP ≥140 or ≥90 mm Hg, respectively or on anti-hypertensive medication. All hypertensive participants benefitted from free medical consultation and were given pre-printed referral letters to the Usokun or Ebubu Primary Health Centre or managed free of charge by a special team of doctors from the Community Medicine Department, University of Port Harcourt Teaching Hospital, Port Harcourt.

Exposure (determinant variable)

The main determinant variable of interest was oil pollution status of the place of residence of participants. This was categorised into two, whether the participants were from oil polluted area or not.

Potential confounders (control variables)

The following control variables were included in the study; they can be grouped into socio-demographics variables (age and sex of the participants, marital status, education attainment,

and employment status), lifestyle factors (smoking status, drinking status, sleep duration, salt and fat-intake) and family history of hypertension.

Ethical considerations

The protocol of this study was reviewed and approved by the Biomedical and Scientific Research Ethics Sub-committee, University of Warwick, United Kingdom and the Research Ethics Committee of the University of Port Harcourt, Nigeria prior to entry to the community. Approval for the study was also obtained from the respective community leaders and council of elders before formal contact with the participants.

Statistical analysis

We used summary statistics to show the distribution of the main variables. The values were expressed as absolute number with percentages and mean with standard deviation for categorical and continuous variables respectively. We performed both univariate and multivariate logistic regression to examine the associations between participants' socio-demographic and other characteristics with the risk of hypertension. We considered variables for inclusion in the final multivariate model if they reached a moderate level of significance ($p<0.25$) or from the conceptual framework underlined in previous studies[23]. It is also possible that these demographic variables may alter not only an individual's overall predisposition towards hypertension, but also the association between living in polluted area and risk of developing hypertension. For instance, the strength of the relationship between living in polluted area and likelihood of developing hypertension may be different for men and women. To explore this possibility, we added interaction terms between living in polluted area and each of the determinants to the multivariate model.

The following regression diagnostics were used to assess the goodness-of-fit of the model and to choose the parsimonious model: the Hosmer-Lemeshow goodness-of-fit test, tolerance test for multicollinearity and link test to check for model specification error. We also performed Receiver Operating Curves (ROC) (c-statistics) analyses to determine the predictive powers of the final multivariate model. The ROC curve plots the sensitivity of the model against 1 minus specificity for different cut-off points of the predicted probability of having hypertension. The greater the area under the ROC curve (upper limit =1), the better the model is at discriminating between hypertension cases. Results were presented as odds ratios (ORs) with 95% confidence intervals (CIs). All statistical analyses were carried out using Stata version 14 for Windows (Stata Corp, College Station, Texas). The significance tests were two-tailed and statistical significance was defined at the alpha level of 0.05. The paper is reported following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [24].

RESULTS

Summary statistics for the study participants

Descriptive statistics for study participants are shown in Table 1. More than one-third of participants were hypertensive (37.4%). Half of the participants were from oil polluted areas (51%) with preponderance of women (57.0%). The overall participants' mean age was 44.3 (standard deviation: 14.0), this differed between the polluted and unpolluted areas (44.7 vs 43.9), *P*=0.09. Most of the participants had secondary or higher education (61.8%) and currently working (81.7%). More than half of the participants were either overweight or obese (55.1%) and were mostly from the polluted area (62.3%). Most of the participants were non-smokers (83.3%) and 39.4% were reported to have never consumed alcohol. A significant percentage of participants in the unpolluted compared to polluted area reported

moderate-to-high salt (74.9% vs 46.8%) and fat intake (59.8% vs 44.2%), *P*=0.01, respectively.

Factors associated with hypertension

The results of univariate and multivariate logistics regression models are presented in Table 2. In the univariate analysis, with the exception of sex of the participants, all the variables were statistically significantly associated with hypertension. However, in the multivariate model, the following factors remained statistically significant with hypertension: pollution status, age, family history of hypertension, body mass index, drinking status, sleep deprivation, level of physical activity and fat intake. Participants living in oil polluted areas were almost five times as likely to have developed hypertension (adjusted odds ratio [aOR] = 4.85, 95% CI 1.84 to 12.82], *P*=0.01).

All the interaction terms, except for age and education attainment were not statistically significant and were dropped from the final multivariate model (Table 3). For every ten years increase in the age of the participants, the odds of developing hypertension increased by 108% (aOR = 2.08, 95% CI 1.77 to 2.43). As shown in Figure 2, age modifies the association between pollution status and risk of hypertension, such that predicted probability of hypertension was significantly higher for participants' between the age of 20 and 40 years old. After the age of 65, the effect of pollution status tended to diminish.

Compared with participants with normal weight, overweight or obese participants were more likely to be hypertensive (aOR = 1.33, 95% CI 1.04 to 1.69). Moderate-to-heavy consumption of alcohol (aOR = 1.63, 95% CI 1.19 to 2.23), moderate-to-severe sleep deprivation (aOR = 4.27, 95% CI 3.14 to 5.81), sedentary behaviour (aOR = 3.09, 95% CI 1.66 to 5.76), moderate-to-high fat intake (aOR = 1.39, 95% CI 1.08 to 1.78) were statistically significantly associated with increased risk of hypertension.

Model fit statistics

None of the model fits results provided reasons for concern. For age interaction (Table 3), the average VIF was 1.36 (ranged: 1.06 to 2.19), since the VIF values and average VIF did not exceed 10 and 6 respectively, we concluded that there was no multi-collinearity problem, such that there is no perfect linear relationship between the determining variables, and the estimates for logistic regression models included these variables can be uniquely computed. The link test (Table 3) indicated that the model was specified correctly (p=0.14), which suggests that it is unlikely that we have omitted relevant variable(s) that could predict the hypertension risk. In addition, the Hosmer-Lemeshow test indicated that model II fits the data well (p=0.28). The area under the ROC curve for final multivariable model was 0.86, indicating relatively good predictive power and has reasonably good discriminatory ability. Other model fit results for other variables are shown in Table 3.

DISCUSSION

This study attempted to explore the prevalence of hypertension and associated risk factors in a highly polluted community, as compared to an unpolluted community, in the Niger Delta region of Nigeria.

In this study, the overall prevalence of hypertension was 37.4%. This was higher than previous review estimates for the whole country in general and the rural communities in particular[18, 19]. It may be argued that the difference could have been due to the participants mean age. However, the fact that our population is younger than study subjects in some urban environments of Niger Delta with lower prevalence[25, 26] makes this estimate more disturbing. We found that hypertension prevalence was significantly higher among participants in polluted environment (43.3%) compared to those in unpolluted area (31.2%), $P<0.01$.

Potential reason could be due to differences in exposure to pollution, or imbalance in socioeconomic and lifestyle factors between the two areas.

Among the potential confounding factors that were positively associated with hypertension, we noted that the proportion of those with no education, family history of hypertension, sleep deprivation, overweight and obesity were higher among the participants in the polluted area compared with those living in the unpolluted area, and that the proportion of participants with moderate-to-high intensity physical activity were lower in the polluted area compared to their counterparts in the unpolluted area. This may have contributed to the apparently higher prevalence of hypertension in the polluted area. After adjusting for these potential confounding factors, however, the risk of hypertension associated with residing in the polluted area remains (and indeed increased by more than 4-fold). The significant difference in risk attributed to polluted environment after adjustment add to our strong view that residents in polluted community have increased risk of hypertension irrespective of their BMI, socio-economic status, lifestyle and other predictors.

Environmental impact assessment (surface and ground water, land and ambient air evaluation) conducted in Ebubu community previously found very high percentage of polycyclic aromatic hydrocarbon (PAH), PM and heavy metals[1]. Evidence found that exposure to heavy metals such as Cadmium, Arsenic and Lead have been associated with hypertension[27-29]. Environmental pollutants such as heavy metals and PM trigger systemic inflammation and oxidative stress leading to hypertension through many pathophysiological mechanisms[4]. Therefore residents of polluted community like Ebubu are exposed not only to various air and soil pollutants but also to water and food pollutants especially due to bioaccumulation of heavy metals and other agents.

In addition to the above assessment, we also found interaction between age and oil pollution status and between education attainment and oil pollution status on overall predisposition towards hypertension. Possible explanation here could be related to increased exposure to pollutants among young adults and lack of awareness of the effect of pollution among those with no education. These cohorts (20-40 years old) are highly mobile and often spend substantial time engaging in work (semi-skilled and unskilled jobs in the oil and gas facilities) and leisure in open air or water. Similar differential exposure to indoor pollution and increased vulnerability has consistently been reported among women who spend between 3-7 hours indoors particularly in low and middle-income countries[30].

Biochemical evidence of exposure to dioxins such as TCDD (a major pollutant from oil and gas pollution) was known to effect the sleep-wake cycle mediated through autonomic nervous system dysfunction [4]. We found that participants in the polluted areas have significantly less than 5 hours of sleep on average compared to their counterparts (28.0% vs 12.9%), $P<0.01$. Consistent with previous study evidence, the short sleep duration may have increased the risk of hypertension in exposed subjects[16, 31.]

Apart from direct biochemical effect, other explanation to the increased hypertension burden in the polluted area could be related to the broader socio-economic and psychosocial impact. Oil pollution and influence of exploration activities of oil industry in the polluted community may increase vulnerability and loss of livelihood and psychosocial stress[32,33]. For instance, subsistent farmers that lost their small farm holding and source of drinking water and fishing source to oil pollution may be affected adversely due to loss of occupational activities, income and leisure. This increases exposure to chronic stress, possibly leading to disturbed sleep patterns, lifestyles and behavioural changes, and reduced access to health care. The results of these are inequality in health outcomes including hypertension.

Our study employed a relatively large sample size and rigorous methodology. However, the cross-sectional study design does not allow establishing temporality and causality for the observed associations. Caution therefore needs to be used in generalising the results because our study findings may not be applicable to the entire Niger delta region or communities that share a great diversity in socio-economic and environmental features. Other extrinsic factors such as healthcare access and availability remain potential important modifiers which must be factored in as well. In addition, the lack of detailed dietary history and our inability to assess other biomarkers, such as lipid profiles, may not only confound but also limit our understanding of the aetiology and influence of these to increased hypertension estimates. Therefore, the usefulness of pollution status in risk prediction of hypertension should be tested in large prospective studies which would take into account these potential confounders or modifiers. Our study has shown that environmental pollution may be useful for screening purposes to identify high risk population long before a diagnosis of hypertension is made and to target interventions appropriately.

Findings from this study suggest that oil and gas pollution be associated with a higher prevalence and risk of hypertension. This is disturbing and particularly so given that it is even higher than most urban estimates in majority of cities in the Niger Delta and Nigeria as a whole. While routine surveillance and management of hypertensive individuals remains an important public health priority, further epidemiological assessment of environmental pollutants in human tissues remains an important area for future research.

AUTHOR CONTRIBUTIONS

All authors contributed to the study concept and design. M. Ezejimofor, O. Maduka, A.Ezeabasili, A. Onwuchekwa, E. Asuquo and B. Ezejimofor contributed in data collection and entry. M. Ezejimofor, O. Uthman and A. Ezeabasili analysed the data. M. Ezejimofor wrote the first draft of the paper and all authors contributed in the manuscript correction and revision. The final manuscript was read and approved by all the authors for submission.

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M. Ezejimofor, reports receiving a Ph.D scholarship from Petroleum Technology Development Fund (PTDF) in Nigeria. The author is responsible for the opinion expressed in the manuscript. The funding source had no role in the study concept, design, data analysis and final manuscript.

DISCLOSURES

We declare that we have no conflicts of interest

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665 **Table 1. Characteristics of the study population by oil pollution status**

Variable	Total (n=2028)	Polluted area (n=1036)	Non-polluted area (n=992)	P-value[a]
Mean age (SD)	44.3 (14.0)	44.7 (13.3)	43.9 (14.7)	0.09
Gender (%)				
Male	871 (43.0)	417(40.0)	454 (46.0)	
Female	1157 (57.0)	619(60.0)	538(54.0)	0.01
Hypertensive[b] (%)				
No	1270 (62.6)	587(56.7)	683(68.9)	
Yes	758 (37.4)	449(43.3)	309(31.2)	0.01
Family history of hypertension (%)				
No	1724 (85.0)	847(81.8)	877(88.4)	
Yes	304 (15.0)	189(18.2)	115(11.6)	0.01
Marital Status (%)				
Never married	544 (26.8)	235(22.7)	309(31.2)	
Ever married	1483 (73.2)	800(77.3)	683(68.9)	0.01
Education attainment (%)				
No education (<6 years)	362 (17.9)	221(21.3)	141(14.2)	
Primary (6-11 years)	411 (20.2)	178(17.2)	233(23.5)	
Secondary/higher (≥ 12 Years)	1255 (61.8)	637(61.5)	618(62.3)	0.01
Employment status (%)				
Unemployed	371 (18.3)	165(15.9)	206(20.8)	
Presently working	1657 (81.7)	871(84.1)	786(79.2)	0.005
Body mass index (%)				
Underweight	35 (1.7)	17(1.6)	18(1.8)	
Normal	876 (43.2)	374(36.1)	502(50.6)	
Overweight/Obese	1117 (55.1)	645(62.3)	472(47.6)	0.01

Smoking status (%)				
Non-smoker	1689 (83.3)	925(89.3)	764(77.0)	
Ever smoker	339 (16.7)	111(10.7)	228(23.0)	0.01
Drinking status (%)				
Non-drinker	799 (39.4)	523(50.5)	276(27.8)	
Mild drinker	656 (32.3)	309(29.8)	347(35.0)	
Moderate-to-heavy drinker	573 (28.3)	204(19.7)	369(37.2)	0.01
Sleep deprivation (%)				
No	1326 (65.4)	564(54.4)	762(76.9)	
Mild	283 (14.0)	182(17.6)	101(10.2)	
Moderate-to-severe	418 (20.6)	290(28.0)	128(12.9)	0.01
Physical activity (%)				
Sedentary	79 (3.9)	33(3.2)	46(4.6)	
Low intensity	624 (30.8)	346(33.4)	278(28.0)	
Moderate-to-high intensity	1325 (65.3)	657(63.4)	668(67.3)	0.01
Salt intake (%)				
Low	800 (39.4)	551(53.2)	249(25.1)	
Moderate-to-high	1228 (60.6)	485(46.8)	743(74.9)	0.01
Fat intake (%)				
Low	977 (48.2)	578(55.8)	399(40.2)	
Moderate-to-high	1051 (51.8)	458(44.2)	593(59.8)	0.01

Data are expressed as mean (standard deviation) or as percentages.

[a]P values for comparison between polluted and non-polluted areas.

[b]Defined as blood pressure ≥ 140/90 mmHg or on antihypertensive medication.

675 **Table 2. Unadjusted and adjusted odd ratio of hypertension for selected risk factors**

Variable	Unadjusted model		Adjusted model	
	Odds ratio (95% CI)	p-value	)dds ratio (95% CI)	p-value
Main effects				
Polluted (vs unpolluted) area	1.69 (1.41 to 2.03)	0.01	4.85 (1.84 to 12.82)	0.01
Age (per 10 years increase)	2.27 (2.09 to 2.47)	0.01	2.08 (1.77 to 2.43)	0.01
Female (vs male)	1.06 (0.88 to 1.27)	0.54	Not included	
Family history of hypertension (vs no)	3.17 (2.46 to 4.08)	0.01	2.41 (1.75 to 3.32)	0.01
Ever (vs. never) married	6.21 (4.72 to 8.18)	0.01	1.62 (1.12 to 2.35)	0.01
Education attainment				
No education	2.43 (1.92 to 3.09)	0.01	1.34 (0.82 to 0.96)	0.002
Primary	1.34 (1.06 to 1.69)	0.01	0.90 (0.67 to 1.23)	0.52
Secondary or higher	1 (reference)		1 (reference)	
Currently employed (vs unemployed)	2.93 (2.23 to 3.86)	0.01	1.43 (0.95 to 2.15)	0.09
Body mass index				
Underweight	0.47 (0.19 to 1.14)	0.09	0.65 (0.22 to 1.89)	0.43
Normal	1 (reference)		1 (reference)	
Overweight/Obese	1.72 (1.43 to 2.08)	0.01	1.33 (1.04 to 1.69)	0.02
Ever- (vs. non-) smoker	1.61 (1.27 to 2.03)	0.01	1.28 (0.92 to 1.77)	0.14
Drinking status				
Non-drinker	1 (reference)		1 (reference)	
Mild drinker	0.67 (0.53 to 0.83)	0.01	0.76 (0.57 to 1.02)	0.06
Moderate-to-heavy drinker	1.80 (1.44 to 2.23)	0.01	1.63 (1.19 to 2.23)	0.02
Sleep deprivation				
No	1 (reference)		1 (reference)	
Mild	3.89 (2.98 to 5.07)	0.01	1.97 (1.44 to 2.68)	0.01

Moderate-to-severe	10.55 (8.16 to 13.64)	0.01	4.27 (3.14 to 5.81)	0.01
Physical activity				
Sedentary	6.50 (3.94 to 10.72)	0.01	3.09 (1.66 to 5.76)	0.01
Low intensity	3.21 (2.64 to 3.92)	0.01	2.81 (2.20 to 3.60)	0.01
Moderate-to-high intensity	1 (reference)		1 (reference)	
Moderate-to-high (vs. low) salt intake	1.50 (1.24 to 1.80)	0.01	1.10 (0.84 to 1.43)	0.50
Moderate-to-high (vs. low) fat intake	1.42 (1.18 to 1.70)	0.01	1.39 (1.08 to 1.78)	0.01

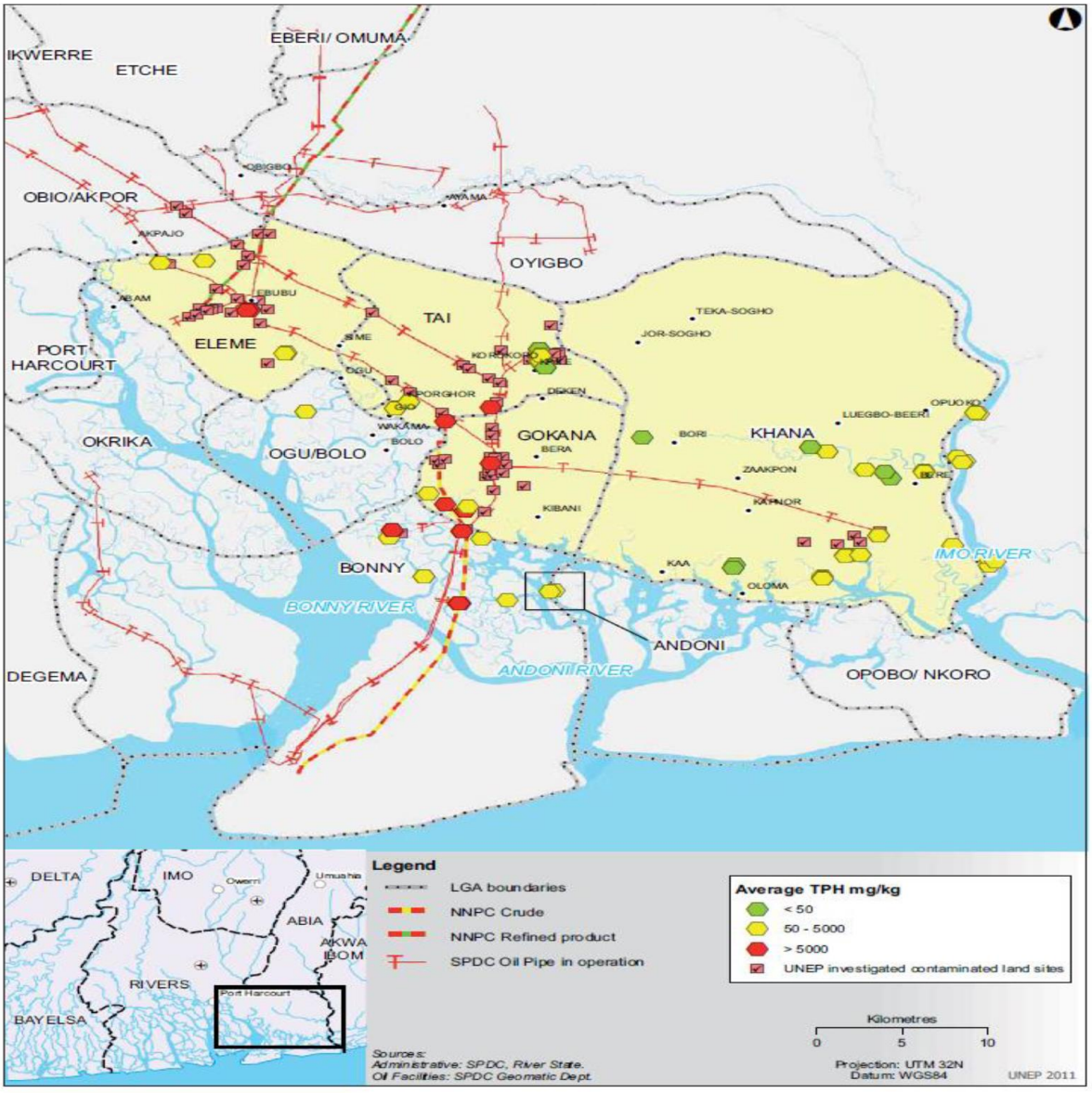
Table 3. Interaction of demographic variables and pollution status

Interaction effects	Odds ratio (95% CI)	p-value
Polluted (vs unpolluted) area #Age	0.78 (0.64 to 0.95)	0.01
Model fit statistics		
Area under ROC[1] curve		0.86
Lemeshow test (p-value)		0.28
Link test (p-value)		0.14
Collinearity diagnostic (mean VIF[2])		1.36
Polluted (vs unpolluted) area # Education attainment	0.39 (0.22 to 0.72)	0.02
Model fit Statistics		
Area under ROC[1] curve		0.86
Lemeshow test (p-value)		0.32
Link test (p-value)		0.09
Collinearity diagnostic (mean VIF[2])		0.20
Polluted (vs unpolluted) area # Sex	Not included	
Polluted (vs unpolluted) area # Marital status	0.88(0.45 to 1.69	0.69
Polluted (vs unpolluted) area # Employment status	0.59(0.26 to 1.29)	0.18

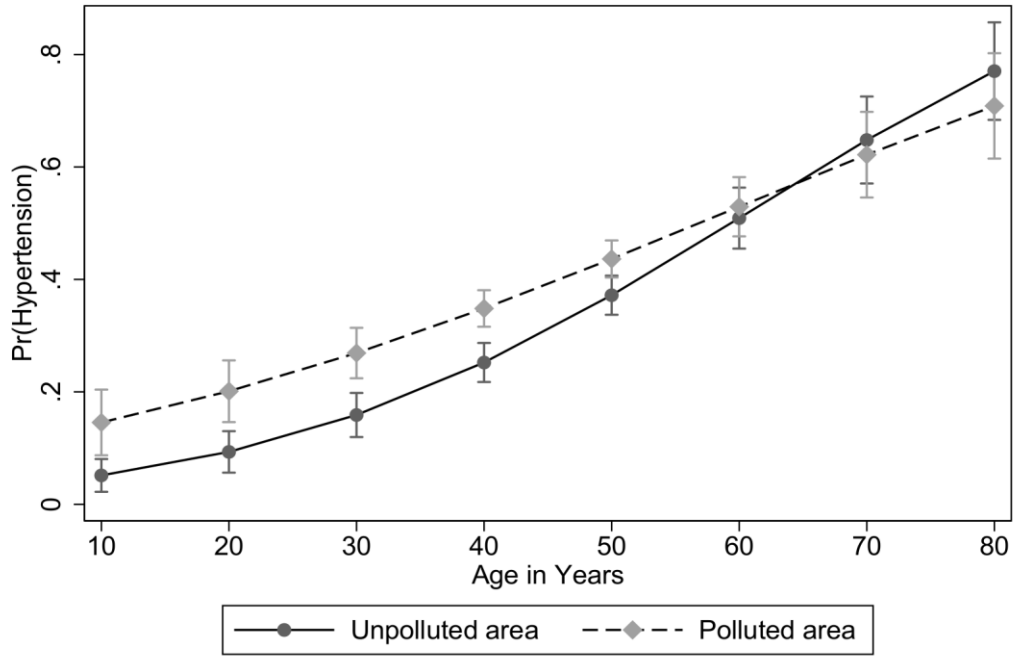
[1] Receiver Operating Characteristic

[2] Variance Inflation Factor

Figure 1. Map of the study local councils of the study areas showing oil and gas production sites investigated by UNEP[1]



713 **Figure 2. Predicted probability of hypertension for all ages by oil pollution status**



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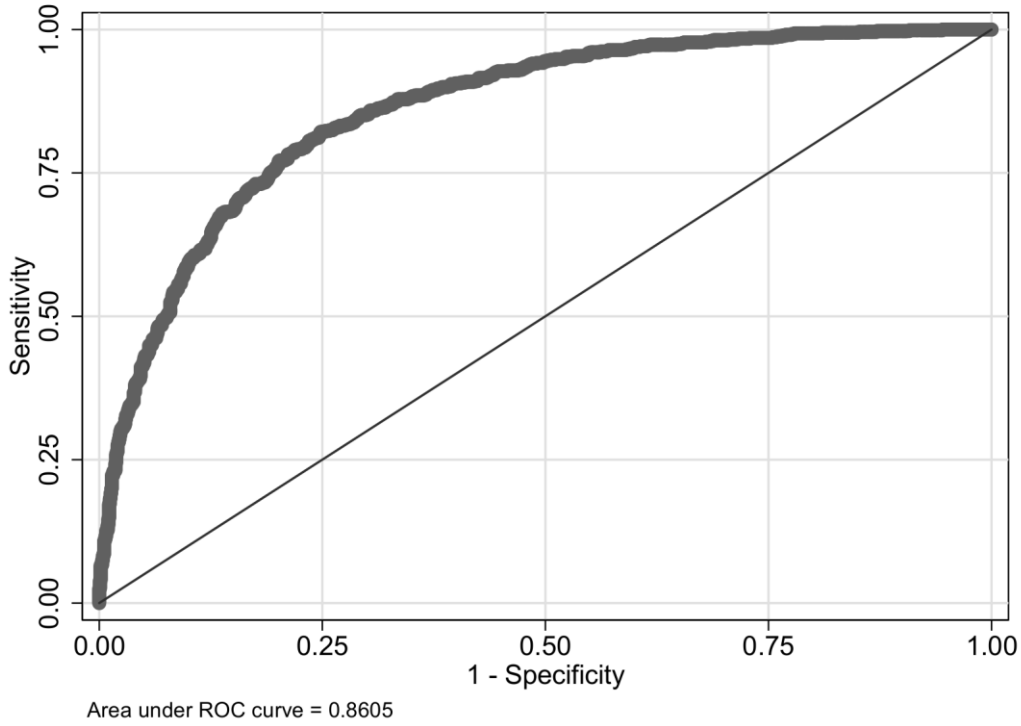
716 **Figure 3. Receiver operating characteristics curves for final multivariable model**



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The burden of hypertension in an oil and gas-polluted environment: a comparative cross-sectional study

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ABSTRACT

Background

Evidence of positive association between traffic-related air pollution and elevated blood pressure has been published widely. However, the risk of hypertension and prolonged exposure to crude oil pollution and gas flares remains unexplored.

Methods

We recruited 2,028 residents (aged 18-80) in cross-sectional survey of both oil/gas polluted and non-polluted communities in the Niger Delta region of Nigeria. Prevalence and risk of hypertension, anthropometric indices, lifestyle and socio-demographic factors, and cardiovascular comorbidities were examined and compared between the two groups. Hypertension was defined as blood pressure ≥140/90 mmHg or on anti-hypertensive medication. Both univariate and multivariate logistic regression models were used to examine factors associated with hypertension. Model fits statistics were used to assess the parsimonious model and predictive power.

Results

More than one-third of participants were hypertensive (37.4%). Half of the participants were from oil polluted areas (51%). Only 15% of participants reported family history of hypertension. In the adjusted model, participants living in oil polluted areas were almost five times as likely to have developed hypertension (adjusted odds ratio [aOR] = 4.85, 95% CI 1.84 to 12.82) compared to participants in unpolluted areas. Age modifies the association between pollution status and risk of hypertension. For every ten years increase in the age of the participants, the odds of developing hypertension increased by 108% (aOR = 2.08, 95% CI 1.77 to 2.43).

Conclusion

The results suggested that exposure to oil/gas pollution may be associated with an increased risk of hypertension. Our findings need to be further investigated in longitudinal studies.

INTRODUCTION

Residents of oil and gas host communities are exposed 24 hours per day and 7 days per week to the emissions of gas flaring and oil polluted surface and underground water[1-3]. Chronic high-level and prolonged low-level exposure to these contaminants comes with huge cost not only to humans, the flora and fauna but also to the environment comprising the air, soil and water bodies.

A meta-analysis of epidemiological studies has established strong and positive associations between exposure to environmental pollution and increased cardiovascular risk[4]. Apart from the particle size of particulate matter (PM), its trace metal (e.g. Cadmium, Arsenic, Lead, Cupper, Mercury, Manganese, Nickle, and Vanadium) components are the active oxidants that triggers inflammatory response and oxidative processes[5]. Toxicological evidence found that inflammatory dose of PM is associated with increase in plasma fibrinogen and viscosity, systemic and local inflammatory events [6], alterations in blood coagulability [7] and endothelial dysfunction [8]. Specifically, longer-term exposure to ambient air pollution and short term exposure to high PM concentration confers increased cardiovascular risk [9-12] through initiation of high blood pressure, an established determinant of atherogenesis and cardiovascular diseases (CVD), and a leading cause of death [13].

Gas flaring and oil refining may affect the sleep-wake–cycle in healthy individuals[14]. Prolonged exposure to dioxins particularly 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD), a major by-product of gas flaring and crude oil refining can cause neurological symptoms including sleep disturbances, neuralgia and severe headache[15]. Evidence found that sleep deprivation is positively associated with increased cardiovascular risk including hypertension[16,17].

Current review found that the prevalence of hypertension and other cardiovascular risk factors are significantly higher in urban cities in Nigeria compared to rural communities due

to urbanisation and nutritional transition [18, 19]. This situation may be different in the Niger Delta rural communities where environmental oil and gas pollution has been increasing over time with a likely but unexplored impact on health outcomes particularly hypertension.

The objective of the present study was to estimate and compare the prevalence of hypertension in both exposed (polluted) and unexposed (non-polluted) residents in the Niger Delta region of Nigeria.

MATERIALS AND METHODS

Study setting and population

The study was carried out among residents exposed to oil and gas pollution in a dominantly oil and gas polluted community, and non-exposed residents from another community without any oil and gas exploration or related activities (Figure 1). The two communities have the same socioeconomic and cultural features in Eleme and Degema local Government area in Rivers State. However, they differ remarkably in terms of environmental pollution and exposure level largely due to oil exploration and gas flaring and allied industrial activities. The communities are about 60km apart with an estimated population of 30,580. Ebubu is a rural farming settlement with sparse social infrastructure. The community has huge functional oilfields and gas flaring sites in addition to a network of oil pipelines. It is a highly polluted community with history of oil and gas exploration activities by Shell Petroleum Development Company[1]. Usokun on the other hand is a pristine rural settlement, a non-oil and gas producing (unpolluted) community. This community constitutes the control population. It is an island, circled by Sambreiro River (an outlet of the Niger) with only one access road connecting it to the urban areas. This rural community is inhabited by people whose predominant occupation is fishing and vegetable farming.

Study design and sample size

The study design is a community-based cross-sectional study. Participants were recruited through a door-to-door visit to randomly selected household and through invitations to attend a data collection sessions at the designated health centres. The sample size of eligible adults was calculated based on the assumed prevalence of hypertension of 18% reported elsewhere [20]. We estimated that a sample size of 2,010 was adequate for the two communities to detect the prevalence of hypertension with 90% power, 3% precision within 95% confidence level.

Data collection

The target population for the study was all men and women aged 18-80 years living continuously in the selected community for at least 10 years. The survey took place between June-September, 2014. Data collection was undertaken by research assistants recruited from the communities and trained according to standard protocols and procedures. All persons who gave informed consent (in writing and/or thumb print) were included in the study. A pre-tested study questionnaire was used to collect information on sociodemographic characteristics and lifestyle factors, as well as medical history of hypertension. Participants were asked about any previous diagnosis and current treatment for hypertension. Those with positive answers for hypertensive medications were asked to show their medication for confirmation. Anthropometric and blood pressure measurements were also taken by research assistants. Height was measured to the nearest 0.1 cm using a portable collapsible stadiometer (Leicester Height Measure SECA, Ltd, Birmingham, UK) placed on a firm level surface with participants wearing no hat and shoes. Participants' body weight was measured to the nearest 0.1 kg while they were dressed in light clothing without footwear using calibrated digital weighing scale (SECA 877, GmbH, Hamburg, Germany). BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). Blood pressure was measured

using an automated and validated[21,22] upper-arm digital blood pressure monitor (Omron M6 HEM-7001-E, Birmingham, UK) with an appropriately sized cuff. Three measures were taken two to three minutes apart, after the participant had been sitting for five minutes with their arm supported. The mean of the last two measurements was used in the analysis.

Outcome variable

We defined hypertension as systolic or diastolic BP ≥140 or ≥90 mm Hg, respectively or on anti-hypertensive medication. All hypertensive participants benefitted from free medical consultation and were given pre-printed referral letters to the Usokun or Ebubu Primary Health Centre or managed free of charge by a special team of doctors from the Community Medicine Department, University of Port Harcourt Teaching Hospital, Port Harcourt.

Exposure (determinant variable)

The main determinant variable of interest was oil pollution status of the place of residence of participants. This was categorised into two, whether the participants were from oil polluted area or not.

Potential confounders (control variables)

The following control variables were included in the study; they can be grouped into socio-demographics variables (age and sex of the participants, marital status, education attainment, and employment status), lifestyle factors (smoking status, drinking status, sleep duration, salt and fat-intake) and family history of hypertension.

Ethical considerations

The protocol of this study was reviewed and approved by the Biomedical and Scientific Research Ethics Sub-committee, University of Warwick, United Kingdom and the Research Ethics Committee of the University of Port Harcourt, Nigeria prior to entry to the community. Approval for the study was also obtained from the respective community leaders and council of elders before formal contact with the participants.

Statistical analysis

We used summary statistics to show the distribution of the main variables. The values were expressed as absolute number with percentages and mean with standard deviation for categorical and continuous variables respectively. We performed both univariate and multivariate logistic regression to examine the associations between participants' socio-demographic and other characteristics with the risk of hypertension. We considered variables for inclusion in the final multivariate model if they reached a moderate level of significance ($p<0.25$) or from the conceptual framework underlined in previous studies[23]. It is also possible that these demographic variables may alter not only an individual's overall predisposition towards hypertension, but also the association between living in polluted area and risk of developing hypertension. For instance, the strength of the relationship between living in polluted area and likelihood of developing hypertension may be different for men and women. To explore this possibility, we added interaction terms between living in polluted area and each of the determinants to the multivariate model.

The following regression diagnostics were used to assess the goodness-of-fit of the model and to choose the parsimonious model: the Hosmer-Lemeshow goodness-of-fit test, tolerance test for multicollinearity and link test to check for model specification error. We also

performed Receiver Operating Curves (ROC) (c-statistics) analyses to determine the predictive powers of the final multivariate model. The ROC curve plots the sensitivity of the model against 1 minus specificity for different cut-off points of the predicted probability of having hypertension. The greater the area under the ROC curve (upper limit =1), the better the model is at discriminating between hypertension cases. Results were presented as odds ratios (ORs) with 95% confidence intervals (CIs). All statistical analyses were carried out using Stata version 14 for Windows (Stata Corp, College Station, Texas). The significance tests were two-tailed and statistical significance was defined at the alpha level of 0.05. The paper is reported following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [24].

RESULTS

Summary statistics for the study participants

Descriptive statistics for study participants are shown in Table 1. More than one-third of participants were hypertensive (37.4%). Half of the participants were from oil polluted areas (51%) with preponderance of women (57.0%). The overall participants' mean age was 44.3 (standard deviation: 14.0), this differed between the polluted and unpolluted areas (44.7 vs 43.9), *P*=0.09. Most of the participants had secondary or higher education (61.8%) and currently working (81.7%). More than half of the participants were either overweight or obese (55.1%) and were mostly from the polluted area (62.3%). Most of the participants were non-smokers (83.3%) and 39.4% were reported to have never consumed alcohol. A significant percentage of participants in the unpolluted compared to polluted area reported moderate-to-high salt (74.9% vs 46.8%) and fat intake (59.8% vs 44.2%), *P*=0.01, respectively.

Factors associated with hypertension

The results of univariate and multivariate logistics regression models are presented in Table 2. In the univariate analysis, with the exception of sex of the participants, all the variables were statistically significantly associated with hypertension. However, in the multivariate model, the following factors remained statistically significant with hypertension: pollution status, age, family history of hypertension, body mass index, drinking status, sleep deprivation, level of physical activity and fat intake. Participants living in oil polluted areas were almost five times as likely to have developed hypertension (adjusted odds ratio [aOR] = 4.85, 95% CI 1.84 to 12.82], *P*=0.01).

All the interaction terms, except for age and education attainment were not statistically significant and were dropped from the final multivariate model (Table 3). For every ten years increase in the age of the participants, the odds of developing hypertension increased by 108% (aOR = 2.08, 95% CI 1.77 to 2.43). As shown in Figure 2, age modifies the association between pollution status and risk of hypertension, such that predicted probability of hypertension was significantly higher for participants' between the age of 20 and 40 years old. After the age of 65, the effect of pollution status tended to diminish.

Compared with participants with normal weight, overweight or obese participants were more likely to be hypertensive (aOR = 1.33, 95% CI 1.04 to 1.69). Moderate-to-heavy consumption of alcohol (aOR = 1.63, 95% CI 1.19 to 2.23), moderate-to-severe sleep deprivation (aOR = 4.27, 95% CI 3.14 to 5.81), sedentary behaviour (aOR = 3.09, 95% CI 1.66 to 5.76), moderate-to-high fat intake (aOR = 1.39, 95% CI 1.08 to 1.78) were statistically significantly associated with increased risk of hypertension.

Model fit statistics

None of the model fits results provided reasons for concern. For age interaction (Table 3), the average VIF was 1.36 (ranged: 1.06 to 2.19), since the VIF values and average VIF did not exceed 10 and 6 respectively, we concluded that there was no multi-collinearity problem, such that there is no perfect linear relationship between the determining variables, and the estimates for logistic regression models included these variables can be uniquely computed. The link test (Table 3) indicated that the model was specified correctly (p=0.14), which suggests that it is unlikely that we have omitted relevant variable(s) that could predict the hypertension risk. In addition, the Hosmer-Lemeshow test indicated that model II fits the data well (p=0.28). The area under the ROC curve for final multivariable model was 0.86, indicating relatively good predictive power and has reasonably good discriminatory ability. Other model fit results for other variables are shown in Table 3.

DISCUSSION

This study attempted to explore the prevalence of hypertension and associated risk factors in a highly polluted community, as compared to an unpolluted community, in the Niger Delta region of Nigeria.

In this study, the overall prevalence of hypertension was 37.4%. This was higher than previous review estimates for the whole country in general and the rural communities in particular[18, 19]. It may be argued that the difference could have been due to the participants mean age. However, the fact that our population is younger than study subjects in some urban environments of Niger Delta with lower prevalence[25, 26] makes this estimate more disturbing. We found that hypertension prevalence was significantly higher among participants in polluted environment (43.3%) compared to those in unpolluted area (31.2%), $P<0.01$.

Potential reason could be due to differences in exposure to pollution, or imbalance in socioeconomic and lifestyle factors between the two areas.

Among the potential confounding factors that were positively associated with hypertension, we noted that the proportion of those with no education, family history of hypertension, sleep deprivation, overweight and obesity were higher among the participants in the polluted area compared with those living in the unpolluted area, and that the proportion of participants with moderate-to-high intensity physical activity were lower in the polluted area compared to their counterparts in the unpolluted area. This may have contributed to the apparently higher prevalence of hypertension in the polluted area. After adjusting for these potential confounding factors, however, the risk of hypertension associated with residing in the polluted area remains (and indeed increased by more than 4-fold). The significant difference in risk attributed to polluted environment after adjustment add to our strong view that residents in polluted community have increased risk of hypertension irrespective of their BMI, socio-economic status, lifestyle and other predictors.

Environmental impact assessment (surface and ground water, land and ambient air evaluation) conducted in Ebubu community previously found very high percentage of polycyclic aromatic hydrocarbon (PAH), PM and heavy metals[1]. Evidence found that exposure to heavy metals such as Cadmium, Arsenic and Lead have been associated with hypertension[27-29]. Environmental pollutants such as heavy metals and PM trigger systemic inflammation and oxidative stress leading to hypertension through many pathophysiological mechanisms[4]. Therefore residents of polluted community like Ebubu are exposed not only to various air and soil pollutants but also to water and food pollutants especially due to bioaccumulation of heavy metals and other agents.

In addition to the above assessment, we also found interaction between age and oil pollution status and between education attainment and oil pollution status on overall predisposition

312 towards hypertension. Possible explanation here could be related to increased exposure to
313 pollutants among young adults and lack of awareness of the effect of pollution among those
314 with no education. These cohorts (20-40 years old) are highly mobile and often spend
315 substantial time engaging in work (semi-skilled and unskilled jobs in the oil and gas
316 facilities) and leisure in open air or water. Similar differential exposure to indoor pollution
317 and increased vulnerability has consistently been reported among women who spend between
318 3-7 hours indoors particularly in low and middle-income countries[30].
319
320 Biochemical evidence of exposure to dioxins such as TCDD (a major pollutant from oil and
321 gas pollution) was known to effect the sleep-wake cycle mediated through autonomic
322 nervous system dysfunction [4]. We found that participants in the polluted areas have
323 significantly less than 5 hours of sleep on average compared to their counterparts (28.0% vs
324 12.9%), $P<0.01$. Consistent with previous study evidence, the short sleep duration may have
325 increased the risk of hypertension in exposed subjects[16, 31.]
326
327 Apart from direct biochemical effect, other explanation to the increased hypertension burden
328 in the polluted area could be related to the broader socio-economic and psychosocial impact.
329 Oil pollution and influence of exploration activities of oil industry in the polluted community
330 may increase vulnerability and loss of livelihood and psychosocial stress[32,33]. For instance,
331 subsistent farmers that lost their small farm holding and source of drinking water and fishing
332 source to oil pollution may be affected adversely due to loss of occupational activities,
333 income and leisure. This increases exposure to chronic stress, possibly leading to disturbed
334 sleep patterns, lifestyles and behavioural changes, and reduced access to health care. The
335 results of these are inequality in health outcomes including hypertension.
336

Our study employed a relatively large sample size and rigorous methodology. However, the cross-sectional study design does not allow establishing temporality and causality for the observed associations. Caution therefore needs to be used in generalising the results because our study findings may not be applicable to the entire Niger delta region or communities that share a great diversity in socio-economic and environmental features. Other extrinsic factors such as healthcare access and availability remain potential important modifiers which must be factored in as well. In addition, the lack of detailed dietary history and our inability to assess other biomarkers, such as lipid profiles, may not only confound but also limit our understanding of the aetiology and influence of these to increased hypertension estimates. Therefore, the usefulness of pollution status in risk prediction of hypertension should be tested in large prospective studies which would take into account these potential confounders or modifiers. Our study has shown that environmental pollution may be useful for screening purposes to identify high risk population long before a diagnosis of hypertension is made and to target interventions appropriately.

Findings from this study suggest that oil and gas pollution be associated with a higher prevalence and risk of hypertension. This is disturbing and particularly so given that it is even higher than most urban estimates in majority of cities in the Niger Delta and Nigeria as a whole. While routine surveillance and management of hypertensive individuals remains an important public health priority, further epidemiological assessment of environmental pollutants in human tissues remains an important area for future research.

AUTHOR CONTRIBUTIONS

All authors contributed to the study concept and design. M. Ezejimofor, O. Maduka, A.Ezeabasili, A. Onwuchekwa, E. Asuquo and B**.** Ezejimofor contributed in data collection and entry. M. Ezejimofor, O. Uthman and A. Ezeabasili analysed the data. M. Ezejimofor wrote the first draft of the paper and all authors contributed in the manuscript correction and revision. The final manuscript was read and approved by all the authors for submission.

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DISCLOSURES

We declare that we have no conflicts of interest

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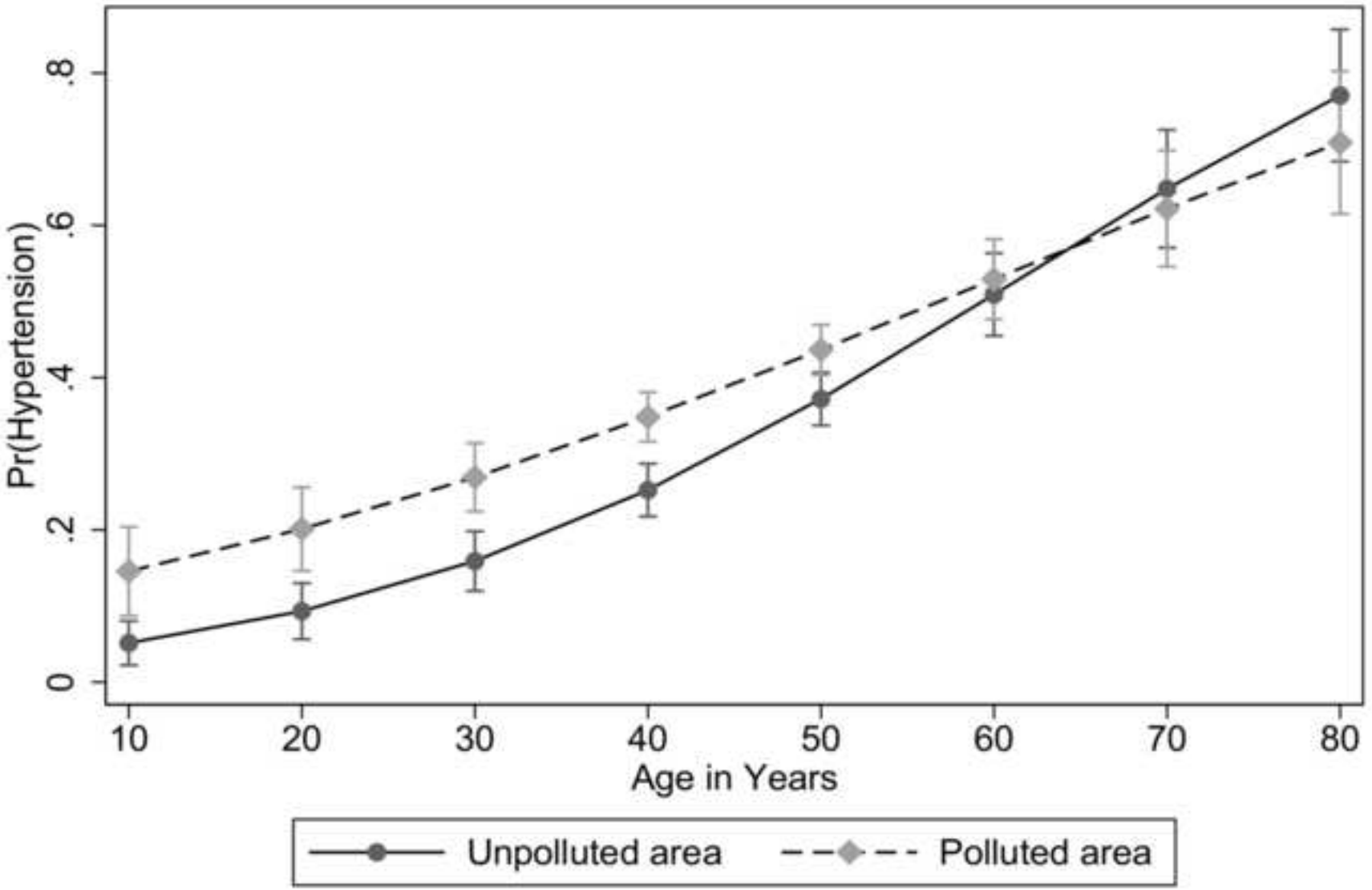
Figure 2. Predicted probability of hypertension for all ages by oil pollution status



BW Figure

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Figure 3. Receiver operating characteristics curves for final multivariable model

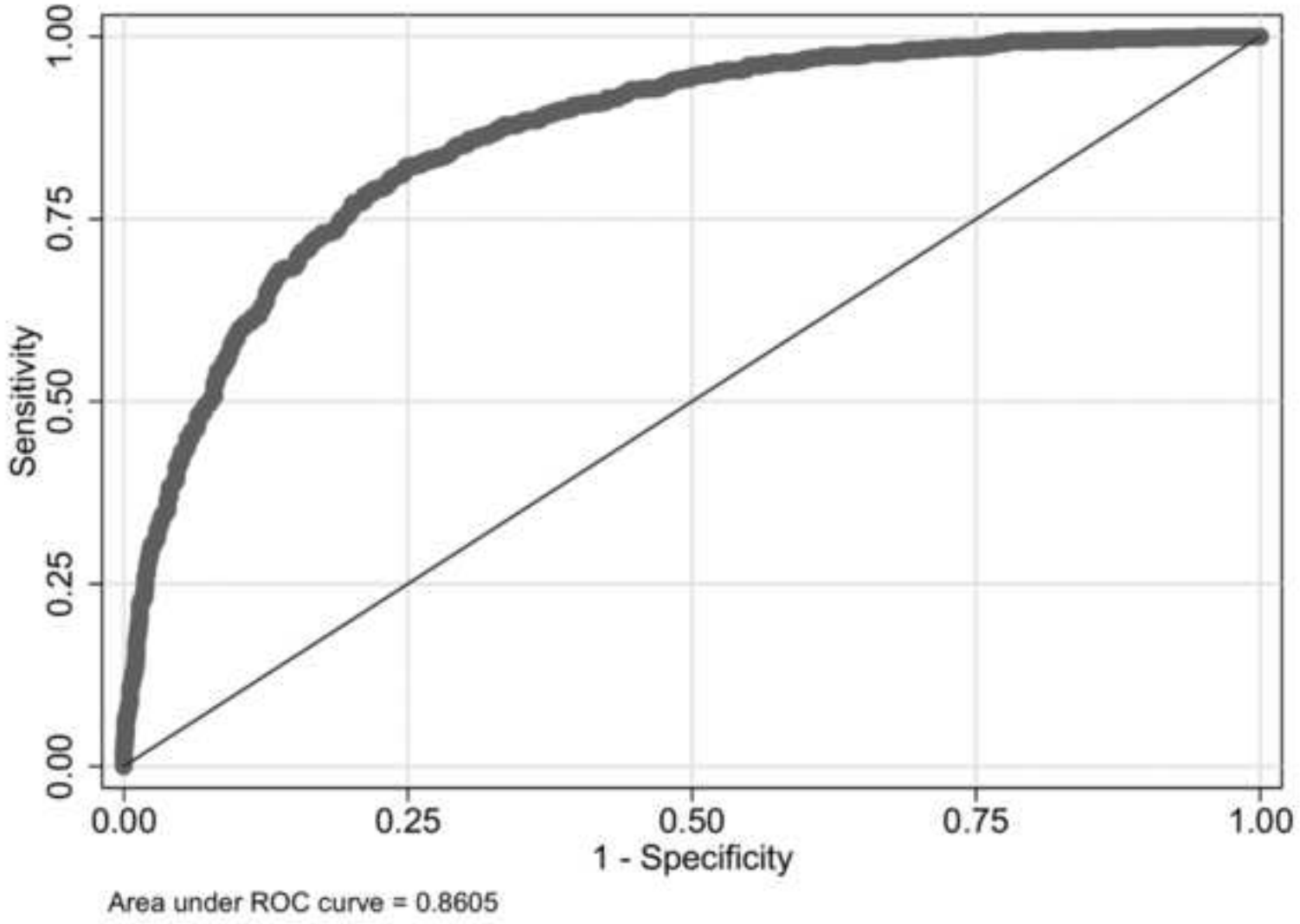

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Figure 1. Map of the study local councils of the study areas showing oil and gas production sites investigated by UNEP[1]

Table

Table 1. Characteristics of the study population by oil pollution status

Variable	Total (n=2028)	Polluted area (n=1036)	Non-polluted area (n=992)	P-value[a]
Mean age (SD)	44.3 (14.0)	44.7 (13.3)	43.9 (14.7)	0.09
Gender (%)				
Male	871 (43.0)	417(40.0)	454 (46.0)	
Female	1157 (57.0)	619(60.0)	538(54.0)	0.01
Hypertensive[b] (%)				
No	1270 (62.6)	587(56.7)	683(68.9)	
Yes	758 (37.4)	449(43.3)	309(31.2)	0.01
Family history of hypertension (%)				
No	1724 (85.0)	847(81.8)	877(88.4)	
Yes	304 (15.0)	189(18.2)	115(11.6)	0.01
Marital Status (%)				
Never married	544 (26.8)	235(22.7)	309(31.2)	
Ever married	1483 (73.2)	800(77.3)	683(68.9)	0.01
Education attainment (%)				
No education (<6 years)	362 (17.9)	221(21.3)	141(14.2)	
Primary (6-11 years)	411 (20.2)	178(17.2)	233(23.5)	
Secondary/higher (≥ 12 Years)	1255 (61.8)	637(61.5)	618(62.3)	0.01
Employment status (%)				
Unemployed	371 (18.3)	165(15.9)	206(20.8)	
Presently working	1657 (81.7)	871(84.1)	786(79.2)	0.005
Body mass index (%)				
Underweight	35 (1.7)	17(1.6)	18(1.8)	
Normal	876 (43.2)	374(36.1)	502(50.6)	
Overweight/Obese	1117 (55.1)	645(62.3)	472(47.6)	0.01

Smoking status (%)				
Non-smoker	1689 (83.3)	925(89.3)	764(77.0)	
Ever smoker	339 (16.7)	111(10.7)	228(23.0)	0.01
Drinking status (%)				
Non-drinker	799 (39.4)	523(50.5)	276(27.8)	
Mild drinker	656 (32.3)	309(29.8)	347(35.0)	
Moderate-to-heavy drinker	573 (28.3)	204(19.7)	369(37.2)	0.01
Sleep deprivation (%)				
No	1326 (65.4)	564(54.4)	762(76.9)	
Mild	283 (14.0)	182(17.6)	101(10.2)	
Moderate-to-severe	418 (20.6)	290(28.0)	128(12.9)	0.01
Physical activity (%)				
Sedentary	79 (3.9)	33(3.2)	46(4.6)	
Low intensity	624 (30.8)	346(33.4)	278(28.0)	
Moderate-to-high intensity	1325 (65.3)	657(63.4)	668(67.3)	0.01
Salt intake (%)				
Low	800 (39.4)	551(53.2)	249(25.1)	
Moderate-to-high	1228 (60.6)	485(46.8)	743(74.9)	0.01
Fat intake (%)				
Low	977 (48.2)	578(55.8)	399(40.2)	
Moderate-to-high	1051 (51.8)	458(44.2)	593(59.8)	0.01

Data are expressed as mean (standard deviation) or as percentages.

[a]P values for comparison between polluted and non-polluted areas.

[b]Defined as blood pressure ≥ 140/90 mmHg or on antihypertensive medication.

Table

Table 2. Unadjusted and adjusted odd ratio of hypertension for selected risk factors

Variable	Unadjusted model		Adjusted model	
	Odds ratio (95% CI)	p-value	Odds ratio (95% CI)	p-value
Main effects				
Polluted (vs unpolluted) area	1.69 (1.41 to 2.03)	0.01	4.85 (1.84 to 12.82)	0.01
Age (per 10 years increase)	2.27 (2.09 to 2.47)	0.01	2.08 (1.77 to 2.43)	0.01
Female (vs male)	1.06 (0.88 to 1.27)	0.54	Not included	
Family history of hypertension (vs no)	3.17 (2.46 to 4.08)	0.01	2.41 (1.75 to 3.32)	0.01
Ever (vs. never) married	6.21 (4.72 to 8.18)	0.01	1.62 (1.12 to 2.35)	0.01
Education attainment				
No education	2.43 (1.92 to 3.09)	0.01	1.34 (0.82 to 0.96)	0.002
Primary	1.34 (1.06 to 1.69)	0.01	0.90 (0.67 to 1.23)	0.52
Secondary or higher	1 (reference)		1 (reference)	
Currently employed (vs unemployed)	2.93 (2.23 to 3.86)	0.01	1.43 (0.95 to 2.15)	0.09
Body mass index				
Underweight	0.47 (0.19 to 1.14)	0.09	0.65 (0.22 to 1.89)	0.43
Normal	1 (reference)		1 (reference)	
Overweight/Obese	1.72 (1.43 to 2.08)	0.01	1.33 (1.04 to 1.69)	0.02
Ever- (vs. non-) smoker	1.61 (1.27 to 2.03)	0.01	1.28 (0.92 to 1.77)	0.14
Drinking status				
Non-drinker	1 (reference)		1 (reference)	
Mild drinker	0.67 (0.53 to 0.83)	0.01	0.76 (0.57 to 1.02)	0.06

Moderate-to-heavy drinker	1.80 (1.44 to 2.23)	0.01	1.63 (1.19 to 2.23)	0.02
Sleep deprivation				
No	1 (reference)		1 (reference)	
Mild	3.89 (2.98 to 5.07)	0.01	1.97 (1.44 to 2.68)	0.01
Moderate-to-severe	10.55 (8.16 to 13.64)	0.01	4.27 (3.14 to 5.81)	0.01
Physical activity				
Sedentary	6.50 (3.94 to 10.72)	0.01	3.09 (1.66 to 5.76)	0.01
Low intensity	3.21 (2.64 to 3.92)	0.01	2.81 (2.20 to 3.60)	0.01
Moderate-to-high intensity	1 (reference)		1 (reference)	
Moderate-to-high (vs. low) salt intake	1.50 (1.24 to 1.80)	0.01	1.10 (0.84 to 1.43)	0.50
Moderate-to-high (vs. low) fat intake	1.42 (1.18 to 1.70)	0.01	1.39 (1.08 to 1.78)	0.01

Table

Table 3. Interaction of demographic variables and pollution status

Interaction effects	Odds ratio (95% CI)	p-value
Polluted (vs unpolluted) area #Age	0.78 (0.64 to 0.95)	0.01
Model fit statistics		
Area under ROC[1] curve		0.86
Lemeshow test (p-value)		0.28
Link test (p-value)		0.14
Collinearity diagnostic (mean VIF[2])		1.36
Polluted (vs unpolluted) area # Education attainment	0.39 (0.22 to 0.72)	0.02
Model fit Statistics		
Area under ROC[1] curve		0.86
Lemeshow test (p-value)		0.32
Link test (p-value)		0.09
Collinearity diagnostic (mean VIF[2])		0.20
Polluted (vs unpolluted) area # Sex	Not included	
Polluted (vs unpolluted) area # Marital status	0.88(0.45 to 1.69	0.69
Polluted (vs unpolluted) area # Employment status	0.59(0.26 to 1.29)	0.18

[1] Receiver Operating Characteristic

[2] Variance Inflation Factor