

# Northumbria Research Link

Citation: Ezejimofor, Martinsixtus, Uthman, Olalekan, Maduka, Omosivie, Ezeabasili, Aloysius, Onwuchekwa, Arthur, Ezejimofor, Benedeth, Asuquo, Eme, Chen, Yen-Fu, Stranges, Saverio and Kandala, Ngianga-Bakwin (2016) The Burden of Hypertension in an Oil- and Gas-Polluted Environment: A Comparative Cross-Sectional Study. American Journal of Hypertension, 29 (8). pp. 925-933. ISSN 0895-7061

Published by: Oxford University Press

URL: <http://dx.doi.org/10.1093/ajh/hpw009> <<http://dx.doi.org/10.1093/ajh/hpw009>>

This version was downloaded from Northumbria Research Link:  
<http://nrl.northumbria.ac.uk/26102/>

Northumbria University has developed Northumbria Research Link (NRL) to enable users to access the University's research output. Copyright © and moral rights for items on NRL are retained by the individual author(s) and/or other copyright owners. Single copies of full items can be reproduced, displayed or performed, and given to third parties in any format or medium for personal research or study, educational, or not-for-profit purposes without prior permission or charge, provided the authors, title and full bibliographic details are given, as well as a hyperlink and/or URL to the original metadata page. The content must not be changed in any way. Full items must not be sold commercially in any format or medium without formal permission of the copyright holder. The full policy is available online: <http://nrl.northumbria.ac.uk/policies.html>

This document may differ from the final, published version of the research and has been made available online in accordance with publisher policies. To read and/or cite from the published version of the research, please visit the publisher's website (a subscription may be required.)

[www.northumbria.ac.uk/nrl](http://www.northumbria.ac.uk/nrl)



174 **Authors' reply:** We have now uploaded publication quality figures.

175

176

Text word count: 2989

177

Abstract word count: 250

178

No. of references: 33

179

No. of figures: 3

180

No. of Tables: 3

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

183

184 Martinsixtus C. Ezejimofor MPH<sup>a</sup>; Olalekan A.Uthman PhD<sup>a,b,c</sup>; Omosivie Maduka MB, BS  
185 FMCPH<sup>d</sup>; Aloysius C. Ezeabasili PhD<sup>e</sup>; Arthur C. Onwuchekwa MB,BS, FCMP<sup>f</sup>; Benedeth  
186 C.Ezejimofor MD, MPH<sup>a</sup>; Eme Asuquo MPH<sup>d</sup>; Yen-Fu Chen PhD<sup>a,b</sup>; Saverio Stranges MD  
187 PhD<sup>a,g</sup>; Ngianga-Bakwin Kandala PhD<sup>a,g,h</sup>

188 <sup>a</sup>Division of Health Sciences, University of Warwick Medical School, Coventry, CV4 7AL,  
189 UK

190 <sup>b</sup>Warwick-Centre for Applied Health Research and Delivery (WCAHRD), Division of health  
191 Sciences, University of Warwick Medical School, Coventry, UK

192 <sup>c</sup>Liverpool School of Tropical Medicine, International Health Group, Liverpool, UK

193 <sup>d</sup>Department of Preventive and Social Medicine, University of Port Harcourt, Port Harcourt,  
194 Nigeria

195 <sup>e</sup>School of the Built Environment, University of Salford, M5 4WT, UK

196 <sup>f</sup>Department of Internal Medicine, University of Port Harcourt, Port Harcourt, Nigeria

197 <sup>g</sup>Department of Population Health, Luxembourg Institute of Health (LIH), 1A-B, rue Thomas  
198 Edison, L-1445 Strassen, Luxembourg

199 <sup>h</sup>Northumbria University, Department of Mathematics and Information sciences, Faculty of  
200 Engineering and Environment, Newcastle upon Tyne, NE1 8ST, United Kingdom.

201

202 **Corresponding Author**

203 Martinsixtus C. Ezejimofor

204 Division of Health Sciences

205 University of Warwick Medical School

206 Coventry, CV4 7AL, UK

207 Tel: +44(0)7944420045

208 Fax: +44(0)24 76573163

209 Email: [m.c.ezejimofor@warwick.ac.uk](mailto:m.c.ezejimofor@warwick.ac.uk)

210 Background

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

214 Methods

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

223 Results

224 More than one-third of participants were hypertensive (37.4%). Half of the participants were  
225 from oil polluted areas (51%). Only 15% of participants reported family history of  
226 hypertension. In the adjusted model, participants living in oil polluted areas were almost five  
227 times as likely to have developed hypertension (adjusted odds ratio [aOR] = 4.85, 95% CI  
228 1.84 to 12.82) compared to participants in unpolluted areas. Age modifies the association  
229 between pollution status and risk of hypertension. For every ten years increase in the age of  
230 the participants, the odds of developing hypertension increased by 108% (aOR = 2.08, 95%  
231 CI 1.77 to 2.43).

232

233 Conclusion

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

236

237

238

239

240

241

242

243

244

245

246

247

248

249

250

251

252

253

254

255

256

257

258

## 259 INTRODUCTION

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

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

276 Gas flaring and oil refining may affect the sleep-wake-cycle in healthy individuals<sup>14</sup>.

277 Prolonged exposure to dioxins particularly 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), a  
278 major by-product of gas flaring and crude oil refining can cause neurological symptoms  
279 including sleep disturbances, neuralgia and severe headache<sup>15</sup>. Evidence found that sleep  
280 deprivation is positively associated with increased cardiovascular risk including  
281 hypertension<sup>16,17</sup>.

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

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

290

## 291 **MATERIALS AND METHODS**

### 292 **Study setting and population**

293 The study was carried out among residents exposed to oil and gas pollution in a dominantly  
294 oil and gas polluted community, and non-exposed residents from another community without  
295 any oil and gas exploration or related activities (Figure 1). The two communities have the  
296 same socioeconomic and cultural features in Eleme and Degema local Government area in  
297 Rivers State. **However, they differ remarkably in terms of environmental pollution and**  
298 **exposure level largely due to oil exploration and gas flaring and allied industrial activities.**

299 The communities are about 60km apart with an estimated population of 30,580. Ebubu is a  
300 rural farming settlement with sparse social infrastructure. **The community has huge functional**  
301 **oilfields and gas flaring sites in addition to a network of oil pipelines.** It is a highly polluted  
302 community with history of oil and gas exploration activities by Shell Petroleum Development  
303 Company<sup>1</sup>. Usokun on the other hand is a pristine rural settlement, a non-oil and gas  
304 producing (unpolluted) community. This community constitutes the control population. **It is**  
305 **an island, circled by Sambreiro River (an outlet of the Niger) with only one access road**

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

308

### 309 **Study design and sample size**

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

317

### 318 **Data collection**

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

331 participants wearing no hat and shoes. Participants' body weight was measured to the nearest  
332 0.1 kg while they were dressed in light clothing without footwear using calibrated digital  
333 weighing scale (SECA 877, GmbH, Hamburg, Germany). BMI was calculated as weight in  
334 kilograms divided by the square of height in meters ( $\text{kg}/\text{m}^2$ ). Blood pressure was measured  
335 using an automated and validated<sup>21,22</sup> upper-arm digital blood pressure monitor (Omron M6  
336 HEM-7001-E, Birmingham, UK) with an appropriately sized cuff. Three measures were  
337 taken two to three minutes apart, after the participant had been sitting for five minutes with  
338 their arm supported. The mean of the last two measurements was used in the analysis.

339

#### 340 **Outcome variable**

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

346

#### 347 **Exposure (determinant variable)**

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

351

#### 352 **Potential confounders (control variables)**

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



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

357

### 358 **Ethical considerations**

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

364

### 365 **Statistical analysis**

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

379

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

393

## 394 **RESULTS**

### 395 **Summary statistics for the study participants**

396 Descriptive statistics for study participants are shown in Table 1. More than one-third of  
397 participants were hypertensive (37.4%). Half of the participants were from oil polluted areas  
398 (51%) with preponderance of women (57.0%). The overall participants' mean age was 44.3  
399 (standard deviation: 14.0), this differed between the polluted and unpolluted areas (44.7 vs  
400 43.9), P=0.09. Most of the participants had secondary or higher education (61.8%) and  
401 currently working (81.7%). More than half of the participants were either overweight or  
402 obese (55.1%) and were mostly from the polluted area (62.3%). Most of the participants were  
403 non-smokers (83.3%) and 39.4% were reported to have never consumed alcohol. A  
404 significant percentage of participants in the unpolluted compared to polluted area reported

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

407

#### 408 **Factors associated with hypertension**

409 The results of univariate and multivariate logistics regression models are presented in Table  
410 2. In the univariate analysis, with the exception of sex of the participants, all the variables  
411 were statistically significantly associated with hypertension. However, in the multivariate  
412 model, the following factors remained statistically significant with hypertension: pollution  
413 status, age, family history of hypertension, body mass index, drinking status, sleep  
414 deprivation, level of physical activity and fat intake. Participants living in oil polluted areas  
415 were almost five times as likely to have developed hypertension (adjusted odds ratio [aOR] =  
416 4.85, 95% CI 1.84 to 12.82],  $P=0.01$ ).

417 All the interaction terms, except for age and education attainment were not statistically  
418 significant and were dropped from the final multivariate model (Table 3). For every ten years  
419 increase in the age of the participants, the odds of developing hypertension increased by  
420 108% (aOR = 2.08, 95% CI 1.77 to 2.43). As shown in Figure 2, age modifies the association  
421 between pollution status and risk of hypertension, such that predicted probability of  
422 hypertension was significantly higher for participants' between the age of 20 and 40 years  
423 old. After the age of 65, the effect of pollution status tended to diminish.

424 Compared with participants with normal weight, overweight or obese participants were more  
425 likely to be hypertensive (aOR = 1.33, 95% CI 1.04 to 1.69). Moderate-to-heavy  
426 consumption of alcohol (aOR = 1.63, 95% CI 1.19 to 2.23), moderate-to-severe sleep  
427 deprivation (aOR = 4.27, 95% CI 3.14 to 5.81), sedentary behaviour (aOR = 3.09, 95% CI  
428 1.66 to 5.76), moderate-to-high fat intake (aOR = 1.39, 95% CI 1.08 to 1.78) were  
429 statistically significantly associated with increased risk of hypertension.

### 430 **Model fit statistics**

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

442

### 443 **DISCUSSION**

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

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

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

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

468

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

478 In addition to the above assessment, we also found interaction between age and oil pollution  
479 status and between education attainment and oil pollution status on overall predisposition  
480 towards hypertension. Possible explanation here could be related to increased exposure to  
481 pollutants among young adults and lack of awareness of the effect of pollution among those  
482 with no education. These cohorts (20-40 years old) are highly mobile and often spend  
483 substantial time engaging in work (semi-skilled and unskilled jobs in the oil and gas  
484 facilities) and leisure in open air or water. Similar differential exposure to indoor pollution  
485 and increased vulnerability has consistently been reported among women who spend between  
486 3-7 hours indoors particularly in low and middle-income countries<sup>30</sup>.

487

488 Biochemical evidence of exposure to dioxins such as TCDD (a major pollutant from oil and  
489 gas pollution) was known to effect the sleep-wake cycle mediated through autonomic  
490 nervous system dysfunction<sup>4</sup>. We found that participants in the polluted areas have  
491 significantly less than 5 hours of sleep on average compared to their counterparts (28.0% vs  
492 12.9%),  $P < 0.01$ . Consistent with previous study evidence, the short sleep duration may have  
493 increased the risk of hypertension in exposed subjects<sup>16, 31</sup>.

494

495 Apart from direct biochemical effect, other explanation to the increased hypertension burden  
496 in the polluted area could be related to the broader socio-economic and psychosocial impact.

497 Oil pollution and influence of exploration activities of oil industry in the polluted community  
498 may increase vulnerability and loss of livelihood and psychosocial stress<sup>32,33</sup>. For instance,  
499 subsistent farmers that lost their small farm holding and source of drinking water and fishing  
500 source to oil pollution may be affected adversely due to loss of occupational activities,  
501 income and leisure. This increases exposure to chronic stress, possibly leading to disturbed  
502 sleep patterns, lifestyles and behavioural changes, and reduced access to health care. The  
503 results of these are inequality in health outcomes including hypertension.

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

518

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

525

526

527

528

529

530 **AUTHOR CONTRIBUTIONS**

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

536

537 **STUDY FUNDING**

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

542

543 **DISCLOSURES**

544 We declare that we have no conflicts of interest

545

546

547

548

549

550

551

552

553

554

555

556

557

558

559

560

561

562



563

## References

564

565 1. United Nations Environment Programme. Environmental Assessment of Ogoniland 2011.  
566 <http://www.unep.org/disastersandconflicts/CountryOperations/Nigeria/EnvironmentalAssessm>  
567 [entofOgonilandreport/tabid/54419/Default.aspx](http://www.unep.org/disastersandconflicts/CountryOperations/Nigeria/EnvironmentalAssessmentofOgonilandreport/tabid/54419/Default.aspx), p. 1-257. Accessed on 20 July 2015.

568 2. Kadafa AA. Environmental impacts of oil exploration and exploitation in the Niger Delta of  
569 Nigeria. *Glo J Sci Front Res* 2012;12:19-28.

570 3. Ite AE, Ibok UJ. Gas flaring and venting associated with petroleum exploration and production  
571 in the Nigeria's Niger Delta. *Am J Environ Protec* 2013;1:70-77.

572 4. Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F,  
573 Hong Y, Luepker RV, Mittleman MA. Particulate matter air pollution and cardiovascular  
574 disease an update to the scientific statement from the American Heart Association. *Circulation*  
575 2010; 121:2331-2378.

576 5. Lodovici M, Bigagli E. Oxidative stress and air pollution exposure. *J Toxicol.* 2011;2011.

577 6. Liao D, Creason J, Shy C, Williams R, Watts R, Zweidinger R. Daily variation of particulate  
578 air pollution and poor cardiac autonomic control in the elderly. *Environ Health Perspec*  
579 1999;107:521.

580 7. Pekkanen J, Brunner E, Anderson H, Tiittanen P, Atkinson R. Daily concentrations of air  
581 pollution and plasma fibrinogen in London. *Occup Environ Med.* 2000;57:818-822.

582 8. Brook RD, Rajagopalan S. Chronic Air Pollution Exposure and Endothelial Dysfunction:  
583 What You Can't See—Can Harm You. *J Am Coll Cardiol* 2012;60:2167-2169.

584 9. Chuang K-J, Yan Y-H, Cheng T-J. Effect of air pollution on blood pressure, blood lipids, and  
585 blood sugar: a population-based approach. *J Occup Environ Med* 2010;52:258-262.

586 10. Chuang K-J, Yan Y-H, Chiu S-Y, Cheng T-J. Long-term air pollution exposure and risk  
587 factors for cardiovascular diseases among the elderly in Taiwan. *Occup Environ Med* 2010;  
588 68:64-68

589 11. Dong G-H, Qian ZM, Xaverius PK, Trevathan E, Maalouf S, Parker J, Yang L, Liu M-M,  
590 Wang D, Ren W-H. Association between long-term air pollution and increased blood pressure  
591 and hypertension in China. *Hypertension* 2013; 61:578-584.

592 12. Coogan PF, White LF, Jerrett M, Brook RD, Su JG, Seto E, Burnett R, Palmer JR, Rosenberg  
593 L, Air pollution and incidence of hypertension and diabetes mellitus in black women living in  
594 Los Angeles. *Circulation* 2012; 125:767-772.

595 13. Pope CA, Burnett RT, Thurston GD, Thun MJ, Calle, EE, Krewski D, Godleski JJ.  
596 Cardiovascular mortality and long-term exposure to particulate air pollution epidemiological  
597 evidence of general pathophysiological pathways of disease. *Circulation* 2004;109:71-77.

598 14. Gobo A, Richard G, Ubong I. Health Impact of Gas Flares on Igwuruta/Umuechem  
599 Communities in Rivers State. *J Appl Sci Environ Mgt* 2009;13

- 600 15. Neuberger M, Kundi M, Jäger R. Chloracne and morbidity after dioxin exposure (preliminary  
601 results). *Toxicol lett* 1998;96:347-350.
- 602 16. Stranges S, Dorn JM, Cappuccio FP, Donahue RP, Rafalson LB, Hovey KM, Freudenheim JL,  
603 Kandala, N-B, Miller MA, Trevisan M. A population-based study of reduced sleep duration  
604 and hypertension: the strongest association may be in premenopausal women. *J Hypertens*  
605 2010; 28:896-902.
- 606 17. Dettoni JL, Consolim-Colombo FM, Drager LF, Rubira MC, de Souza SBPC, Irigoyen MC,  
607 Mostarda C, Borile S, Krieger EM, Moreno H. Cardiovascular effects of partial sleep  
608 deprivation in healthy volunteers. *J Appl Physiol* 2012; 113:232-236.
- 609 18. Adeloye D, Basquill C, Aderemi AV, Thompson JY, Obi FA. An estimate of the prevalence of  
610 hypertension in Nigeria: a systematic review and meta-analysis. *J Hypertens* 2015;33:230-242.
- 611 19. Ogah O, Okpechi I, Chukwuonye I, Akinyemi J, Onwubere B, Falase A, Stewart S, Sliwa K.  
612 Blood pressure, prevalence of hypertension and hypertension related complications in Nigerian  
613 Africans: A review. *World J Cardiol* 2012;4:327-340
- 614 20. Onwuchekwa AC, Mezie-Okoye MM, Babatunde S. Prevalence of hypertension in Kegbara-  
615 Dere, a rural community in the Niger Delta region, Nigeria. *Ethn Dis* 2012;22:340-346.
- 616 21. Altunkan S, Ilman N, Kayatürk N, Altunkan E. Validation of the Omron M6 (HEM-7001-E)  
617 upper-arm blood pressure measuring device according to the International Protocol in adults  
618 and obese adults. *Blood Press Monit* 2007;12:219-225.
- 619 22. Altunkan S, Ilman N, Altunkan E. Validation of the Omron M6 (HEM-7001-E) upper arm  
620 blood pressure measuring device according to the International Protocol in elderly patients.  
621 *Blood Press Monit* 2008;13:117-122.
- 622 23. Mickey J, Greenland S. A study of the impact of confounder-selection criteria on effect  
623 estimation. *Am J Epidemiol* 1989;129:125-13
- 624 24. Vandembroucke JP, Von EE, Altman DG, Gøtzsche PC, Mulrow CD, Pocock SJ, Poole C,  
625 Schlesselman JJ, Egger M. Strengthening the Reporting of Observational Studies in  
626 *Epidemiology (STROBE): explanation and elaboration. Ann Intern Med* 2007; 147:163-194.
- 627 25. Oguoma VM, Nwose EU, Skinner TC, Digban KA, Onyia IC, Richards RS. Prevalence of  
628 cardiovascular disease risk factors among a Nigerian adult population: relationship with  
629 income level and accessibility to CVD risks screening. *BMC Public Health* 2015;15:397.
- 630 26. Ordinioha B. The prevalence of hypertension and its modifiable risk factors among lecturers of  
631 a medical school in Port Harcourt, south-south Nigeria: implications for control effort. *Niger J*  
632 *Clin Pract* 2013;16:1-11.
- 633 27. Rahman M, Tondel M, Ahmad SA, Chowdhury IA, Faruquee MH, Axelson O. Hypertension  
634 and arsenic exposure in Bangladesh. *Hypertension* 1999;33:74-78.

- 635 28. Lu Y, Liu X, Deng Q, Duan Y, Dai H, Li Y, Xiao T, Ning X, Fan J, Zhou L . Continuous Lead  
636 Exposure Increases Blood Pressure but Does Not Alter Kidney Function in Adults 20-44 Years  
637 of Age in a Lead-Polluted Region of China. *Kidney Blood Press Res* 2015; 40:207-214.
- 638 29. Caciari T, Sancini A, Fioravanti M, Capozzella A, Casale T, Montuori L, Fiaschetti M,  
639 Schifano M, Andreozzi G. Cadmium and hypertension in exposed workers: A meta-analysis.  
640 *Int J Occup Med Environ Health* 2013; 26:440-456.
- 641 30. Bruce N, Rogelio P-P, Rachel A. The health effects of indoor air pollution exposure in  
642 developing countries. Geneva: World Health Organization 2002; 11  
643 <http://www.bioenergylists.org/stovesdoc/Environment/WHO/OEH02.5.pdf> p. 1-40. Accessed  
644 on 20 November, 2015.
- 645 31. Cappuccio FP, Stranges S, Kandala N-B Miller MA, Taggart FM, Kumari M, Ferrie JE,  
646 Shipley MJ, Brunner EJ, Marmot MG. Gender-specific associations of short sleep duration  
647 with prevalent and incident hypertension the Whitehall II study. *Hypertension* 2007; 50:693-  
648 700.
- 649 32. Crighton EJ, Elliott SJ, Van Der Meer J, Small I, Upshur R. Impacts of an environmental  
650 disaster on psychosocial health and well-being in Karakalpakstan. *Soc Sci Med* 56, 551-567.
- 651 33. Couch SR, Coles CJ. Community stress, psychosocial hazards, and EPA decision-making in  
652 communities impacted by chronic technological disasters. *Am J Public Health* 2011;101:140-  
653 148.

654

655

656

657

658

659

660

661

662

663

664

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

<b>Variable</b>	<b>Total</b>	<b>Polluted area</b>	<b>Non-polluted area</b>	<b>P-value<sup>a</sup></b>
	<b>(n=2028)</b>	<b>(n=1036)</b>	<b>(n=992)</b>	
<b>Mean age (SD)</b>	44.3 (14.0)	44.7 (13.3)	43.9 (14.7)	0.09
<b>Gender (%)</b>				
Male	871 (43.0)	417(40.0)	454 (46.0)	
Female	1157 (57.0)	619(60.0)	538(54.0)	0.01
<b>Hypertensive<sup>b</sup> (%)</b>				
No	1270 (62.6)	587(56.7)	683(68.9)	
Yes	758 (37.4)	449(43.3)	309(31.2)	0.01
<b>Family history of hypertension (%)</b>				
No	1724 (85.0)	847(81.8)	877(88.4)	
Yes	304 (15.0)	189(18.2)	115(11.6)	0.01
<b>Marital Status (%)</b>				
Never married	544 (26.8)	235(22.7)	309(31.2)	
Ever married	1483 (73.2)	800(77.3)	683(68.9)	0.01
<b>Education attainment (%)</b>				
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 ( $\geq$ 12 Years)	1255 (61.8)	637(61.5)	618(62.3)	0.01
<b>Employment status (%)</b>				
Unemployed	371 (18.3)	165(15.9)	206(20.8)	
Presently working	1657 (81.7)	871(84.1)	786(79.2)	0.005
<b>Body mass index (%)</b>				
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

---

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

667 <sup>a</sup>P values for comparison between polluted and non-polluted areas.

668 <sup>b</sup>Defined as blood pressure  $\geq$  140/90 mmHg or on antihypertensive medication.

669

670

671

672

673

674

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	Odds ratio (95% CI)	p-value
<b>Main effects</b>				
<b>Polluted (vs unpolluted) area</b>	1.69 (1.41 to 2.03)	0.01	4.85 (1.84 to 12.82)	0.01
<b>Age (per 10 years increase)</b>	2.27 (2.09 to 2.47)	0.01	2.08 (1.77 to 2.43)	0.01
<b>Female (vs male)</b>	1.06 (0.88 to 1.27)	0.54	Not included	
<b>Family history of hypertension (vs no)</b>	3.17 (2.46 to 4.08)	0.01	2.41 (1.75 to 3.32)	0.01
<b>Ever (vs. never) married</b>	6.21 (4.72 to 8.18)	0.01	1.62 (1.12 to 2.35)	0.01
<b>Education attainment</b>				
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)	
<b>Currently employed (vs unemployed)</b>	2.93 (2.23 to 3.86)	0.01	1.43 (0.95 to 2.15)	0.09
<b>Body mass index</b>				
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
<b>Ever- (vs. non-) smoker</b>	1.61 (1.27 to 2.03)	0.01	1.28 (0.92 to 1.77)	0.14
<b>Drinking status</b>				
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
<b>Sleep deprivation</b>				
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
<b>Physical activity</b>				
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)	
<b>Moderate-to-high (vs. low) salt intake</b>	1.50 (1.24 to 1.80)	0.01	1.10 (0.84 to 1.43)	0.50
<b>Moderate-to-high (vs. low) fat intake</b>	1.42 (1.18 to 1.70)	0.01	1.39 (1.08 to 1.78)	0.01

676

677

678

679

680

681

682

683

684

685

686

687

688

689

690

691

692

693

**Table 3. Interaction of demographic variables and pollution status**

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

695 <sup>1</sup> Receiver Operating Characteristic696 <sup>2</sup> Variance Inflation Factor

697

698

699

700

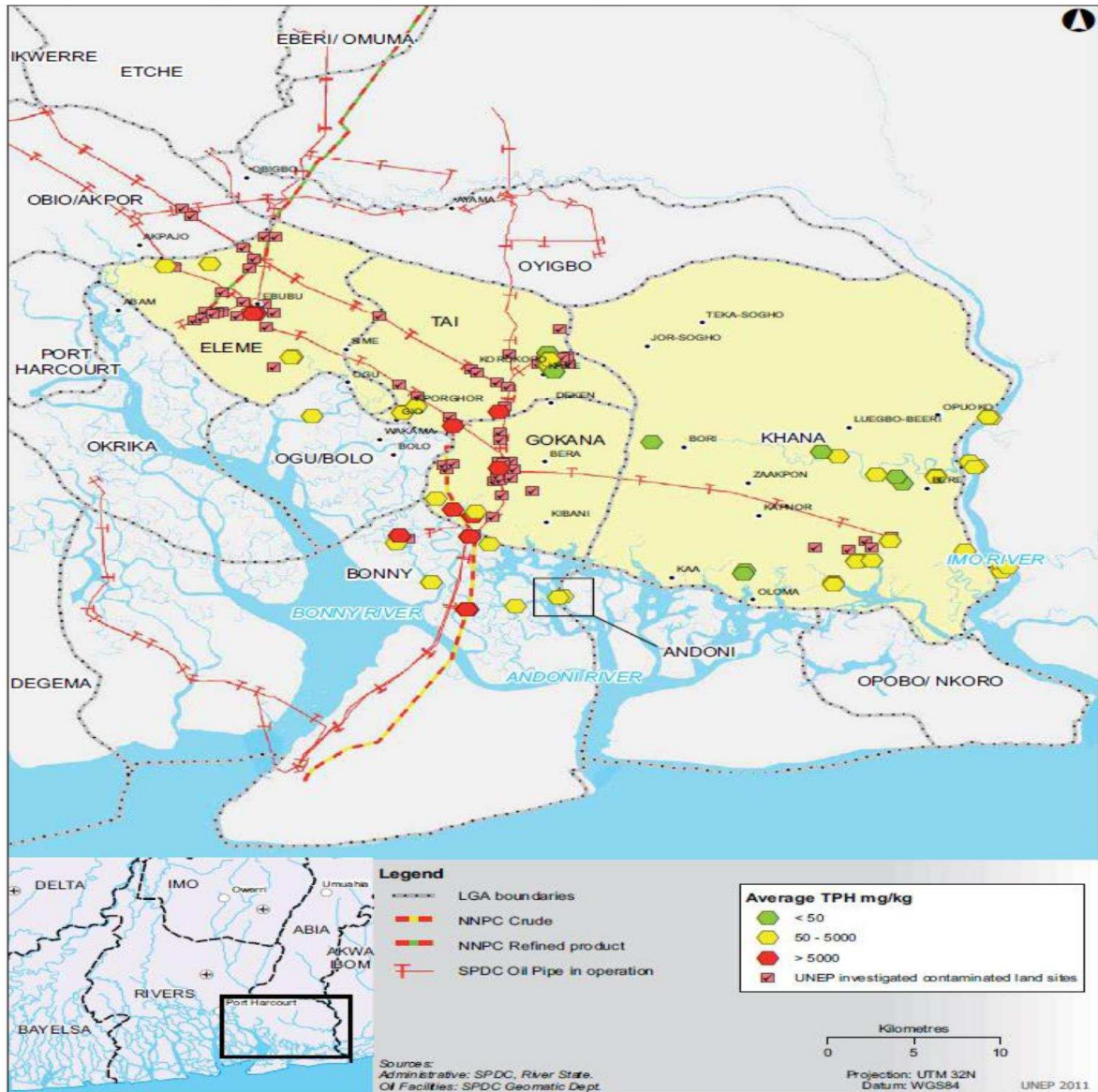
701

702

703



704 **Figure 1. Map of the study local councils of the study areas showing oil and gas**  
 705 **production sites investigated by UNEP<sup>1</sup>**



706

707

708

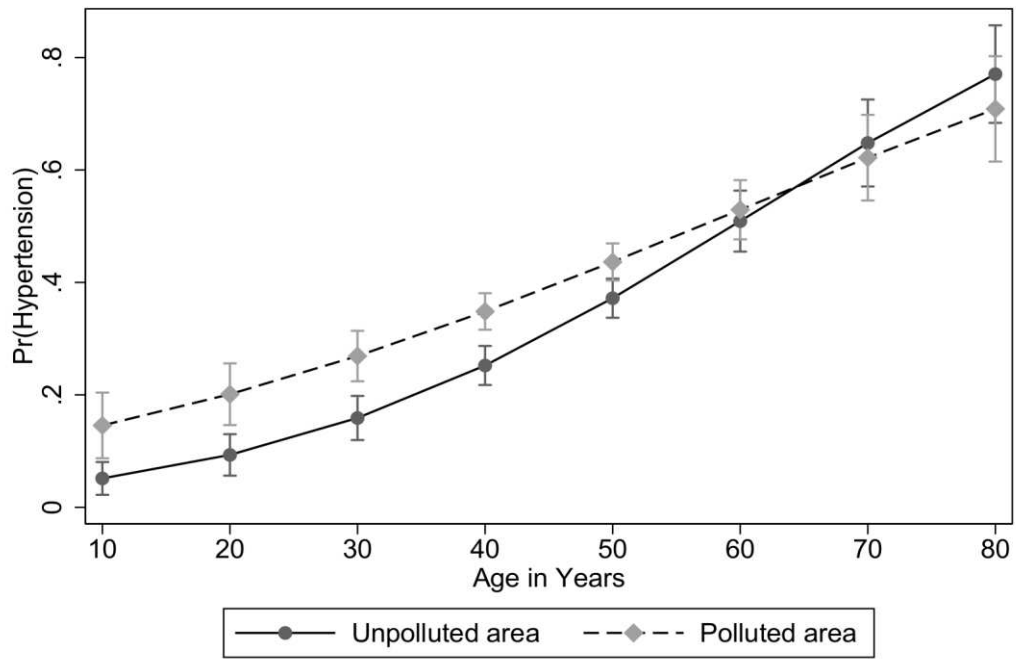
709

710

711

712

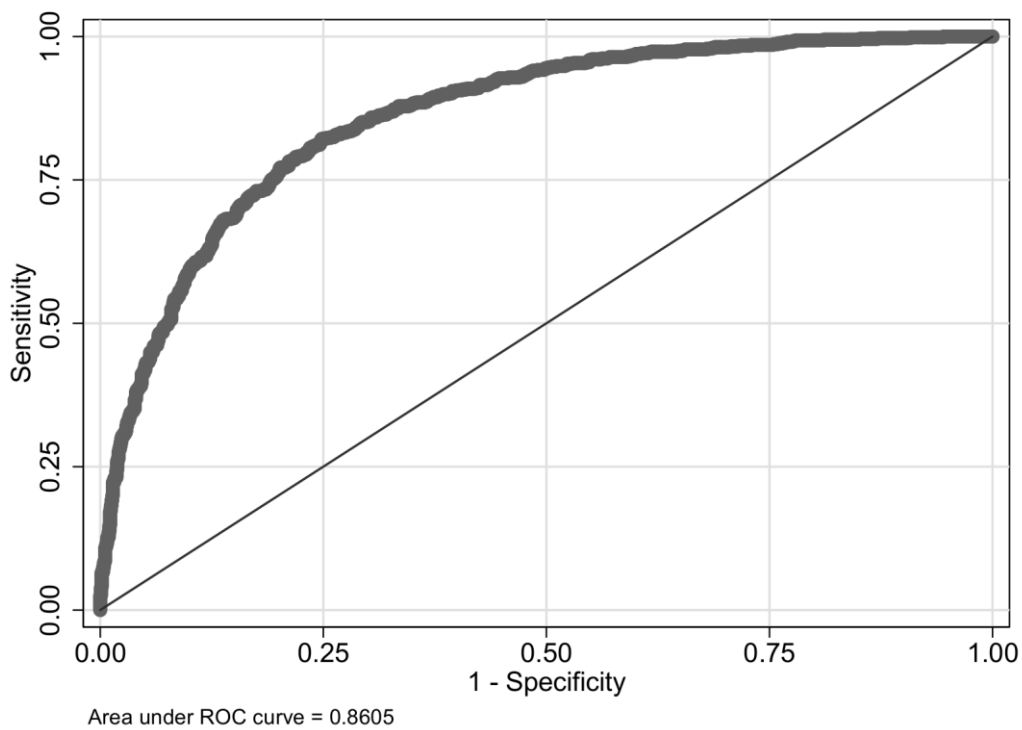
713 **Figure 2. Predicted probability of hypertension for all ages by oil pollution status**



714

715

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



717

Text word count: 2989  
Abstract word count: 250  
No. of references: 33  
No. of figures: 3  
No. of Tables: 3

1  
2  
3  
4  
5  
6  
7 **The burden of hypertension in an oil and gas-polluted environment: a comparative**  
8 **cross-sectional study**  
9

10 Martinsixtus C. Ezejimofor MPH<sup>a</sup>; Olalekan A.Uthman PhD<sup>a,b,c</sup>; Omosivie Maduka MB, BS  
11 FMCPH<sup>d</sup>; Aloysius C. Ezeabasili PhD<sup>e</sup>; Arthur C. Onwuchekwa MB,BS, FCMP<sup>f</sup>; Benedeth  
12 C.Ezejimofor MD, MPH<sup>a</sup>; Eme Asuquo MPH<sup>d</sup>; Yen-Fu Chen PhD<sup>a,b</sup>; Saverio Stranges MD  
13 PhD<sup>a,g</sup>; Ngianga-Bakwin Kandala PhD<sup>a,g,h</sup>

14 <sup>a</sup>Division of Health Sciences, University of Warwick Medical School, Coventry, CV4 7AL,  
15 UK

16 <sup>b</sup>Warwick-Centre for Applied Health Research and Delivery (WCAHRD), Division of health  
17 Sciences, University of Warwick Medical School, Coventry, UK

18 <sup>c</sup>Liverpool School of Tropical Medicine, International Health Group, Liverpool, UK

19 <sup>d</sup>Department of Preventive and Social Medicine, University of Port Harcourt, Port Harcourt,  
20 Nigeria

21 <sup>e</sup>School of the Built Environment, University of Salford, M5 4WT, UK

22 <sup>f</sup>Department of Internal Medicine, University of Port Harcourt, Port Harcourt, Nigeria

23 <sup>g</sup>Department of Population Health, Luxembourg Institute of Health (LIH), 1A-B, rue Thomas  
24 Edison, L-1445 Strassen, Luxembourg

25 <sup>h</sup>Northumbria University, Department of Mathematics and Information sciences, Faculty of  
26 Engineering and Environment, Newcastle upon Tyne, NE1 8ST, United Kingdom.

27  
28 **Corresponding Author**

29 Martinsixtus C. Ezejimofor  
30 Division of Health Sciences  
31 University of Warwick Medical School  
32 Coventry, CV4 7AL, UK  
33 Tel: +44(0)7944420045  
34 Fax: +44(0)24 76573163  
35 Email: [m.c.ezejimofor@warwick.ac.uk](mailto:m.c.ezejimofor@warwick.ac.uk)  
36  
37  
38

39 **ABSTRACT**

40

41 **Background**

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

45 **Methods**

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

54 **Results**

55 More than one-third of participants were hypertensive (37.4%). Half of the participants were  
56 from oil polluted areas (51%). Only 15% of participants reported family history of  
57 hypertension. In the adjusted model, participants living in oil polluted areas were almost five  
58 times as likely to have developed hypertension (adjusted odds ratio [aOR] = 4.85, 95% CI  
59 1.84 to 12.82) compared to participants in unpolluted areas. Age modifies the association  
60 between pollution status and risk of hypertension. For every ten years increase in the age of  
61 the participants, the odds of developing hypertension increased by 108% (aOR = 2.08, 95%  
62 CI 1.77 to 2.43).

63

64 Conclusion

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

67

68

69

70

71

72

73

74

75

76

77

78

79

80

81

82

83

84

85

86

87

88

## 89 INTRODUCTION

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

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

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

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

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

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

120

## 121 **MATERIALS AND METHODS**

### 122 **Study setting and population**

123 The study was carried out among residents exposed to oil and gas pollution in a dominantly  
124 oil and gas polluted community, and non-exposed residents from another community without  
125 any oil and gas exploration or related activities (Figure 1). The two communities have the  
126 same socioeconomic and cultural features in Eleme and Degema local Government area in  
127 Rivers State. However, they differ remarkably in terms of environmental pollution and  
128 exposure level largely due to oil exploration and gas flaring and allied industrial activities.

129 The communities are about 60km apart with an estimated population of 30,580. Ebubu is a  
130 rural farming settlement with sparse social infrastructure. The community has huge functional  
131 oilfields and gas flaring sites in addition to a network of oil pipelines. It is a highly polluted  
132 community with history of oil and gas exploration activities by Shell Petroleum Development  
133 Company<sup>1</sup>. Usokun on the other hand is a pristine rural settlement, a non-oil and gas  
134 producing (unpolluted) community. This community constitutes the control population. It is  
135 an island, circled by Sambreiro River (an outlet of the Niger) with only one access road  
136 connecting it to the urban areas. This rural community is inhabited by people whose  
137 predominant occupation is fishing and vegetable farming.

138

139 **Study design and sample size**

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

146

147 **Data collection**

148 The target population for the study was all men and women aged 18-80 years living  
149 continuously in the selected community for at least 10 years. The survey took place between  
150 June-September, 2014. Data collection was undertaken by research assistants recruited from  
151 the communities and trained according to standard protocols and procedures. All persons who  
152 gave informed consent (in writing and/or thumb print) were included in the study. A pre-  
153 tested study questionnaire was used to collect information on sociodemographic  
154 characteristics and lifestyle factors, as well as medical history of hypertension. Participants  
155 were asked about any previous diagnosis and current treatment for hypertension. Those with  
156 positive answers for hypertensive medications were asked to show their medication for  
157 confirmation. Anthropometric and blood pressure measurements were also taken by research  
158 assistants. Height was measured to the nearest 0.1 cm using a portable collapsible stadiometer  
159 (Leicester Height Measure SECA, Ltd, Birmingham, UK) placed on a firm level surface with  
160 participants wearing no hat and shoes. Participants' body weight was measured to the nearest  
161 0.1 kg while they were dressed in light clothing without footwear using calibrated digital  
162 weighing scale (SECA 877, GmbH, Hamburg, Germany). BMI was calculated as weight in  
163 kilograms divided by the square of height in meters (kg/m<sup>2</sup>). Blood pressure was measured



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

168

### 169 **Outcome variable**

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

175

### 176 **Exposure (determinant variable)**

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

180

### 181 **Potential confounders (control variables)**

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

186

187

188

189 **Ethical considerations**

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

195

196 **Statistical analysis**

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

210

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

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

224

## 225 **RESULTS**

### 226 **Summary statistics for the study participants**

227 Descriptive statistics for study participants are shown in Table 1. More than one-third of  
228 participants were hypertensive (37.4%). Half of the participants were from oil polluted areas  
229 (51%) with preponderance of women (57.0%). The overall participants' mean age was 44.3  
230 (standard deviation: 14.0), this differed between the polluted and unpolluted areas (44.7 vs  
231 43.9),  $P=0.09$ . Most of the participants had secondary or higher education (61.8%) and  
232 currently working (81.7%). More than half of the participants were either overweight or  
233 obese (55.1%) and were mostly from the polluted area (62.3%). Most of the participants were  
234 non-smokers (83.3%) and 39.4% were reported to have never consumed alcohol. A  
235 significant percentage of participants in the unpolluted compared to polluted area reported  
236 moderate-to-high salt (74.9% vs 46.8%) and fat intake (59.8% vs 44.2%),  $P=0.01$ ,  
237 respectively.

238

239 **Factors associated with hypertension**

240 The results of univariate and multivariate logistics regression models are presented in Table  
241 2. In the univariate analysis, with the exception of sex of the participants, all the variables  
242 were statistically significantly associated with hypertension. However, in the multivariate  
243 model, the following factors remained statistically significant with hypertension: pollution  
244 status, age, family history of hypertension, body mass index, drinking status, sleep  
245 deprivation, level of physical activity and fat intake. Participants living in oil polluted areas  
246 were almost five times as likely to have developed hypertension (adjusted odds ratio [aOR] =  
247 4.85, 95% CI 1.84 to 12.82], P=0.01).

248 All the interaction terms, except for age and education attainment were not statistically  
249 significant and were dropped from the final multivariate model (Table 3). For every ten years  
250 increase in the age of the participants, the odds of developing hypertension increased by  
251 108% (aOR = 2.08, 95% CI 1.77 to 2.43). As shown in Figure 2, age modifies the association  
252 between pollution status and risk of hypertension, such that predicted probability of  
253 hypertension was significantly higher for participants' between the age of 20 and 40 years  
254 old. After the age of 65, the effect of pollution status tended to diminish.

255 Compared with participants with normal weight, overweight or obese participants were more  
256 likely to be hypertensive (aOR = 1.33, 95% CI 1.04 to 1.69). Moderate-to-heavy  
257 consumption of alcohol (aOR = 1.63, 95% CI 1.19 to 2.23), moderate-to-severe sleep  
258 deprivation (aOR = 4.27, 95% CI 3.14 to 5.81), sedentary behaviour (aOR = 3.09, 95% CI  
259 1.66 to 5.76), moderate-to-high fat intake (aOR = 1.39, 95% CI 1.08 to 1.78) were  
260 statistically significantly associated with increased risk of hypertension.

261

262

## 263 **Model fit statistics**

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

275

## 276 **DISCUSSION**

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

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

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

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

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

310 In addition to the above assessment, we also found interaction between age and oil pollution  
311 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<sup>30</sup>.

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<sup>4</sup>. 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<sup>16, 31</sup>.

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<sup>32,33</sup>. 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

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

351

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

358

359

360

361



362 **AUTHOR CONTRIBUTIONS**

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

368  
369 **STUDY FUNDING**

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

374

375 **DISCLOSURES**

376 We declare that we have no conflicts of interest

377

378

379

380

381

382

383

384

385

386

387

388

389

390

391 **References**

392

393 1. United Nations Environment Programme. Environmental Assessment of Ogoniland  
394 2011.<http://www.unep.org/disastersandconflicts/CountryOperations/Nigeria/EnvironmentalAssessmentofOgonilandreport/tabid/54419/Default.aspx>, p. 1-257. Accessed on  
395 20 July 2015.

397 2. Kadafa AA. Environmental impacts of oil exploration and exploitation in the Niger  
398 Delta of Nigeria. *Glo J Sci Front Res* 2012;12:19-28.

399 3. Ite AE, Ibok UJ. Gas flaring and venting associated with petroleum exploration and  
400 production in the Nigeria's Niger Delta. *Am J Environ Protec* 2013;1:70-77.

401 4. Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin  
402 F, Hong Y, Luepker RV, Mittleman MA. Particulate matter air pollution and  
403 cardiovascular disease an update to the scientific statement from the American Heart  
404 Association. *Circulation* 2010; 121:2331-2378.

405 5. Lodovici M, Bigagli E. Oxidative stress and air pollution exposure. *J Toxicol.*  
406 2011;2011.

407 6. Liao D, Creason J, Shy C, Williams R, Watts R, Zweidinger R. Daily variation of  
408 particulate air pollution and poor cardiac autonomic control in the elderly. *Environ*  
409 *Health Perspec* 1999;107:521.

410 7. Pekkanen J, Brunner E, Anderson H, Tiittanen P, Atkinson R. Daily concentrations of  
411 air pollution and plasma fibrinogen in London. *Occup Environ Med.* 2000;57:818-822.

412 8. Brook RD, Rajagopalan S. Chronic Air Pollution Exposure and Endothelial  
413 Dysfunction: What You Can't See—Can Harm You. *J Am Coll Cardiol* 2012;60:2167-  
414 2169.

- 415 9. Chuang K-J, Yan Y-H, Cheng T-J. Effect of air pollution on blood pressure, blood  
416 lipids, and blood sugar: a population-based approach. *J Occup Environ Med*  
417 2010;52:258-262.
- 418 10. Chuang K-J, Yan Y-H, Chiu S-Y, Cheng T-J. Long-term air pollution exposure and  
419 risk factors for cardiovascular diseases among the elderly in Taiwan. *Occup Environ*  
420 *Med* 2010; 68:64-68
- 421 11. Dong G-H, Qian ZM, Xaverius PK, Trevathan E, Maalouf S, Parker J, Yang L, Liu M-  
422 M, Wang D, Ren W-H. Association between long-term air pollution and increased  
423 blood pressure and hypertension in China. *Hypertension* 2013; 61:578-584.
- 424 12. Coogan PF, White LF, Jerrett M, Brook RD, Su JG, Seto E, Burnett R, Palmer JR,  
425 Rosenberg L, Air pollution and incidence of hypertension and diabetes mellitus in  
426 black women living in Los Angeles. *Circulation* 2012; 125:767-772.
- 427 13. Pope CA, Burnett RT, Thurston GD, Thun MJ, Calle, EE, Krewski D, Godleski JJ.  
428 Cardiovascular mortality and long-term exposure to particulate air pollution  
429 epidemiological evidence of general pathophysiological pathways of disease.  
430 *Circulation* 2004;109:71-77.
- 431 14. Gobo A, Richard G, Ubong I. Health Impact of Gas Flares on Igwuruta/Umuechem  
432 Communities in Rivers State. *J Appl Sci Environ Mgt* 2009;13
- 433 15. Neuberger M, Kundi M, Jäger R. Chloracne and morbidity after dioxin exposure  
434 (preliminary results). *Toxicol lett* 1998;96:347-350.
- 435 16. Stranges S, Dorn JM, Cappuccio FP, Donahue RP, Rafalson LB, Hovey KM,  
436 Freudenheim JL, Kandala, N-B, Miller MA, Trevisan M. A population-based study of  
437 reduced sleep duration and hypertension: the strongest association may be in  
438 premenopausal women. *J Hypertens* 2010; 28:896-902.

- 439 17. Dettoni JL, Consolim-Colombo FM, Drager LF, Rubira MC, de Souza SBPC, Irigoyen  
440 MC, Mostarda C, Borile S, Krieger EM, Moreno H. Cardiovascular effects of partial  
441 sleep deprivation in healthy volunteers. *J Appl Physiol* 2012; 113:232-236.
- 442 18. Adeloye D, Basquill C, Aderemi AV, Thompson JY, Obi FA. An estimate of the  
443 prevalence of hypertension in Nigeria: a systematic review and meta-analysis. *J*  
444 *Hypertens* 2015;33:230-242.
- 445 19. Ogah O, Okpechi I, Chukwuonye I, Akinyemi J, Onwubere B, Falase A, Stewart S,  
446 Sliwa K. Blood pressure, prevalence of hypertension and hypertension related  
447 complications in Nigerian Africans: A review. *World J Cardiol* 2012;4:327-340
- 448 20. Onwuchekwa AC, Mezie-Okoye MM, Babatunde S. Prevalence of hypertension in  
449 Kegbara-Dere, a rural community in the Niger Delta region, Nigeria. *Ethn Dis*  
450 2012;22:340-346.
- 451 21. Altunkan S, Ilman N, Kayatürk N, Altunkan E. Validation of the Omron M6 (HEM-  
452 7001-E) upper-arm blood pressure measuring device according to the International  
453 Protocol in adults and obese adults. *Blood Press Monit* 2007;12:219-225.
- 454 22. Altunkan S, Ilman N, Altunkan E. Validation of the Omron M6 (HEM-7001-E) upper  
455 arm blood pressure measuring device according to the International Protocol in elderly  
456 patients. *Blood Press Monit* 2008;13:117-122.
- 457 23. Mickey J, Greenland S. A study of the impact of confounder-selection criteria on  
458 effect estimation. *Am J Epidemiol* 1989;129:125-13
- 459 24. Vandembroucke JP, Von EE, Altman DG, Gøtzsche PC, Mulrow CD, Pocock SJ, Poole  
460 C, Schlesselman JJ, Egger M. Strengthening the Reporting of Observational Studies in  
461 Epidemiology (STROBE): explanation and elaboration. *Ann Intern Med* 2007;  
462 147:163-194.

- 463 25. Oguoma VM, Nwose EU, Skinner TC, Digban KA, Onyia IC, Richards RS.  
464 Prevalence of cardiovascular disease risk factors among a Nigerian adult population:  
465 relationship with income level and accessibility to CVD risks screening. *BMC Public*  
466 *Health* 2015;15:397.
- 467 26. Ordinioha B. The prevalence of hypertension and its modifiable risk factors among  
468 lecturers of a medical school in Port Harcourt, south-south Nigeria: implications for  
469 control effort. *Niger J Clin Pract* 2013;16:1-11.
- 470 27. Rahman M, Tondel M, Ahmad SA, Chowdhury IA, Faruquee MH, Axelson O.  
471 Hypertension and arsenic exposure in Bangladesh. *Hypertension* 1999;33:74-78.
- 472 28. Lu Y, Liu X, Deng Q, Duan Y, Dai H, Li Y, Xiao T, Ning X, Fan J, Zhou L .  
473 Continuous Lead Exposure Increases Blood Pressure but Does Not Alter Kidney  
474 Function in Adults 20-44 Years of Age in a Lead-Polluted Region of China. *Kidney*  
475 *Blood Press Res* 2015; 40:207-214.
- 476 29. Caciari T, Sancini A, Fioravanti M, Capozzella A, Casale T, Montuori L, Fiaschetti M,  
477 Schifano M, Andreozzi G. Cadmium and hypertension in exposed workers: A meta-  
478 analysis. *Int J Occup Med Environ Health* 2013; 26:440-456.
- 479 30. Bruce N, Rogelio P-P, Rachel A. The health effects of indoor air pollution exposure in  
480 developing countries. Geneva: World Health Organization 2002; 11  
481 <http://www.bioenergylists.org/stovesdoc/Environment/WHO/OEH02.5.pdf> p. 1-40.  
482 Accessed on 20 November, 2015.
- 483 31. Cappuccio FP, Stranges S, Kandala N-B Miller MA, Taggart FM, Kumari M, Ferrie  
484 JE, Shipley MJ, Brunner EJ, Marmot MG. Gender-specific associations of short sleep  
485 duration with prevalent and incident hypertension the Whitehall II study. *Hypertension*  
486 2007; 50:693-700.

487 32. Crighton EJ, Elliott SJ, Van Der Meer J, Small I, Upshur R. Impacts of an  
488 environmental disaster on psychosocial health and well-being in Karakalpakstan. Soc  
489 Sci Med 56, 551-567.

490 33. Couch SR, Coles CJ. Community stress, psychosocial hazards, and EPA decision-  
491 making in communities impacted by chronic technological disasters. Am J Public  
492 Health 2011;101:140-148.

493

#### 494 **List of Tables**

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

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

497 Table 3. Interaction of demographic variables and pollution status

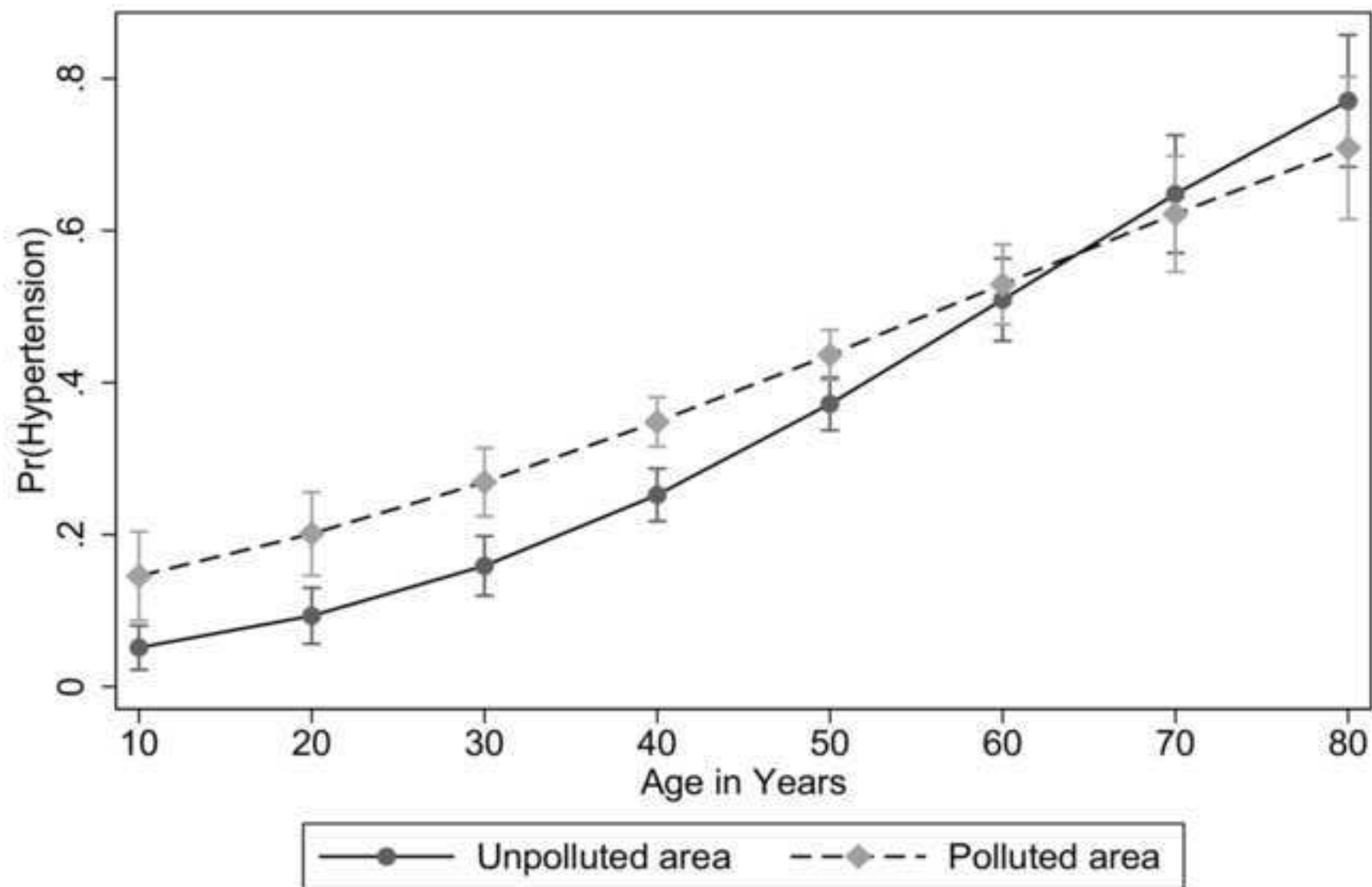
498

#### 499 **List of Figures**

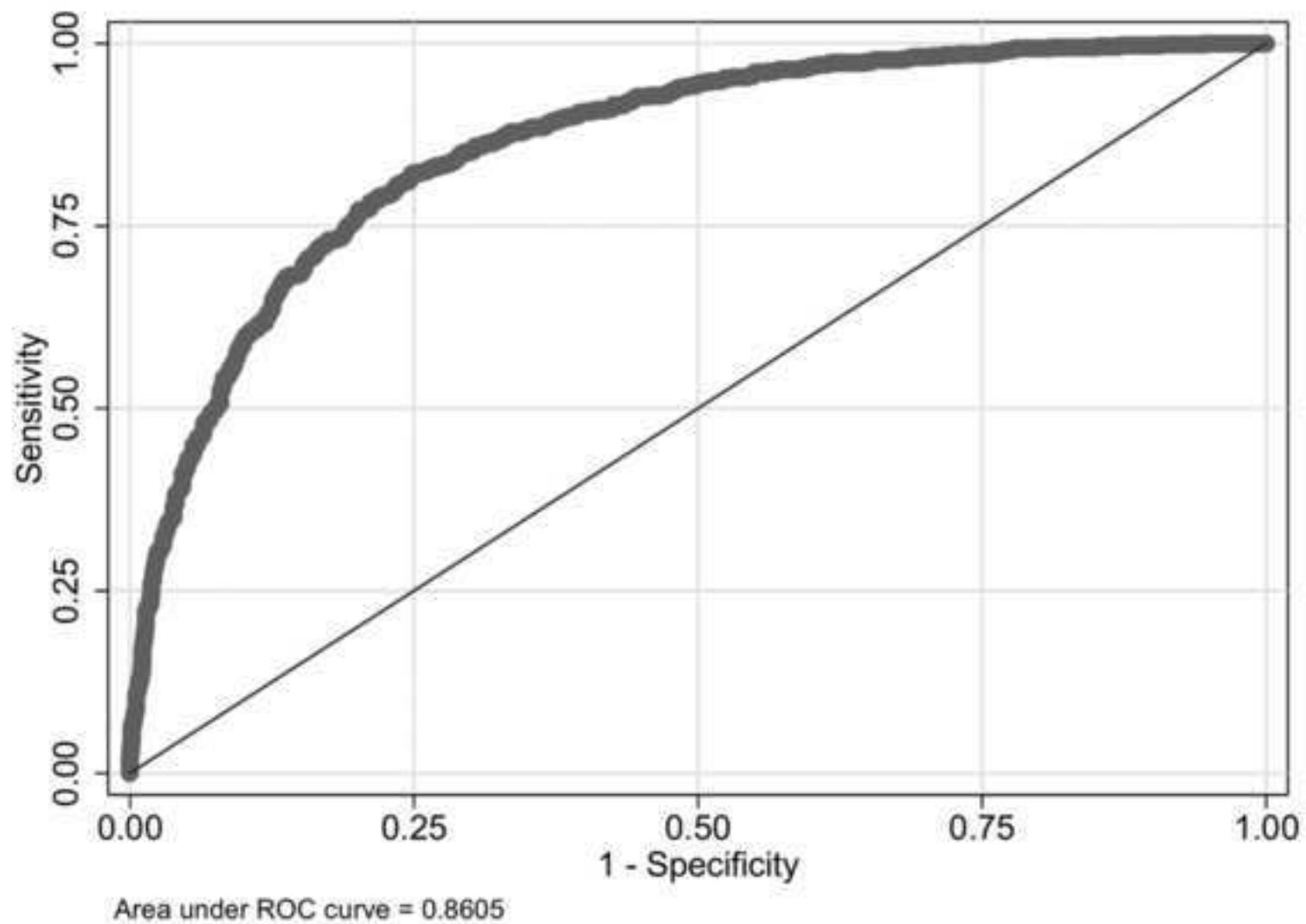
500 Figure 1. Map of the study local councils of the study areas showing oil and gas production  
501 sites investigated by UNEP<sup>1</sup>

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

503 Figure 3. Receiver operating characteristics curves for final multivariable model

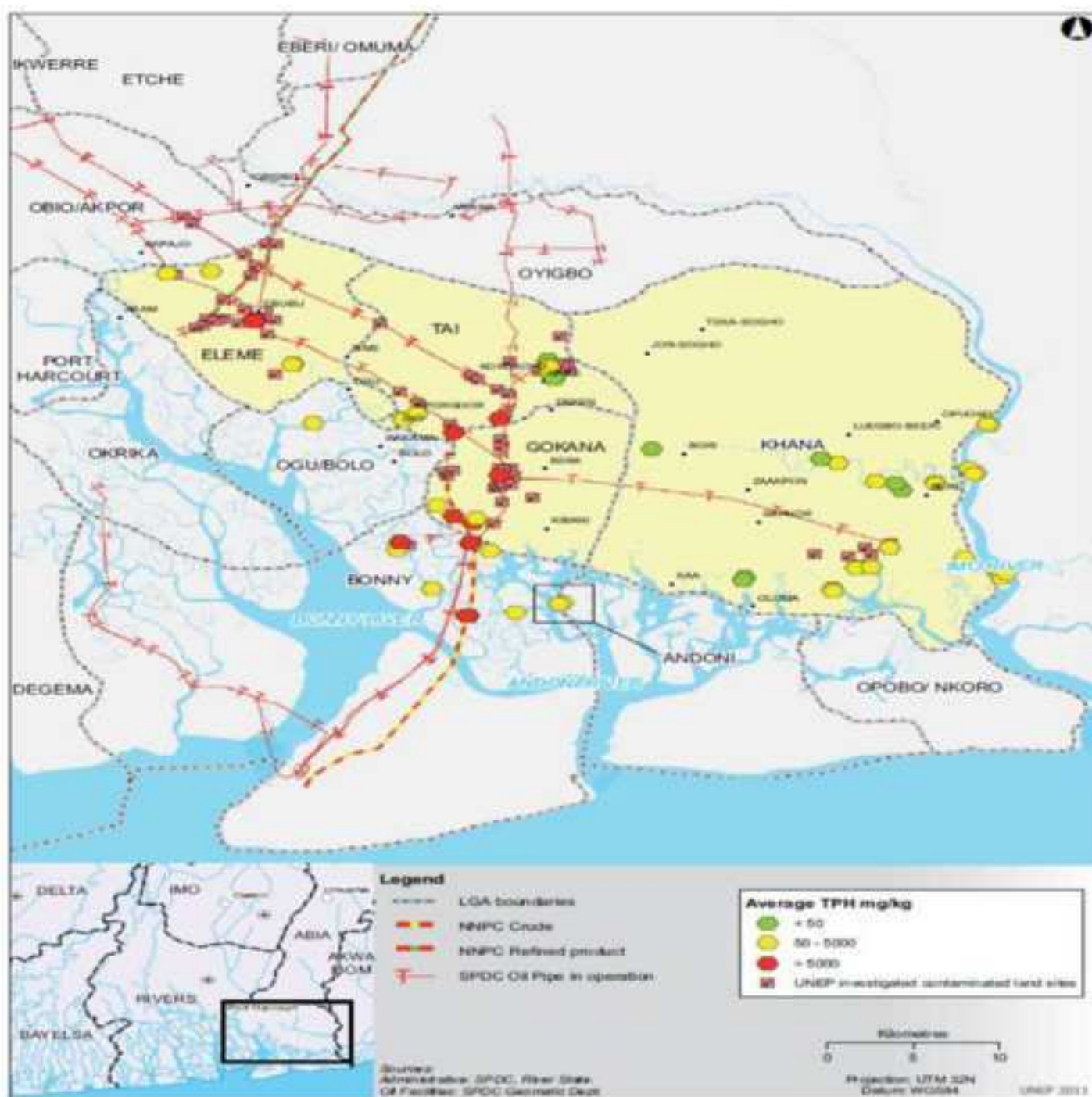
**Figure 2. Predicted probability of hypertension for all ages by oil pollution status**

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





**Figure 1. Map of the study local councils of the study areas showing oil and gas production sites investigated by UNEP<sup>1</sup>**



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

<b>Variable</b>	<b>Total</b>	<b>Polluted area</b>	<b>Non-polluted area</b>	<b>P-value<sup>a</sup></b>
	<b>(n=2028)</b>	<b>(n=1036)</b>	<b>(n=992)</b>	
<b>Mean age (SD)</b>	44.3 (14.0)	44.7 (13.3)	43.9 (14.7)	0.09
<b>Gender (%)</b>				
Male	871 (43.0)	417(40.0)	454 (46.0)	
Female	1157 (57.0)	619(60.0)	538(54.0)	0.01
<b>Hypertensive<sup>b</sup> (%)</b>				
No	1270 (62.6)	587(56.7)	683(68.9)	
Yes	758 (37.4)	449(43.3)	309(31.2)	0.01
<b>Family history of hypertension (%)</b>				
No	1724 (85.0)	847(81.8)	877(88.4)	
Yes	304 (15.0)	189(18.2)	115(11.6)	0.01
<b>Marital Status (%)</b>				
Never married	544 (26.8)	235(22.7)	309(31.2)	
Ever married	1483 (73.2)	800(77.3)	683(68.9)	0.01
<b>Education attainment (%)</b>				
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 ( $\geq$ 12 Years)	1255 (61.8)	637(61.5)	618(62.3)	0.01
<b>Employment status (%)</b>				
Unemployed	371 (18.3)	165(15.9)	206(20.8)	
Presently working	1657 (81.7)	871(84.1)	786(79.2)	0.005
<b>Body mass index (%)</b>				
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

---

<b>Smoking status (%)</b>				
Non-smoker	1689 (83.3)	925(89.3)	764(77.0)	
Ever smoker	339 (16.7)	111(10.7)	228(23.0)	0.01
<b>Drinking status (%)</b>				
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
<b>Sleep deprivation (%)</b>				
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
<b>Physical activity (%)</b>				
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
<b>Salt intake (%)</b>				
Low	800 (39.4)	551(53.2)	249(25.1)	
Moderate-to-high	1228 (60.6)	485(46.8)	743(74.9)	0.01
<b>Fat intake (%)</b>				
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.

<sup>a</sup>P values for comparison between polluted and non-polluted areas.

<sup>b</sup>Defined as blood pressure  $\geq$  140/90 mmHg or on antihypertensive medication.

**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
<b><u>Main effects</u></b>				
<b>Polluted (vs unpolluted) area</b>	1.69 (1.41 to 2.03)	0.01	4.85 (1.84 to 12.82)	0.01
<b>Age (per 10 years increase)</b>	2.27 (2.09 to 2.47)	0.01	2.08 (1.77 to 2.43)	0.01
<b>Female (vs male)</b>	1.06 (0.88 to 1.27)	0.54	Not included	
<b>Family history of hypertension (vs no)</b>	3.17 (2.46 to 4.08)	0.01	2.41 (1.75 to 3.32)	0.01
<b>Ever (vs. never) married</b>	6.21 (4.72 to 8.18)	0.01	1.62 (1.12 to 2.35)	0.01
<b>Education attainment</b>				
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)	
<b>Currently employed (vs unemployed)</b>	2.93 (2.23 to 3.86)	0.01	1.43 (0.95 to 2.15)	0.09
<b>Body mass index</b>				
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
<b>Ever- (vs. non-) smoker</b>	1.61 (1.27 to 2.03)	0.01	1.28 (0.92 to 1.77)	0.14
<b>Drinking status</b>				
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
<b>Sleep deprivation</b>				
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
<b>Physical activity</b>				
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)	
<b>Moderate-to-high (vs. low) salt intake</b>	1.50 (1.24 to 1.80)	0.01	1.10 (0.84 to 1.43)	0.50
<b>Moderate-to-high (vs. low) fat intake</b>	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**

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

<sup>1</sup> Receiver Operating Characteristic<sup>2</sup> Variance Inflation Factor