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

183

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

188 ^aDivision of Health Sciences, University of Warwick Medical School, Coventry, CV4 7AL,
189 UK

190 ^bWarwick-Centre for Applied Health Research and Delivery (WCAHRD), Division of health
191 Sciences, University of Warwick Medical School, Coventry, UK

192 ^cLiverpool School of Tropical Medicine, International Health Group, Liverpool, UK

193 ^dDepartment of Preventive and Social Medicine, University of Port Harcourt, Port Harcourt,
194 Nigeria

195 ^eSchool of the Built Environment, University of Salford, M5 4WT, UK

196 ^fDepartment of Internal Medicine, University of Port Harcourt, Port Harcourt, Nigeria

197 ^gDepartment of Population Health, Luxembourg Institute of Health (LIH), 1A-B, rue Thomas
198 Edison, L-1445 Strassen, Luxembourg

199 ^hNorthumbria 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

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.

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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 water¹⁻³. 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 risk⁴. 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 processes⁵. Toxicological evidence found
270 that inflammatory dose of PM is associated with increase in plasma fibrinogen and viscosity,
271 systemic and local inflammatory events⁶, alterations in blood coagulability⁷ and endothelial
272 dysfunction⁸. Specifically, longer-term exposure to ambient air pollution and short term
273 exposure to high PM concentration confers increased cardiovascular risk⁹⁻¹² through
274 initiation of high blood pressure, an established determinant of atherogenesis and
275 cardiovascular diseases (CVD), and a leading cause of death¹³.

276 Gas flaring and oil refining may affect the sleep-wake-cycle in healthy individuals¹⁴.

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 headache¹⁵. Evidence found that sleep
280 deprivation is positively associated with increased cardiovascular risk including
281 hypertension^{16,17}.

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 ^{18,19}. 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 Company¹. 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 ²⁰. 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 (kg/m^2). Blood pressure was measured
335 using an automated and validated^{21,22} 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 ≥ 140 or ≥ 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 studies²³. 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 ²⁴.

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^{18, 19}. 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^{25, 26} 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 metals¹. Evidence found that
472 exposure to heavy metals such as Cadmium, Arsenic and Lead have been associated with
473 hypertension²⁷⁻²⁹. Environmental pollutants such as heavy metals and PM trigger systemic
474 inflammation and oxidative stress leading to hypertension through many pathophysiological
475 mechanisms⁴. 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 countries³⁰.

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 dysfunction⁴. 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^{16, 31}.

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^{32,33}. 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.

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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

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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

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

| Variable | Total | Polluted area | Non-polluted area | P-value^a |
|---|-----------------|----------------------|--------------------------|----------------------------|
| | (n=2028) | (n=1036) | (n=992) | |
| 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 |

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

667 ^aP values for comparison between polluted and non-polluted areas.

668 ^bDefined as blood pressure \geq 140/90 mmHg or on antihypertensive medication.

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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 |
| 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 |

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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 |

695 ¹ Receiver Operating Characteristic696 ² Variance Inflation Factor

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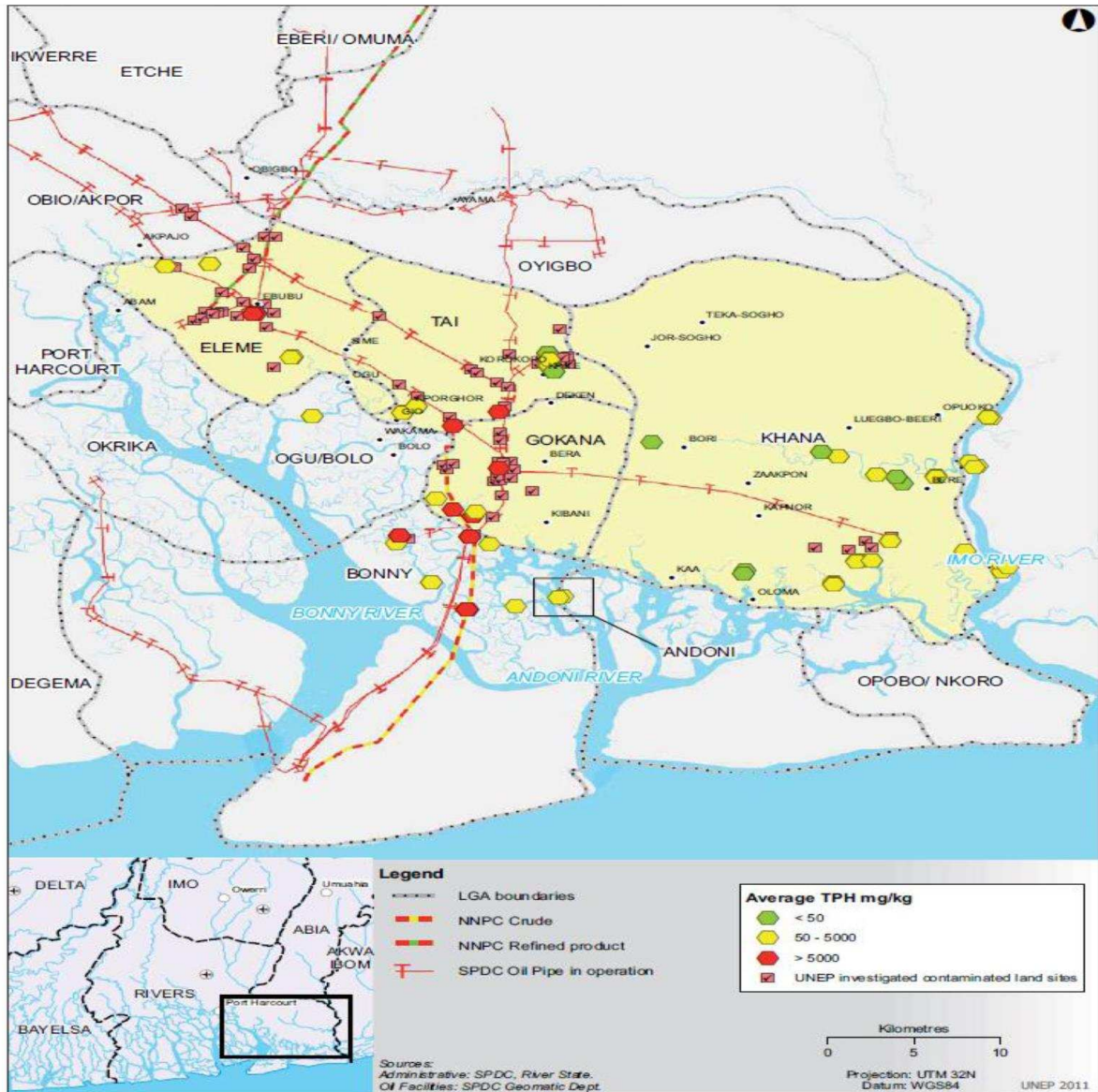
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704 **Figure 1. Map of the study local councils of the study areas showing oil and gas**
 705 **production sites investigated by UNEP¹**



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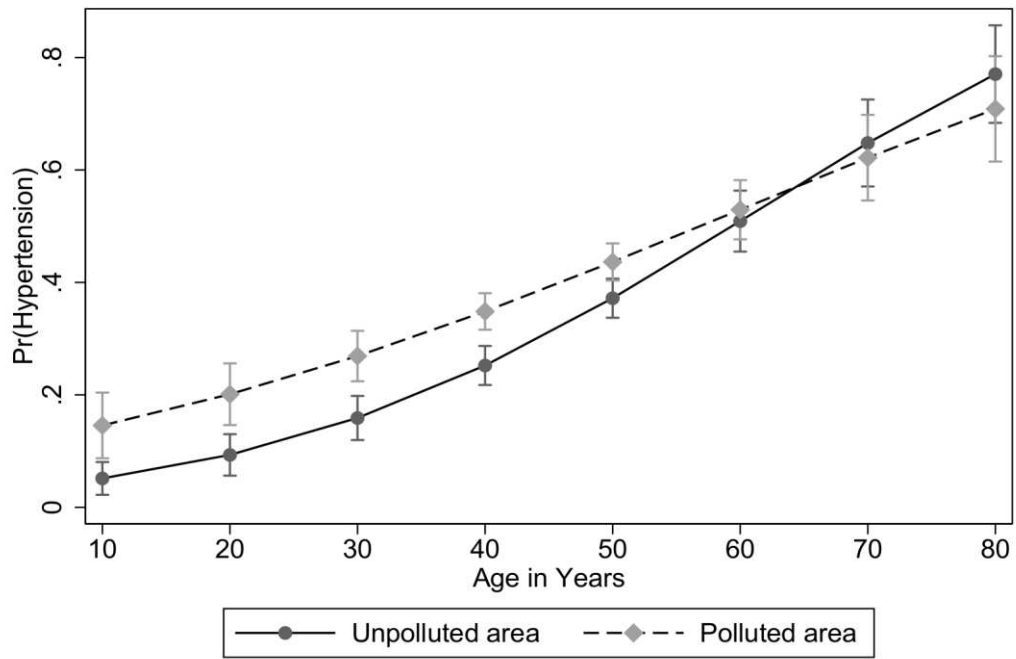
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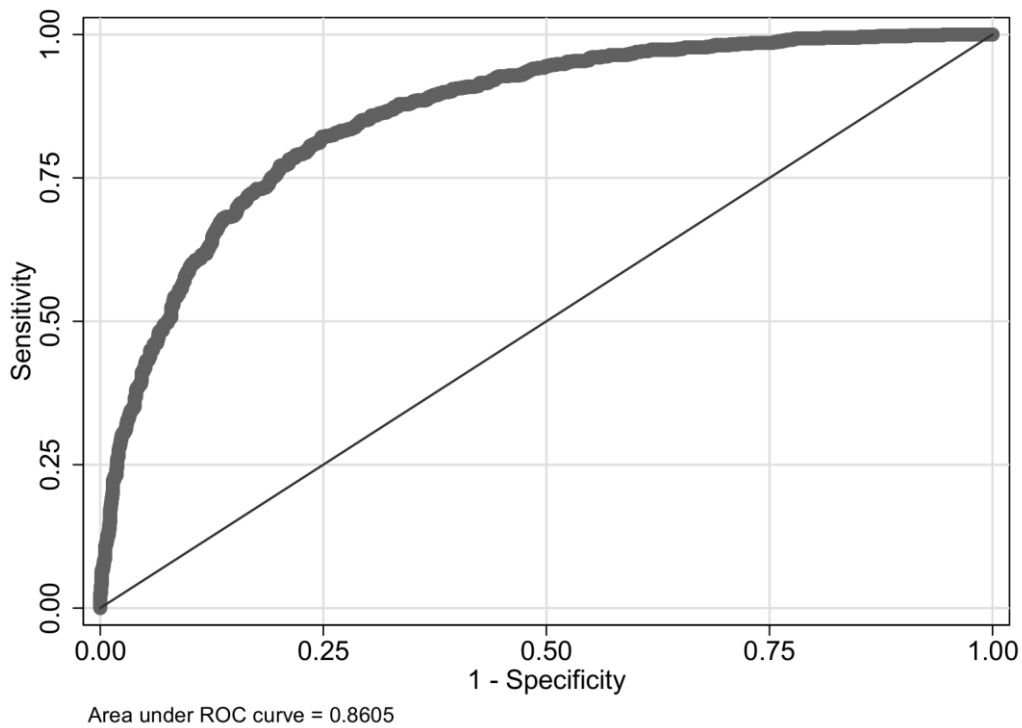
713 **Figure 2. Predicted probability of hypertension for all ages by oil pollution status**



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



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7 **The burden of hypertension in an oil and gas-polluted environment: a comparative**
8 **cross-sectional study**
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10 Martinsixtus C. Ezejimofor MPH^a; Olalekan A.Uthman PhD^{a,b,c}; Omosivie Maduka MB, BS
11 FMCPH^d; Aloysius C. Ezeabasili PhD^e; Arthur C. Onwuchekwa MB,BS, FCMP^f; Benedeth
12 C.Ezejimofor MD, MPH^a; Eme Asuquo MPH^d; Yen-Fu Chen PhD^{a,b}; Saverio Stranges MD
13 PhD^{a,g}; Ngianga-Bakwin Kandala PhD^{a,g,h}

14 ^aDivision of Health Sciences, University of Warwick Medical School, Coventry, CV4 7AL,
15 UK

16 ^bWarwick-Centre for Applied Health Research and Delivery (WCAHRD), Division of health
17 Sciences, University of Warwick Medical School, Coventry, UK

18 ^cLiverpool School of Tropical Medicine, International Health Group, Liverpool, UK

19 ^dDepartment of Preventive and Social Medicine, University of Port Harcourt, Port Harcourt,
20 Nigeria

21 ^eSchool of the Built Environment, University of Salford, M5 4WT, UK

22 ^fDepartment of Internal Medicine, University of Port Harcourt, Port Harcourt, Nigeria

23 ^gDepartment of Population Health, Luxembourg Institute of Health (LIH), 1A-B, rue Thomas
24 Edison, L-1445 Strassen, Luxembourg

25 ^hNorthumbria 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
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39 **ABSTRACT**

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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).

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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.

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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 water¹⁻³. 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 risk⁴. 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 processes⁵. Toxicological evidence found
100 that inflammatory dose of PM is associated with increase in plasma fibrinogen and viscosity,
101 systemic and local inflammatory events⁶, alterations in blood coagulability⁷ and endothelial
102 dysfunction⁸. Specifically, longer-term exposure to ambient air pollution and short term
103 exposure to high PM concentration confers increased cardiovascular risk⁹⁻¹² through
104 initiation of high blood pressure, an established determinant of atherogenesis and
105 cardiovascular diseases (CVD), and a leading cause of death¹³.

106 Gas flaring and oil refining may affect the sleep-wake-cycle in healthy individuals¹⁴.
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 headache¹⁵. Evidence found that sleep
110 deprivation is positively associated with increased cardiovascular risk including
111 hypertension^{16,17}.

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^{18,19}. 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 Company¹. 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 ²⁰. 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/m²). Blood pressure was measured

164 using an automated and validated^{21,22} 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 ≥ 140 or ≥ 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.

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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 studies²³. 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) statement²⁴.

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^{18, 19}. 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^{25, 26} 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 Ebubu community previously found very high percentage of
303 polycyclic aromatic hydrocarbon (PAH), PM and heavy metals¹. Evidence found that
304 exposure to heavy metals such as Cadmium, Arsenic and Lead have been associated with
305 hypertension²⁷⁻²⁹. Environmental pollutants such as heavy metals and PM trigger systemic
306 inflammation and oxidative stress leading to hypertension through many pathophysiological
307 mechanisms⁴. Therefore residents of polluted community like Ebubu 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 countries³⁰.

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 dysfunction⁴. 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

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.

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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
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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

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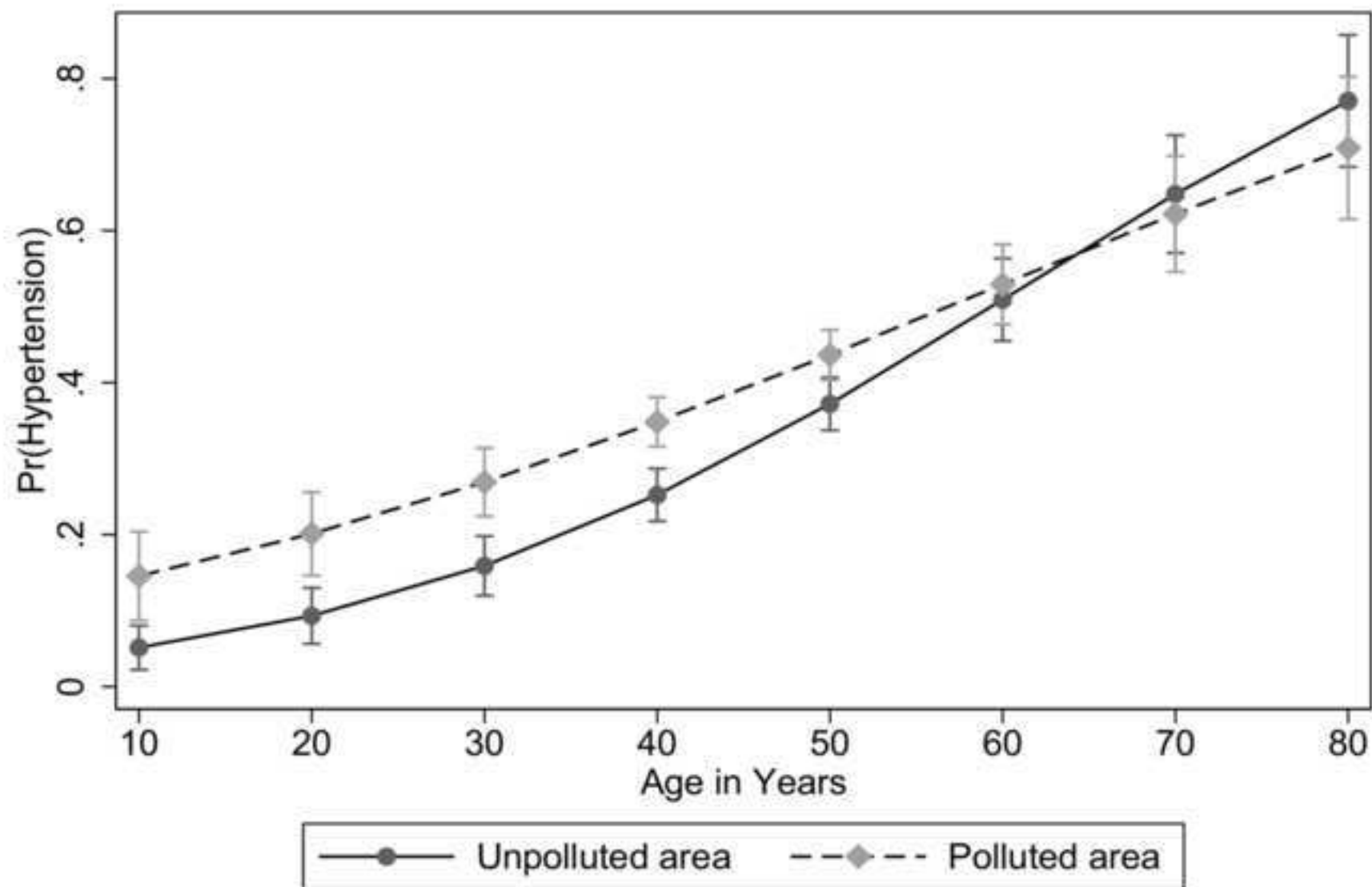
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Figure 3. Receiver operating characteristics curves for final multivariable model

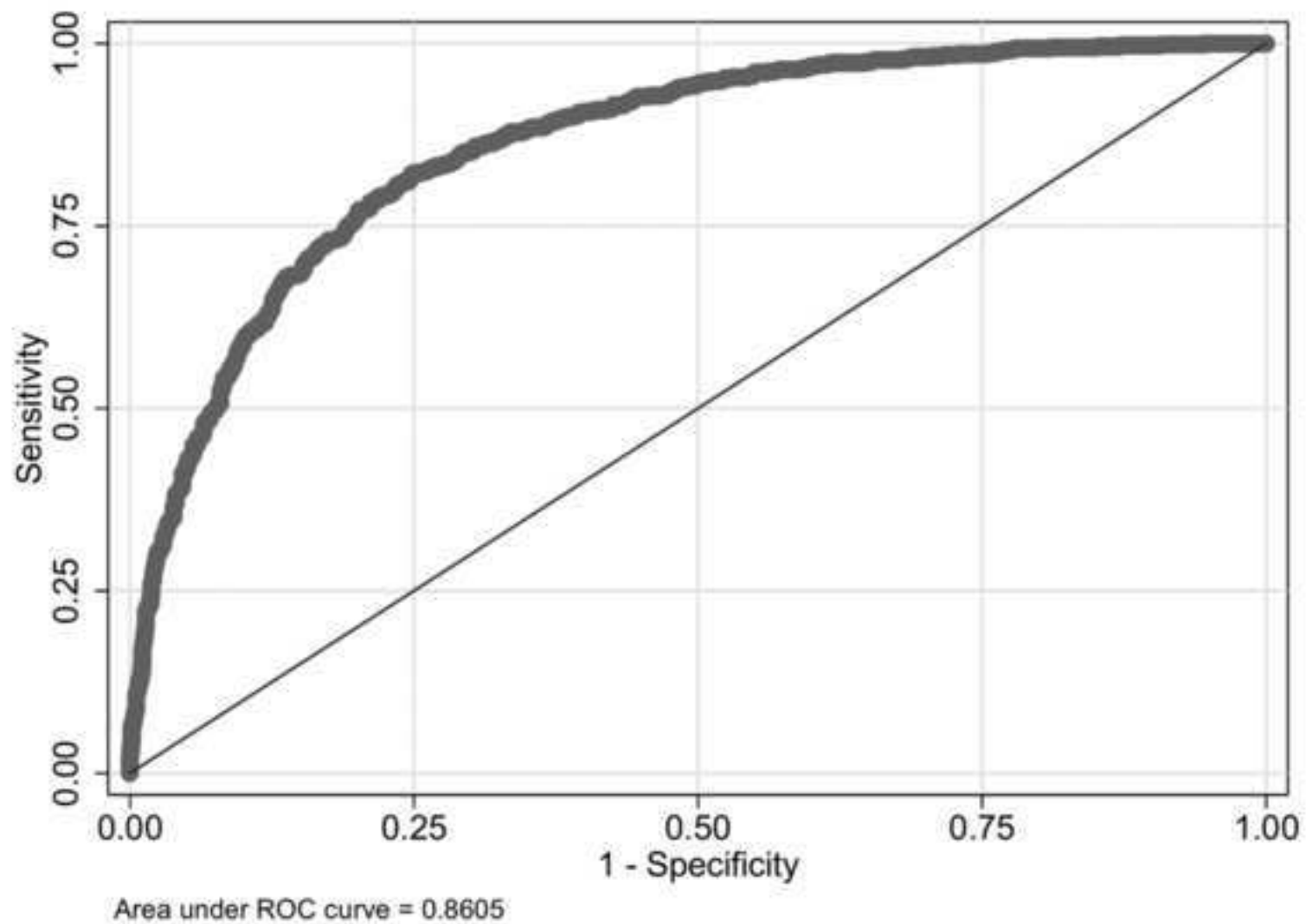


Figure 1. Map of the study local councils of the study areas showing oil and gas production sites investigated by UNEP¹

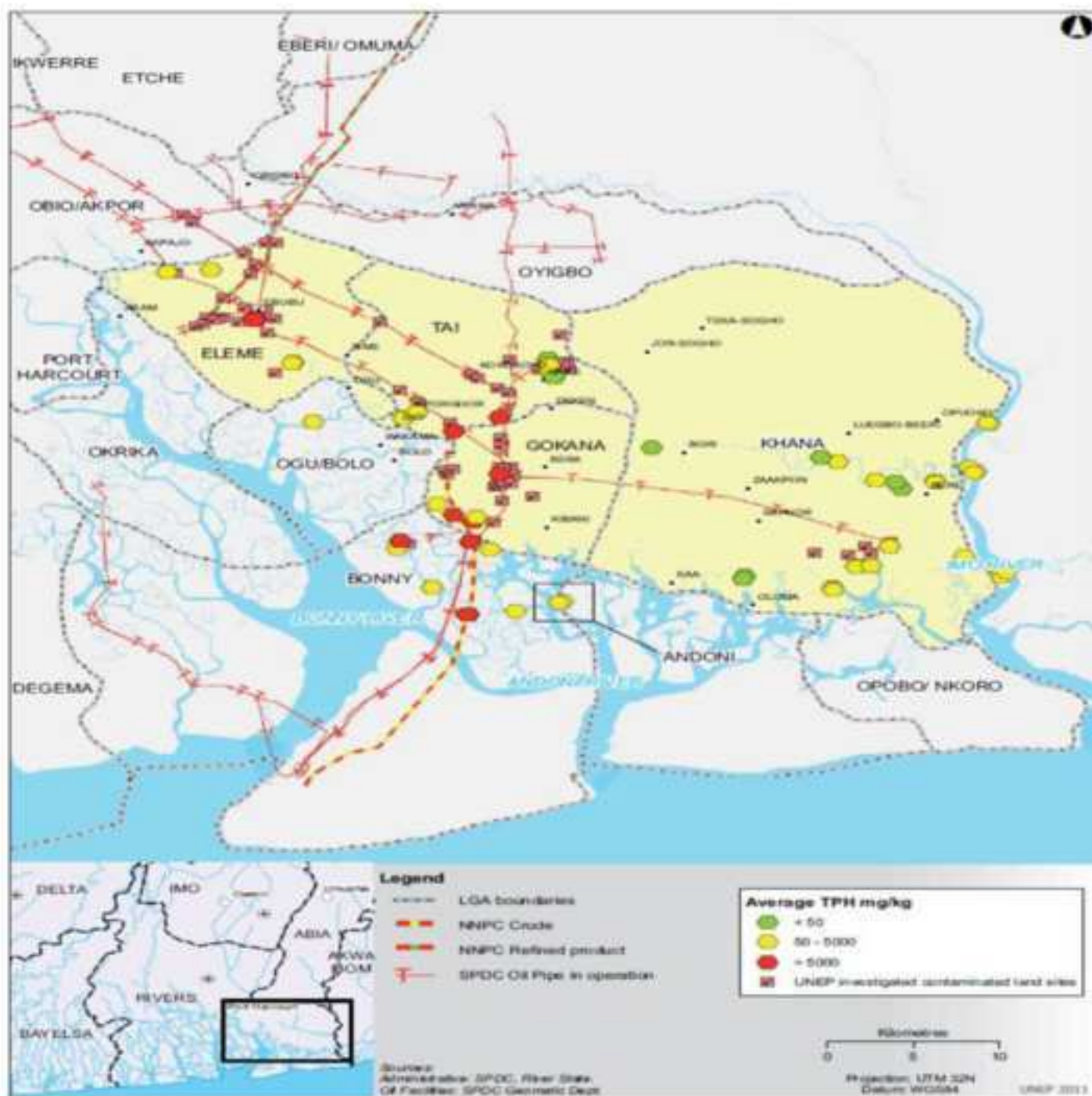


Table 1. Characteristics of the study population by oil pollution status

| Variable | Total | Polluted area | Non-polluted area | P-value^a |
|---|-----------------|----------------------|--------------------------|----------------------------|
| | (n=2028) | (n=1036) | (n=992) | |
| 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.

^aP values for comparison between polluted and non-polluted areas.

^bDefined 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 |
| <u>Main effects</u> | | | | |
| 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 |
| <u>Model fit statistics</u> | | |
| 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 |
| <u>Model fit Statistics</u> | | |
| 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 Characteristic² Variance Inflation Factor