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Air pollution-associated chronic kidney disease (APA-CKD): evidence from a cross-sectional study of Niger Delta communities

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ABSTRACT

Objective Air pollution is an emerging risk factor for chronic kidney disease (CKD) that is typically ignored in preventive interventions. This study investigated whether long-term exposure to ambient air pollution in communities near petrochemical industries in the Niger Delta was associated with CKD.

Design A cross-sectional study with an embedded citizen science inquiry.

Settings Four communities situated at varying distances from a petrochemical refinery in Niger Delta, Nigeria.

Participants We obtained sociodemographic, behavioural, exposure history and clinical data from 1460 participants who have resided for at least 5 years in the four communities. A citizen science approach was used to monitor air pollutant concentrations with eight community volunteers.

Results The mean $PM_{2.5}$, PM_{10} and volatile organic compounds (VOC) concentrations exceeded the WHO-acceptable limits in all four communities. CO_2 was acceptable in the farthest communities from the refinery, while O_3 was within acceptable limits in all communities. The total hazard quotient was relatively higher in the two communities near the refinery (11.27, 11.63) than those farther (9.63, 10.68), F=0.038, p=0.989. The overall prevalence of CKD was 12.3%; it was 17.9% in the community closest to the refinery and 8.0% in the farthest (χ^2=18.292, p=0.004). Increasing age was the only independent risk factor for CKD after adjusting for confounding factors and intrahousehold design effect (adjusted OR 1.26; 95% CI 1.09 to 1.45, p=0.002).

Conclusion Long-term exposure to ambient air pollution may increase CKD risk in susceptible populations. Social factors and environmental exposures associated with CKD are prevalent in the communities, necessitating multifaceted and inclusive approaches to mitigate air pollution and the associated kidney disease risks. More studies are required to explore the mechanism of air pollution-associated kidney disease and interventions to reverse or limit it.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ The limitations of this study include its cross-sectional design and the use of one-off laboratory assessments to define chronic kidney disease (CKD).
⇒ This study demonstrates the innovative combination of citizen science and traditional research to achieve public inclusion in generating evidence on air pollution health risks.
⇒ The large sample size, use of probability sampling and adjustment for confounding factors reinforce the validity and reliability of the study.
⇒ The outcome, CKD, was defined using the Kidney Disease: Improving Global Outcomes (KDIGO) recommendations while long-term air pollution exposure was assessed using the location of residence supported by real-time air pollutant monitoring.

INTRODUCTION

The disproportionately high prevalence and severity of chronic kidney disease (CKD) in the young population of low-income or middle-income countries (LMIC), despite preventive interventions, is a matter of global concern. CKD is a devastating yet neglected non-communicable disease (NCD) that is responsible for 3.4 million deaths annually worldwide and ranks 8–10th among the risk factors for global deaths and disability-adjusted life years.[1 2] The largely asymptomatic nature of the disease and complex multicausality further contribute to late presentation, diagnosis and poor treatment outcomes in LMIC.

The causes of CKD, particularly in disadvantaged populations, have yet to be fully elucidated. Although traditional risk factors such as hypertension (HTN), diabetes, chronic infections and glomerular diseases are well known and are often incorporated into preventive interventions, it would appear that these interventions have not sufficiently addressed the problem. Consequently, the search for other modifiable environmental risk factors is of profound importance. The association of NCDs with environmental risk factors such as diet change, physical inactivity and air pollution is well documented.[3–8] However, while there is a proliferation of

epidemiological and experimental evidence for respiratory, cardiovascular, neurological diseases and cancers, the evidence for air pollution-associated CKD (APA-CKD) is still emerging but scarce from LMIC.[9–13] Drawing from the multicausation theory of NCDs, we define APA-CKD as CKD caused by prolonged air pollution exposure, amplified by other risk factors.

Ambient air pollution is a global environmental risk and the most critical driver of climate change, a global crisis described as a super-wicked problem characterised by four features: 'time is running out; people responsible for the issue want to solve it; there is little to no central authority to deal with it; and policy solutions irrationally devalue the future.[14] Climate change and air pollution exist in a vicious cycle whereby one is both a cause and effect of the other. Air pollution is the leading environmental risk factor for seven million NCD deaths annually.[6] The Global Burden of Disease 2019 report revealed that although exposure to harmful environmental risks was declining globally, exposure to ambient air pollutants (particulate matter) persists, contributing to poor public health indices, especially in LMIC.[1] Toxic industrial emissions near residential areas particularly persist in many developing, emerging countries and minority populations in developed countries, mainly due to poorly enforced regulations, lack of the finances to adopt cleaner technologies and non-inclusion of the public in ongoing mitigation efforts.[15–19] Communities near industries are likely at a higher risk of exposure to air pollution and its associated health effects. However, such communities require further study, especially in disadvantaged countries.

Both air pollution and CKD are persistent global health problems associated with significant morbidity and mortality and disparities across the globe. Although air pollution associated with kidney disease is biologically plausible, as shown in toxicological studies, epidemiological research is lacking from disadvantaged and susceptible populations. The reasons for this lack may be a combination of reasons: research focused on traditional risk factors, poor research funding, lack of interest, insufficient research skills and crowding-out effects.[15 20] Some studies have shown that introducing effective measures and policies can mitigate air pollution and its associated health effects,[21] but high-quality research is needed to inform effective air pollution and CKD policies in LMIC.

Therefore, this study aimed to determine whether persons residing near petrochemical industries are exposed to ambient air pollution and whether long-term exposure is associated with CKD. The study outcomes may contribute reliable information to the growing evidence of APA-CKD and inform future preventive and management strategies.

MATERIALS AND METHODS

Study design

This study design was cross-sectional with an embedded citizen science inquiry. Community volunteers partnered with the research team to monitor air pollutants in the ambient air of their immediate environment.

Study setting

The study location is Warri, Delta State, one of the nine states that make up the Niger Delta region of Nigeria, which is the petrochemical hub. The Nigerian National Petroleum Corporation (NNPC) has four linked petroleum refineries in Nigeria (two in Port Harcourt and one each in Warri and Kaduna) responsible for downstream crude oil processing. The Warri Refining and Petrochemical Company (WRPC), located in Ekpan, Uvwie local government area (LGA), is the first government wholly owned refinery with a capacity to process 125 000 barrels of crude oil daily. However, at the time of this study, the government had reportedly temporarily shut down the refinery for about 6 years to carry out maintenance.[22] Other multinational oil, power and energy companies and several manufacturing companies are situated in Warri. In addition, some of the community members engage in illegal oil refining.

Warri is located on latitude 5.544 and longitude 5.760, with GPS coordinates of 5° 30′ 39.2280″ N, 5° 45′ 36.9684″ E and covering an area of 4021 km.[23] The town comprises three LGAs, namely, Warri-South, Warri North and Warri South-West; however, LGAs like Uvwie, Okpe and Udi have been incorporated into the larger metropolitan area of Warri. Warri-South has the highest population density (792.3/km^2) among the LGAs and covers an area of 542 km^2 but Uvwie, which is one of the neighbouring and newly incorporated LGAs, has an even higher population density of 2812/km^2. Warri is predominantly riverine with large areas of mangrove forests. The climate is humid, with a wet season from March to October and a shorter dry season from November to February. The South-West monsoon and North-East trade winds in the dry seasons influence the climate. The annual rainfall is up to 2500 mm with a double-peak rainfall regime, which takes place both in June and September, while the yearly average temperature is 27°C with minimal seasonal departure from the average.[24]

The area has a projected population of 751 761 with an almost equal male-to-female ratio (males 51.3%, females 48.7%), and the majority (59.1%) of the population in the 15–64 years age group.[23] Warri-South LGA, the largest LGA, has a projected population of 415 344. The people mainly speak English or pidgin English. The prevailing occupations are trading, commercial transportation and farming. Several public institutes of higher learning and several public and private secondary schools are in Warri. There are six public secondary healthcare institutions and 39 state-owned primary healthcare centres in Warri and Uvwie, and several privately owned primary healthcare centres and hospitals in Warri.

Four communities in Warri were purposively selected for this study based on their distance from the Warri refinery gas flaring tower base and urbanicity. The communities included *A* (nearest/semiurban, 3.0 km), *B* (near/urban,

3.5 km), *C* (far/urban, 10 km) and *D* (farthest/rural, 13 km). A combination of urban and rural communities at varying distances from the Warri refinery was selected to achieve a good representation and to provide the opportunity for comparing health risks across different degrees of exposure and social contexts.

Sample size calculation

The minimum sample size was calculated on OpenEpi version 3, using the formula for a cross-sectional survey comparing proportions, which was 1366 (683 per subgroup).[25] The sample size was calculated based on an estimated prevalence of CKD of 14.3% in a less-exposed neighbouring rural population;[26] 20% representing assumed prevalence in the exposed population, power of 80%, a 5% margin of error and a 95% confidence level. A 10% attrition rate was added to the minimum sample size to give 1502 and rounded to 1600 for ease of allocation.

Study participants

The study population comprised adults between 18 and 64 years of age who had resided in the four selected communities for at least 5 years to ensure long-term exposure.[27] Choosing the 18–64 age group reduces the confounding effect of age and other age-associated comorbidities such as HTN and diabetes. Participants were excluded if they were unable or unwilling to provide informed consent, institutionalised persons (prisoners, nursing home residents), or unlikely or unable to participate in required study procedures as assessed by the lead researcher (OCO). Such participants included pregnant women or lactating mothers, menstruating females (excluded from urinalysis), critically ill persons, persons participating in another research study that adds significantly to the participant's burden, persons receiving chemotherapy for systemic cancer and prior solid organ or bone marrow transplant based on participant self-report.

Participant recruitment

This study required a total of 1600 participants (400 per community). The average number of adults per household in Nigeria is two.[28] Based on this, an estimate of 800 households (at least 200 households per community) was needed to achieve the sample size. After gaining entry into each community through the leaders and community executives, participants were selected using the multistage sampling technique, starting from the community leader's house (first cluster) and then every other house clockwise. All members of every household in a selected house who met the inclusion criteria were invited to the community hall at a specified date for data collection. Halls in worship centres were used on days when the town hall was not available. Recruitment lasted 10 months, including repeat visits 3 months after the initial round of recruitments to repeat the laboratory tests of those who had either proteinuria or reduced kidney function (glomerular filtration rate, GFR<60 mL/min/1.73 m^3).

Study instruments

The questionnaire used for the survey was developed by combining and modifying the WHO STEPwise approach to surveillance (WHO STEPS) and the benzene, toluene, ethylbenzene and xylene (BTEX) exposure assessment questionnaire. Additionally, we integrated findings from an initial qualitative study conducted in one of the study communities into the questionnaire to achieve a more context-sensitive tool. A pilot study was conducted with a sample of 200 from one of the communities to determine the internal consistency of the questions assessing environmental exposures. The minimum sample size calculated for this purpose was 176, assuming minimum acceptable Cronbach's alpha coefficient as 0.65, expected Cronbach's alpha as 0.75, significance level=0.05, p=80% and a non-response rate of 15%.[29] Cronbach's alpha for 21 items assessing household chemical and occupational exposure was 0.65, considered acceptable.[30] The questions evaluating air pollution risk perception were a combination of open-ended and multiple-option closed-ended questions, and their reliability was assessed on face value only as Cronbach's alpha was not applicable.

The final questionnaire had 157 items distributed across five topic areas: demographic characteristics, behavioural characteristics, exposure history, risk perception, medical history and clinical measurements. A summary of the contents is as follows: (1) demographic characteristics: age, sex, marital status, level of education, work status and occupation; (2) behavioural: social habits, diet and physical activity; (3) health status (self-reported): comorbidities, health behaviours, family history, drug history, history of hospitalisations in the last 1 month and history of mortalities in the household; (4) exposure history: duration of residence in community, ambient air pollution exposure, occupational exposures, household air pollution exposure, household chemical exposures and place of birth; (5) air pollution risk perception: subjective assessment of ambient air, source of air pollution, health effect associated with air pollution, opinion on air pollution control (effective not effective), who is responsible for air pollution control, their role in control (if any); (6) clinical measurements: weight, height, waist and hip circumference and blood pressure and (7) laboratory measurements: dipstick urine examination, serum creatinine and glucometer test for blood sugar.

Demographic, behavioural, exposure history and some clinical data were based on participants' self-reports; however, measures were taken to minimise information bias. Follow-up questions, a request for names of medications, and an inspection of these medicines were measures taken to verify the participants' medical history. Furthermore, the clinical and laboratory measurements helped confirm some of their reported diagnoses, but verifying other demographic, socioeconomic and behavioural history was less feasible. However, during the consent process, the participants were given thorough information on the purpose and importance of the study and their roles in helping to produce a credible outcome.

Serum creatinine was assessed using the compensated isotope dilution mass spectrometry (IDMS)-traceable Jaffe-based non-enzymatic creatinine—picrate method on an Abbott Architect C4000 machine, a fully automated chemistry analyser primarily used for chemical tests. GFR was estimated using the CKD Epidemiology Collaboration (CKD-EPI) equation (without the ethnicity criteria).[31 32]

Citizen science instruments

The citizen science component of this study was necessary because of the lack of continuous air monitoring data in the study area and in most of Nigeria.[33] The air monitoring devices were similar in size to smartphones, with some larger and some smaller. Four devices could measure multiple parameters, namely $PM_{2.5}$, PM_{10}, CO_2 or VOC, relative humidity and temperature, while the devices for O_3 and NO_2 measured these pollutants exclusively. Record sheets bound in a booklet were provided to each of the eight volunteers to record ambient air pollutant measurements for the 4 weeks. Each sheet contained a list of all air pollutants ($PM_{2.5}$, PM_{10}, NO_2, CO_2, VOC and O_3) and columns to enter measurements two times per day (8–10 am and 5–6 pm) for 3 days per week. Measurements of relative humidity and temperature were also entered for each measurement time. The environmental scientists trained the volunteers to download a free GPS application onto their smartphones and measure the geographical coordinates of their location during air monitoring.

Variables

The primary outcome variable was CKD, defined as an estimated GFR (eGFR)<60 mL/min irrespective of the presence of proteinuria or the presence of ≥trace or 1+dipstick proteinuria irrespective of the eGFR.[34] The primary exposure variable is the air pollution exposure status (exposed/less exposed), defined by the proximity of a community to the Warri refinery and supported by the total hazard quotient (HQ). The total HQ is the sum of the HQs of individual air pollutants exceeding minimum WHO acceptable limits. The HQ was calculated as follows:

HQ concentration of pollutant/WHO acceptable limit; HQ>1 represents higher than acceptable exposure.[35]

The secondary exposure measure was a community's urbanicity, that is, rural versus urban/semiurban. Although ambient air pollution sources may be relatively more in urban compared with rural communities due to industrial activities and heavier traffic, household air pollution might be relatively higher in rural communities due to the use of solid fuels. Therefore, the consequent exposure may not be distinguishable based on urbanicity.

The predictor variables included sociodemographic characteristics such as age, gender, socioeconomic status, level of education, annual household income and occupation. However, to avoid multicollinearity, the level of education was selected as the indicator of socioeconomic status, being the most reliable bridge of socioeconomic conditions across generations.[36] Other variables include (1) health status: diabetes, HTN, obesity and family history of CKD; (2) behavioural characteristics: smoking (current/previous/never), alcohol use (current/previous/never and mild/moderate/heavy) habitual nephrotoxin exposure (use of skin lighteners, herbal mixtures, non-steroidal anti-inflammatory drugs (NSAIDS), hair dyes, physical activity (mild/moderate/severe), time spent outdoors, diet (salt intake, fast foods) and (3) other exposures: occupational exposures, use of pesticides, household chemicals, household cooking fuel and water source.

The potential confounders of the association between air pollution exposure and CKD include age, smoking, nephrotoxic exposures (NSAIDs, skin bleaching and hair dyes), HTN, diabetes and obesity. Other contributing factors include occupational exposure, household air pollution and chemical exposure. The confounders were preidentified based on confounding variables addressed in previous studies and known risk factors of CKD. However, some variables such as HTN, diabetes and obesity cannot be regarded as actual confounding variables because although related to both exposure and outcome, they also occur along the causal pathway. Further measures were taken during data analysis to identify and adjust confounders in the final logistic regression models.

Patient and public involvement

Eight community volunteers participated as citizen scientists in this study. They were involved in data collection (air pollution monitoring), collation and interpretation. We shared the study results with the volunteers at a debriefing meeting. The authors also plan to disseminate the complete study findings to the volunteers, study community members and their leaders.

Data analysis

Data analysis was performed using recommended practical guidelines for SPSS statistics[37] and grounded in relevant theoretical assumptions, definitions and concepts in epidemiology.[38] The primary outcome (prevalence of CKD) and other covariates for the four communities are reported as frequencies and percentages. Categorical variables were compared between groups using the χ^2 test with adjustment for non-independent observations. In contrast, continuous variables were compared using the student t-test or analysis of variance followed by Tukey HSD post-hoc analysis when groups were >3.

The logistic regression analysis was used to create three consecutive prediction models with CKD as a binary outcome. Predictor variables included age, smoking and other traditional risk factors for CKD (smoking, HTN and diabetes) based on previously published literature and variables found to be statistically significant ($p \leq 0.15$) in a univariate logistic regression. A lower significance threshold was chosen for this specific analysis to prevent ignoring variables that could be important. A further

logistic mixed model was performed using *R* to adjust for intrahousehold homogeneity and its design effect.

Missing data occurred randomly. Less than 5% of data entries that were grossly incomplete or not identifiable were removed during collation. The statistical software excluded other missing data, if any, from specific subgroups before analysis. Outliers or influential data points were identified and excluded before analyses.

RESULTS

Of the 1600 (400 per community) adults aged 18–64 who were systematically selected and invited for the health survey, 1460 from 804 households enrolled and completed the study, giving a response rate of 91.25% (online supplemental file 1). The names of the four communities are deliberately designated 'nearest/semiurban', 'near/urban', 'far/urban' and 'farthest/rural' to maintain confidentiality and protect the privacy of the participants in this research.

Sociodemographic and behavioural characteristics of participants

Table 1 shows the demographic characteristics of the participants. Most participants were females (71%), and there was no significant difference across the four communities (χ^2=0.079, df=3, p=0.994). The overall mean age was 44±13 years; it was highest in the far/urban community and lowest in the nearest/semiurban community (F=11.092, df=3, (p≤0.001). The highest proportion of all participants were 31–50 years old. However, the far/urban had a significantly higher proportion of participants above 50 years.

About two-thirds of all participants (68.5%) had at least a secondary level of education; the proportion was slightly higher in the nearest/semiurban (71.6%) compared with other communities. Self-employed persons constituted 62.3% of all participants; only 5.9% were government employees; 39.2% were in occupational class I, although most were small business owners. Half of all participants (50.6%) earned less than the minimum annual wage in Nigeria, that is, N360 000 (£633), and there was no statistically significant difference across the four communities.

Table 2 shows the participants' behavioural characteristics, including dietary habits and lifestyle. All communities' average fruit and vegetable consumption was one serving per day, 3 days per week. None of the respondents ingested adequate fruits or vegetables based on the WHO recommendation of ≥5 servings daily, every day in a typical week. Salt intake was excessive in 15% of all participants; it was highest (23.4%) in the farthest/rural community compared with other communities (χ^2=26.888, df=3, p≤0.001). Local fish (caught from community river) consumption was most prevalent (33.9%) in the farthest/rural compared with other communities (χ^2=23.873, df=3, p≤0.001). Overall, the participants' dietary habits were suboptimal.

Physical activity was mild, moderate and vigorous, in 47.2%, 24.9% and 27.9% of all participants, respectively. Vigorous physical activity was lowest (24.4%) in the near/urban and highest (35.0%) in the farthest/rural community (χ^2=18.136, df=3, p=0.006). A higher proportion of residents in the nearest/semiurban (52.2%) spent more time outdoors than indoors compared with other communities (χ^2=17.078, df=3, p=0.009). Alcohol intake was common among all participants (41%). However, only 6.3% reported heavy intake. Current and heavy alcohol intake was highest in the nearest/semiurban community (50.8% and 8.0%, respectively). Only 3.8% of all participants currently smoked, and there was no statistically significant difference across communities. However, exposure to secondhand smoke was 4.9% in the nearest/semiurban community compared with ≤3.0% in the other communities (χ^2=8.546, df=3, p=0.036). In summary, alcohol ingestion was more prevalent than smoking in all communities. However, the community nearest to the refinery had a relatively higher prevalence of secondhand smoking and alcohol use. Physical exertion was more common in the rural community than in urban and semiurban ones.

Table 3 details the exposure history of the participants across all communities. The use of petrochemical products was prevalent across all communities (80%); it was higher among residents in nearest/semiurban (84.3%) and farthest/rural communities (85.1%) compared with the urban communities (χ=23.744, df=3, p≤0.001). Almost half (48.9%) of participants reported occupational exposure to toxic chemical products or dust. Again, it was higher in the nearest/semiurban (55.8%) and farthest/rural (54.8%) communities compared with the urban communities χ^2=22.767, df=3, p≤0.001). Eighteen per cent of residents in the nearest/semiurban community used moth balls, compared with 11.5%–16.3% in the other communities (χ^2=10.542, df=3, p=0.014). Therefore, exposure to toxic chemicals was common in all four communities but relatively higher in the community nearest to the refinery and the farthest rural community.

Only 3.4% of all participants currently fished, while 14% had fished in the past. The nearest/semiurban community had the highest proportion of residents (18.4%) who fished in the past, compared with the other communities (χ^2=15.301, df=3, p=0.002), but currently, only 5% fished. Fishing history was obtained to further explore the suggestions from our prior qualitative study[39] that the local river was contaminated with petroleum and had adversely affected their source of livelihood. Fishing was also assumed to be a potential source of chemical exposure. Propane gas was the most commonly used cooking fuel (62.2%) among all the participants; this was relatively higher among residents in the nearest/semiurban community (71.8%) compared with the other communities (χ^2=24.421, df=3, p≤0.001). There was no statistically significant difference across the four communities regarding using herbal remedies, skin lighteners, hair dye, kerosene, pesticides and drinking water sources.

Table 1 Demographic profile of participants in the four communities

Parameter	Communities				Total	Test	P value
	A 3.0 km	B 3.5 km	C 10 km	D 13 km	n=1460	Statistic H, F or χ^2 (df)	
Sex (%)							
Male	28.5	28.7	28	27.8	28.2	0.079	0.994
Female	71.5	71.3	72	72.2	71.8	(3)	
N	369	328	400	363	1460		
Age (years)*						11.092	<0.0001
Mean±SD	41±12	45±12	46±13	43±13	44±13	(3)	
Age category (years) %							
≤20	3.8	0.6	5.5	6.3	4.2	54.991	<0.0001
21–30	15.2	11.0	7.8	11.8	11.4	(5)	
31–40	31.2	28.4	20.3	24.8	26.0		
41–50	23.6	26.5	24.8	28.9	25.9		
51–60	15.7	9.5	23.5	16.8	19.0		
61–65	10.6	14.0	18.3	11.3	13.6		
N	369	328	400	363	1460		
Duration of stay in area (years)							
Median (IQR)	11 (6–20)	15 (7–28)	12 (7–23)	10 (5–20)	11 (6–21)	H=24.147	<0.0001
Educational level (%)							
No formal	3.3	7.6	3.3	6.3	5	χ^2=41.906	<0.0001
<Primary	5.1	15.8	8	4.7	6	(5)	
Primary	20.1	18.9	23.5	19.3	20.5		
Secondary	53.4	42.4	45.8	42.4	46.1		
University	14.9	21.3	16.8	25.9	19.6		
Postgraduate	3.3	4	2.8	1.4	2.8		
N	369	328	400	363	1460		
Marital status (%)							
Never married	25.3	21.3	27.8	22.3	24.4	χ^2=30.952	0.009
Currently married	52.9	57.0	49.1	56.2	53.6	(5)	
Separated	5.2	2.4	2.8	6.1	4.1		
Divorced	3	4.9	4.0	1.1	3.2		
Widowed	10.4	12.5	15.0	12.1	12.6		
Cohabiting	3.3	1.8	1.3	2.2	2.1		
N	367	328	399	363	1457		

*Rounded up to the nearest whole number.
ANOVA, analysis of variance; df, degree of freedom; F, ANOVA test; H, Kruskal-Wallis's test; χ^2, Chi-square test.

Air pollutant concentrations in the four communities over 4 weeks

We measured six air pollutants: particulate matter ($PM_{2.5}$ and PM_{10}), volatile organic compounds (VOC), ozone (O_3), nitrogen dioxide (NO_2) and carbon dioxide (CO_2). CO_2 is not strictly regarded as an air pollutant because it primarily occurs naturally; however, anthropogenic CO_2 emissions occur and are the greatest greenhouse gas and contributor to climate change.[40] The mean concentrations of the air pollutants were computed except for NO_2 because the NO_2 air detector was not sensitive enough to detect values in the 0.0–0.1 ppm range, and since the WHO permissible limit (0.005 ppm) is within this range, frequency counts of readings exceeding the WHO permissible limits were recorded. The overall mean concentrations of $PM_{2.5}$, PM_{10}, VOC and CO_2 were higher than the WHO minimum allowable limits in all four communities, while O_3 was within acceptable limits. The mean concentrations for most air pollutants

Table 2 Behavioural characteristics of participants in the four communities

Variables		A n=369 %	B n=328	C n=400	D n=383	Total n=1460	P value
Fruit intake	Adequate*	30.1	27.4	28.5	34.2	30.0	0.218
Vegetable intake	Adequate*	12.5	9.5	9.5	12.1	10.9	0.390
Salt intake	Excess	11.9	12.2	12.5	23.4	15.0	<0.001
Local fish intake	Regular	25.2	17.7	25.3	33.9	25.7	<0.001
Physical activity	Mild	48.8	45.1	51.2	43.3	47.2	0.006
	Moderate	24.4	30.5	23.5	21.8	24.9	
	Vigorous	26.8	24.4	25.3	35.0	27.9	
Time spent	Mostly outdoors	52.2	39.0	40.8	44.6	44.3	
Outdoors	Mostly indoors	31.1	41.2	41.8	35.7	37.4	0.009
	Both equally	16.7	19.8	17.4	19.8	18.3	
Alcohol intake	Current	50.9	36.3	34.0	43.0	41.0	<0.001
	Former	7.3	15.9	14.0	10.7	11.9	
	Never	41.8	47.8	52.0	46.3	47.1	
Alcohol intake	Heavy	8.4	6.1	5.0	5.8	6.3	0.252
Tobacco smoking	Current	4.6	2.4	3.5	4.7	3.8	0.368
	Ever smoked	11.7	8.5	9.0	11.3	10.1	0.399
	Heavy smoker	3.3	1.2	2.3	3.3	2.5	0.254
Smokeless tobacco	Current use	1.9	0.6	1.3	0.8	1.2	0.394
	Heavy use	1.4	0.3	1.0	0.6	0.8	
Secondhand smoking	Exposed	4.9	1.8	3.0	1.7	2.9	0.036
	Heavy exposure	0.8	0.0	0.3	0.0	0.3	0.120

df=1.
*Adequate refers to at least average consumption for the population, which was one serving a day, 3 days/week.

were significantly higher in the communities near the refinery compared with the farther communities; however, $PM_{2.5}$ levels were comparable among the four communities (online supplemental file 2). The mean $PM_{2.5}$ concentration in all four communities ranged from 22 to 28 µg/m^3, corresponding with moderate air quality.[41]

The calculated total HQ for $PM_{2.5}$, PM_{10}, VOC and CO_2 based on the WHO minimum allowable limits were elevated in all four communities (online supplemental file 3). $PM_{2.5}$ and PM_{10} had the highest HQ in all communities compared with other pollutants. The total HQ was relatively higher for the communities (11.27, 11.63), which are close to the refinery compared with those farther (9.63, 10.68), but this difference was not statistically significant (F=0.038, p=0.989).

Prevalence of chronic kidney disease and other kidney-related health outcomes

The primary objective of this study was to determine the relative prevalence of CKD and other kidney-related outcomes in the four chosen communities (table 4). The overall prevalence of CKD, based on dipstick proteinuria and eGFR<60 mL/min was 12.3% (95% CI=10.7%–14.1%, n=180); age-adjusted prevalence was 11.8% (95% CI=7.0%–19.5%, n=180). The prevalence was highest in nearest/semiurban (17.9%) compared with 13.1%, 10.5% and 8.0% in the near/urban, far/urban and farthest/rural communities, respectively (χ^2=18.292, p=0.004). Proteinuria was detected in 6.8% (n=99) of participants; 3.6% had trace proteinuria and 3.2% had ≥1+. Ninety-six (6.6%) participants had reduced eGFR<60 mL/min; of these, 15.6% had proteinuria compared with 6.2% among those with higher eGFR (χ^2=12.695, df=1, p=0.001). Reduced eGFR<60 mL/min was more prevalent in the nearest/semiurban (7.6%) and far/urban communities (7.8%) but did not reach statistical significance. The highest proportion (40%) of participants with CKD were in stage 3A, that is, eGFR 45–59 mL/min.

Attempts to repeat the kidney function tests after 3 months to establish chronicity in all participants with abnormalities were unsuccessful. Efforts included repeated visits to the respective communities, counselling via phone calls and additional arrangements to collect biological samples in any of the two tertiary hospitals serving the area. The main reasons for non-response were

Table 3 Toxic exposure history of participants in the four communities

Variables		A n=369 %	B n=328	C n=400	D n=383	Total n=1460	P value
Herbal remedy use	Yes	41.7	41.8	39.1	40.9	40.8	0.876
Skin lightener use	Yes	14.5	14.5	11.6	10.7	12.7	0.307
Hair dye use	Yes	22.0	16.2	18.2	21.0	19.4	0.219
Kerosene use	Yes	46.8	42.4	48.2	39.8	44.4	0.081
Moth balls/crystals use	Yes	18.8	16.3	11.9	11.5	14.4	0.014
Exposure to pesticide	Yes	55.0	56.7	50.7	50.4	53.1	0.241
Petrochemical exposure*	Yes	84.3	72.3	77.8	85.1	80.0	<0.001
Household chemicals	Yes	71.5	66.8	71.8	78.2	72.2	0.009
Occupational exposure*	Yes	55.8	40.5	44.8	54.0	48.9	<0.001
Fishing history	Ever	18.4	8.2	14.8	13.8	14.0	0.002
	Current	5.1	2.3	0.3	6.0	3.4	<0.001
Drinking water source	Well, rivers	0.5	0.6	0.0	1.4	0.6	0.114
	Others†	99.5	99.4	100.0	98.6	99.4	
Cooking fuel	Propane gas	71.8	58.8	55.3	63.1	62.2	<0.001
	Others‡	28.2	41.2	44.8	36.9	37.8	

df=3.
*All sources.
†Bottled water, indoor tap water, outdoor standing pipe, borehole and sachet water.
‡Firewood, coal and kerosene.

a lack of interest and fear of confirming a new diagnosis. Of the 96 persons who had eGFR<60 mL/min, only 12 had repeated tests and of these, 8 (66.7%) were confirmed, while the other four had slightly higher GFR than initially obtained, moving them from stage 3A (eGFR 45–59 mL/min) to stage 2 CKD (60–89 mL/min). Similarly, of the 99 persons with proteinuria, 26 had repeated tests; of these, 22 (84.6%) remained positive. Four persons who had only proteinuria at the first assessment were found to additionally have reduced eGFR (<60 mL/min) at the second assessment.

The overall prevalence of HTN was 33.3% (95% CI=30.9%–35.7%, n=486); it was higher in the near/urban (38.4%) and far/urban (37.5%) communities compared with the near/semiurban (33.6%) and farthest/rural communities (23.7%), χ^2=22.148, df=3, p≤0.001. The prevalence of diabetes mellitus (DM) was 6.0% (95% CI=4.8%–7.3%, n=87); it was higher in urban communities (7.3% and 6.8%) but did not reach statistical significance. Obesity defined using the participants' BMI and WHR was 28.2% (95% CI=25.9%–30.5%, n=411) and 35% (95% CI=32.1%–37.0%, n=504), respectively; there was no statistically significant difference across the four communities. However, mean WHR among males was higher in the far communities by 0.01 units. A summary of the prevalence of four primary health outcomes for the four communities is shown in figure 1.

Risk factors of CKD

A higher proportion (60.6%) of those with CKD resided near the refinery compared with 46% of the non-CKD population (χ^2=18.292, df=1, p≤0.001). Similarly, 83.9% of those with CKD lived in urban or semiurban settings compared with 73.9% of the non-CKD population (χ^2=8.418, df=1, p=0.003). A higher proportion of the CKD population were older and had low levels of education than the non-CKD population. Less than secondary education was reported by 45.5% of those with CKD compared with 29.5% of those without CKD (χ^2=38.324, df=3, p≤0.001). Slightly more than half (56.7%) of those with CKD were >50 years old, compared with 29.2% of those without CKD (χ^2=58.830, df=5, p≤0.001). There were no statistically significant differences regarding sex and annual income.

There was no statistically significant difference between CKD and non-CKD populations regarding most behavioural factors except time spent indoors/outdoors. Among the CKD group, a higher proportion spent more time indoors than outdoors (47.2% and 42%, respectively); conversely, among those without CKD, a higher proportion spent more time outdoors than indoors (44.6% and 36.1%, respectively) (χ^2=11.515, df=2, p=0.003). A higher proportion of the CKD group currently smoked compared with the non-CKD group (6.1% and 3.5%, respectively); the difference was not statistically significant. Similarly, a relatively higher proportion of the CKD population reported positive dietary habits (adequate

Table 4 Clinical and laboratory parameters of participants in the four communities

Parameter	Region				Total (n=1460)	P value
	A (n=369)	B (n=328)	C (n=400)	D (n=363)		
Proteinuria						
Positive	12.0	9.1	3.8	2.8	6.8	<0.001
Negative	88.0	90.9	96.2	97.2	93.2	
Serum creatinine	0.9±0.3	0.9±0.5	0.9±0.2	0.9±0.2	0.9±0.3	0.209
eGFR<60 mL/min						
Yes	7.6	5.2	7.8	5.5	6.6	0.359
No	92.4	94.8	92.2	94.5	93.4	
Mean±SD	91.2±20.9	92.4±18.4	89.7±19.9	92.1±19.3	91.3±19.7	0.390
CKD (proteinuria+eGFR<60 mL/min)						
Yes	17.9	13.1	10.5	8.0	12.3	0.004
No	82.1	86.9	89.5	92.0	87.7	
BMI	%					
Under weight	8.1	5.9	9.0	5.5	7.2	0.293
Normal	38.2	38.1	37.0	36.4	37.4	
Over weight	23.8	28.0	29.3	27.8	27.3	
Obese	29.7	28.0	24.7	30.3	28.2	
Mean±SD	27.2±7.9	27.1±6.7	26.9±8.7	27.4±6.8	27.1±7.6	0.497
Obesity (WHR)						
Yes	40.1	32.1	36.0	31.3	35.0	0.064
No	59.9	67.9	64.0	68.7	65.0	
Mean±SD$^{\alpha}$	0.88±0.06	0.89±0.08	0.90±0.08	0.90±0.05	0.89±0.07	0.011
Mean±SD$^{\beta}$	0.89±0.09	0.88±0.12	0.89±0.08	0.90±0.07	0.89±0.09	0.474
Hypertensive*						
Yes	33.6	38.4	37.5	23.7	33.3	<0.001
No	66.4	61.6	62.5	76.3	66.7	
Diabetes mellitus*						
Yes	4.1	7.3	6.8	5.8	6.0	0.353
No	95.9	92.7	93.3	94.2	94.0	
RBS	99.8±33.5	98.7±42.9	84.6±38.0	92.1±36.6	93.5±38.2	<0.001

df=3.
*Based on history, medication use and clinical measurements.
CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; RBS, random blood sugar; WHR, waist-hip-ratio; α, males; β, females.

fruit and vegetable intake, normal salt intake) but did not reach statistical significance.

There was no statistically significant difference between CKD and non-CKD populations regarding most toxic exposures except using hair dyes. Among the CKD group, a higher proportion used hair dyes than the non-CKD group (25.4% and 18.5%, respectively) (χ^2=4.646, df=1, p=0.039). Although a slightly higher proportion of the CKD group compared with the non-CKD group used herbal remedies, kerosene, mothballs, household chemicals, wood or fossil fuel for cooking, consumed locally caught fish and had exposure to petrochemical and occupational toxins, these differences were not statistically significant. A higher proportion of the CKD population had HTN (50.6%) compared with 30.9% among those without CKD (χ^2=27.568, df=1, p≤0.001). Similarly, a higher proportion of the CKD population had diabetes (11.7%) compared with 5.2% among those without CKD (χ^2=11.936, df=1, p=0.002). There were no statistically significant differences regarding obesity measured by BMI or WHR.

Independent risk factors of CKD

A binary logistic regression analysis with adjustment for confounding factors was used to estimate the probability of CKD among the study population. The result of the final

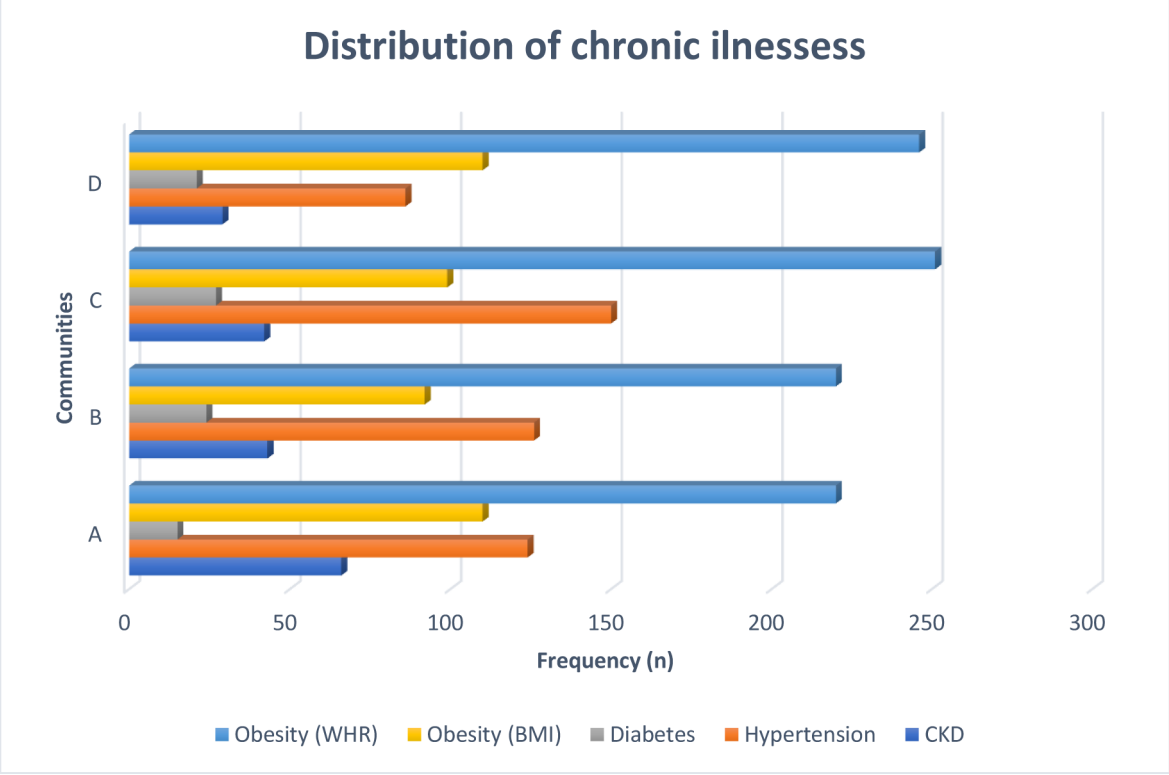

Figure 1 Distribution of the major disease outcomes among participants in the four communities. A, nearest/semiurban; B, near/urban; C, far/urban; D, farthest/rural. Chi-square test: CKD (χ^2=18.292, p=0.004), hypertension (χ^2=22.148, p<0.001), obesity (χ^2=9.699, p=0.293) and diabetes (χ^2=3.908, p=0.353). BMI, body mass index; CKD, chronic kidney disease, WHR, waist-hip-ratio.

model is shown in table 5. Place of residence, increasing age, level of education and HTN were the independent risk factors of CKD. Other associated factors such as marital status, diabetes, time outdoors/indoors and use of hair dyes did not statistically significantly influence the probability of CKD in the adjusted model. Residing near the petrochemical industry increased the probability of having CKD by twofold (100%), while having at least secondary-level education reduced the probability by 37%. HTN increased the probability of having CKD by 61%, while a unit increase in age resulted in a predicted 2% increase in the probability of having CKD. The ROC for the regression analysis produced an AUC=0.70 (95% CI=0.65–0.73); SE=0.021; p<0.001, sensitivity=75.3%; specificity=56.1%, indicating that the model has an acceptable discriminating ability.[42] A further mixed logistic model, conducted on *R* to account for design effects due to intrahousehold homogeneity, showed that only increasing age was a statistically significant independent risk factor for CKD, table 6.

DISCUSSION

This cross-sectional study aimed to determine whether persons residing near petrochemical industries are exposed to ambient air pollution and whether long-term exposure is associated with CKD. Our findings show that all communities studied were exposed to unacceptable air pollutantion but was cumulatively worse in the communities closest to the petrochemical refinery. The prevalence of CKD and HTN was significantly higher in the communities nearest to the refinery, but there was no significant difference in the prevalence of diabetes and obesity. Proteinuria was prominent in more exposed communities, supporting the possibility of inflammatory glomerular injury as the pathophysiological mechanism for air pollution-associated kidney disease. Social and biological risk factors for CKD were generally prevalent in the study sample. However, those who resided near the refinery and in the urban communities had poorer health indices than those in the farthest/rural community.

This study showed that the prevalence of CKD was highest in the community closest to the petrochemical refinery compared with the other communities. The prevalence of CKD was 18%, 13%, 10.5% and 8%, respectively, from the closest to the farthest community, suggesting a possible dose–response relationship. The current study found that people who have lived in communities near (3–3.5 km) the petroleum refinery for at least 5 years have a twofold higher risk of developing CKD than people who have lived at least 10 km away. However, this estimate was not sustained after adjusting for intrahousehold homogeneity, possibly because of the smaller intragroup sizes, variability and rarity of events.

Table 5 Multivariate binary logistic regression analysis for independent risk factors of CKD (n=1381)

Variables	B	AOR (CI)	P value
Age	0.02	1.02 (1.005 to 1.04)	<0.001
Gender			
Male	1		
Female	0.21	0.81 (0.55 to 1.19)	0.287
Place of residence			
Far (C+D)	1		
Near (A+B)	0.69	2.00 (1.43 to 2.81)	<0.001
Marital status			
Never married	1		
Currently married+cohab.	0.19	0.91 (0.47 to 1.79)	0.914
Separated/divorced	0.52	0.76 (0.46 to 1.24)	0.756
Widowed	0.09	0.54 (0.26 to 1.13)	0.545
Level of education			
Primary and below	1		
Secondary and above	0.46	0.63 (0.44 to 0.91)	0.013
Time outdoors			
Mostly outdoors	1		
Mostly indoors	0.13	1.14 (0.80 to 1.61)	0.465
Use of hair dyes	0.27	1.31 (0.89 to 1.94)	0.175
Hypertension	0.48	1.61 (1.12 to 2.31)	0.010
Diabetes	0.39	1.64 (0.83 to 2.66)	0.180
WHR	0.94	1.64 (0.26 to 10.28)	0.595

Omnibus test = <0.001 | Hosmer and Lemeshow=0.639 | accuracy=87.6 | Cox & Snell R^2=0.049 | Nagelkerke R^2=0.094.
AOR, adjusted OR; CKD, chronic kidney disease; WHR, waist hip ratio.

Table 6 Generalised logistic mixed model showing age as an independent risk factor for CKD

	CKD_Fact		
Predictors	ORs	CI	P value
Age	1.26	1.09 to 1.45	0.002
Residence (ref: near)	0.37	0.04 to 3.78	0.400
Sex (ref: male)	0.18	0.03 to 1.08	0.060
Hypertension	4.10	0.72 to 23.54	0.113
Level of education	0.82	0.15 to 4.62	0.826
Random effects			
σ^2	3.29		
$\tau_{00\ iddo\_Fact}$	1267.42		
ICC	1.00		
N_{iddo_Fact}	804		
Marginal R^2/conditional R^2	0.01/0.99		

CKD, chronic kidney disease; ICC, intraclass coefficient.

Our findings are consistent with those from the few studies in similar exposed settings. Yuan *et al*, in two separate cross-sectional studies conducted in Taiwan, found that residing near a petrochemical complex and a naphtha cracking complex was associated with a 1.5 and 2.7-fold increase in the probability for CKD, respectively.[43 44] In the two studies, those residing within a 10 km radius were regarded as highly exposed, while those farther away were classified as less exposed. To assess exposure, the researchers measured individual urine concentrations of heavy metals and polyaromatic hydrocarbons. While this may be a valid exposure assessment, there is a possible bias of reverse causation. In another study, Benedetti *et al* reported that living near industrial pollution sources in Italy was associated with a 1.28-fold increase in the probability of hospitalisations from kidney diseases in adult males aged 20–59 years but not in females and other age groups.[45] The researchers combined the location of the residence with air dispersion data modelling to define exposure to cadmium and $PM_{2.5}$. They used existing hospitalisation data from the national discharge registers in an ecological study design, which does not allow for adequate consideration of confounding variables at the individual level.

Emerging studies show that long-term exposure to air pollution is associated with increased risk for CKD. Seven systematic reviews on the subject, including one by the current authors, were published between 2020 and 2022, demonstrating that APA-CKD is a growing research area.[10 20 46–50] Although all the systematic reviews/meta-analyses suggest an increased risk for CKD (pooled effect size range from 1.04 to 1.70), they were limited by inadequate number of individual studies, population and methodological heterogeneity. A majority of the included studies were conducted in Asia and fewer in the USA and Australia; there were limited studies from Africa. Furthermore, most of the studies were on the general population and fewer on susceptible groups like veterans and pregnant women, but the findings were still mostly positive. Although the effect sizes reported in the general population are not as high as those from populations living near industries, it is understandable because proximity to pollution sources combined with other social determinants of health are likely to amplify CKD risks. Regardless of the highlighted limitations, there appears to be increasing evidence for APA-CKD, only few studies have reported no association for specific pollutants such as CO,[10 46] SO_2[46] and PM.[51 52]

In this study, proteinuria (not reduced eGFR) was mainly responsible for the significant differences in CKD prevalence observed across the four communities; this suggests that the pathophysiology of kidney damage from air pollution may be through inflammatory glomerular damage. Air pollution causes a systematic effect mainly through inflammation and oxidative stress, leading to epithelial and endothelial cell injury, which has been

widely reported, especially in the lungs and cardiovascular system. However, evidence for a similar effect in the kidneys is emerging.[11 13 53] Specifically, an experimental study in mice showed that exposure to concentrated PM for 16 weeks was associated with glomerulosclerosis.[53] Similarly, other researchers have reported PM exposure associated with glomerulonephritis or albuminuria.[54–57] These findings support the need for urine protein assessment as part of routine medical screening at the community level and within health institutions, as part of general patient care. While serum creatinine is a late marker of kidney damage which only rises after significant kidney function is lost, proteinuria provides an opportunity for early detection and treatment of glomerular injury.[34]

The risk factors associated with CKD in this study were older age, lower educational level, proximity to the refinery, residing in urban communities, spending more time outdoors, using hair dyes, HTN and DM. Although there is no consensus across studies regarding independent risk factors of CKD, older age and HTN have been reported consistently. Other inconsistently reported predictors include gender,[58–60] DM,[58 60 61] family history of kidney disease,[62] HIV,[59 61] high BMI/obesity,[60 62] low BMI,[63] hyperuricaemia,[64 65] anaemia,[63 66] high educational level,[67] living in urban communities,[59 68] living in rural communities[67] and the presence of the APOL1 risk alleles.[61]

Almost half of the participants with CKD in this study did not have any of the leading CKD risk factors (HTN, diabetes or obesity), suggesting that CKD may have occurred through other exposures or mechanisms like air pollution. Similarly, Muiru *et al*[69] and Stanifer *et al*[68] found that 66% and 49% of their study sample with CKD in East Africa did not have the traditional risk factors, including HTN, diabetes and HIV. In contrast, Nakanga *et al*,[67] also in East Africa, showed that only 19.4% were free of these leading CKD risks, while Hodel *et al*[63] reported that 15% of 952 outpatients in a hospital in Tanzania had no known CKD risk (including HTN, diabetes, anaemia, tuberculosis and schistosomiasis). The absence of the traditional risk factors in persons with CKD typically raises suspicion for other environmental exposures or genetic predispositions. In the current study, the absence of traditional CKD risk factors in many participants and the high prevalence of CKD observed among people more exposed to air pollution support the likelihood of APA-CKD. Nevertheless, having traditional risk factors of CKD does not necessarily exclude air pollution as a possible predisposing factor since air pollution has been associated with all three traditional risk factors of CKD (DM, HTN and obesity). Therefore, regardless of whether the cause of CKD seems evident, it is reasonable to obtain a thorough environmental exposure history.

Some researchers spread across the health sciences, environmental sciences and geography have measured and reported air pollution monitoring data in the Niger Delta region. The most recent study conducted in Warri from 2017 to 2018 showed a significant inverse relationship between distance from the refinery and concentrations of $PM_{2.5}$, PM_{10}, NO_2 and VOC; however, H_2S was comparable at all points.[70] The authors noted that the air pollutants measured exceeded the WHO acceptable limits in all measuring points regardless of distance. However, the mean concentrations of $PM_{2.5}$, PM_{10}, NO_2 and VOC recorded in our study were lower than those obtained. Two other studies conducted in Warri in 2014[71] and 2017[72] also reported higher levels of $PM_{2.5}$ and NO_2, respectively, compared with the current study. A possible explanation for the differences in the reported air pollutant concentrations could be that air pollutant levels have declined with the declining activities of the Warri refinery since it was officially closed in 2015. Second, the current study was conducted in the wet season, which is usually associated with lower pollutant levels than the dry season. Balogun *et al*[70] measured air pollutants weekly for 12 months, encompassing dry and wet seasons. Finally, technical differences may have influenced the results since the air monitoring equipment used varied across the studies.

Although all studies report high levels of air pollution in Warri and the Niger Delta, the inconsistent air monitoring techniques and data necessitate government-coordinated continuous air monitoring, risk communication and information transparency. Continuous air monitoring provides more comprehensive and representative data since variations in air pollutant concentrations are captured. Time and financial constraints make prolonged continuous air monitoring difficult in research. Without continuous air monitoring data, researchers have used air dispersion models and, more recently, AI-based collation systems, which are not as accurate.[73 74] Accessible air quality data will make air pollution studies more reliable and feasible, but more importantly, it may heighten public concern for air quality and stimulate positive behavioural changes.

The current study is an improvement on existing studies from the region because exposure classification was based on historical exposure from the location of residence and real-time air monitoring data with calculated HQ. We have demonstrated the feasibility and reliability of relatively cheap and portable air monitoring devices, which the government can adopt in low-resource settings to mitigate air pollution and climate change. Air quality monitoring, standards and management strategies are two of the nine clean air targets set by the United Nations.[33] Interestingly, as of 2021, only 57 out of 195 UN member countries had national air quality monitoring networks, and of these, 18 were either monitored periodically or suboptimally. Furthermore, one-third of the world's nations had no national legislation on ambient air quality standards, and among the countries that could introduce such guidelines, 31% have yet to adopt them. Expectedly, LMICs like Nigeria performed relatively worse, underpinning the need for cost-effective interventions while they await the promised, although delayed, support from wealthier member countries.[75]

The relatively lower prevalence of CKD and other NCDs in the rural community, despite some evidence of air pollution and toxic exposures, may be due to their relatively healthier lifestyle (higher physical activity, healthier diet and better social habits) and greener living environment. This information reinforces the need to emphasise cost-effective interventions such as positive behavioural changes and greener spaces to prevent NCDs attributable to air pollution and climate change. Other mitigation and adaptation measures have been widely discussed in the literature, including adopting newer efficient technologies, renewable energy, tax incentives and energy communities.[33 76 77]

Finally, air pollution and its attributable diseases are issues of global concern and cut across multiple disciplines and sectors. Findings from this study have implications for all stakeholders involved in air pollution risk management, CKD and NCD prevention and control. The key stakeholders include educators, health professionals (clinicians, public health professionals, community health workers), researchers, the industry, civil societies, non-governmental organisations, the general public and policymakers from community to international levels. Policies that promote kidney and environmental health are needed and should be integrated with air quality and climate change policies and interventions. There is a need for more stringent environmental protection regulations. However, positive behavioural changes are as relevant and require public education and inclusion in planning and implementing interventions. Furthermore, air pollution and other environmental health risks should feature prominently in health professionals' and other academic training curricula. Physicians should incorporate screening for social and environmental determinants of health into clinical assessment of patients with CKD and other relevant NCDs.

The limitations of this study include its cross-sectional design, so results should not be taken as evidence for causation. However, our study is sufficiently powered to suggest an association between air pollution and CKD, which creates the basis for future large longitudinal studies. The large population, probability sampling methods, valid outcome and exposure assessment and attention to confounding factors reinforce the cross-sectional design. However, the possibility of residual confounding cannot be excluded entirely. Another limitation is the *design effect* due to intrahousehold homogeneity and variable household sizes, which, although adjusted in the final mixed logistic model, may have led to a type II error. Sampling more households (clusters), intrahousehold sample selection and less variable cluster sizes should minimise large design effects in future studies. However, this may require more resources and pose challenges in a low-resource setting.

A further limitation was using one-off laboratory assessments to define CKD rather than repeating tests after 3 months as recommended. Despite repeated phone calls and visits, the authors' efforts to repeat tests for participants with abnormalities were unsuccessful. Many participants refused to have repeat tests due to fear of confirming a new diagnosis. Using more reliable renal function biomarkers such as cystatin C may remove the need to repeat creatinine assays in community-based studies. However, the high cost remains a significant challenge, necessitating more effective public research education and public inclusion in research, as was attempted in some parts of this study.

Due to resource limitations, urine protein excretion was measured using dipsticks, rather than albumin: creatinine ratio; this may have led to overestimation or underestimation of proteinuria. However, a urine dipstick is recommended to assess proteinuria and is highly specific, though less sensitive in detecting low levels of proteinuria. Finally, there is the possibility of information bias from participants' self-reports and exposure misclassification due to the spatial distribution of air pollutants and mobility of the participants. Measures were taken (described in the methods section) to minimise these biases.

Despite its limitations, this is one of the few existing large population cross-sectional studies on APA-CKD in susceptible populations. The relatively large sample size and probability sampling method increase the study power and enable adequate inferential analyses and effect size estimations. The extensive individual-level behavioural, clinical, social and environmental data allowed for the adjustment of confounding factors, making the effect sizes reported more valid and reliable. The outcome measurement was reliable as CKD was defined using the recommended Kidney Disease: Improving Global Outcomes (KDIGO) criteria, and serum creatinine was measured using an IDMS-traceable assay. Long-term air pollution exposure was assessed using location of residence. However, this was supported by historical evidence of long-term exposure and real-time air pollutant monitoring, which provided a surrogate for ambient air quality in recent times.

This study demonstrates the feasibility of innovatively combining citizen science with traditional research methods to achieve public inclusion in generating epidemiologic data from low-resource settings. The citizen science arm of this study includes community members as research collaborators and not just information providers, thereby stimulating their interests and concerns about the problem and empowering them with first-hand information. Finally, our findings and associated sizeable database can be used to plan future health interventions for the study population and similar oil and gas-situated communities.

In conclusion, we report a significantly higher prevalence of CKD in communities nearest a petrochemical refinery than in farther communities, and that long-term exposure to ambient air pollution may be associated with CKD and HTN, which corroborates with previously published evidence. In addition, we provide real-time evidence of unacceptable air pollutant concentrations in

the four communities and behavioural, environmental and socioeconomic factors possibly associated with air pollution health risks in already vulnerable populations. Our findings are generalisable to similarly exposed populations in low-resource settings.

Environmental injustices such as disparities in access to clean air threaten sustainable global health and well-being. Therefore, addressing air pollution-associated CKD will require a multifaceted approach and collaboration of key role players within and outside the government. By incorporating this issue into clinical practice, policy-making, medical education and public education campaigns, it is possible to reduce the burden of chronic diseases such as CKD, improve public health outcomes, minimise social inequity and sustain our environment. Large community-based epidemiological cohort studies are needed from LMIC to demonstrate the possible causal role of air pollution. Experimental studies are required to determine the mechanism of APA-CKD and interventions that might reverse this process, such as antioxidants.

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Contributors OCO conceived and designed the study and collected, entered, analysed and interpreted the data. OCO is the study's guarantor. OCO drafted the initial manuscript, while EC and LM, who served as the project supervisors, reviewed it and approved the final draft.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants. Ethical approval for this study was obtained from the Research Integrity Committee of the School of Health and Social Care, Edinburgh Napier University (#2782647) and the Human Research and Ethics Committee of the Hospital Management Board, Warri, Delta State, Nigeria (CHW/ECC VOL1/226). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

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