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Review article

Ambient air pollution and health in Sub-Saharan Africa: Current evidence, perspectives and a call to action.



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ABSTRACT

Background: People from low- and middle-income countries are disproportionately affected by the global burden of adverse health effects caused by ambient air pollution (AAP). However, data from Sub-Saharan Africa (SSA) are still scarce. We systematically reviewed the literature to describe the existing knowledge on AAP and health outcomes in SSA.

Methods: We searched PubMed, Medline-OVID, EMBASE and Scopus databases to identify studies of AAP and health outcomes published up to November 15, 2017. We used a systematic review approach to critically analyze and summarize levels of outdoor air pollutants, and data on health effects associated with AAP. We excluded occupational and indoor exposure studies.

Results: We identified 60 articles, with 37 only describing levels of AAP and 23 assessing the association between air pollution and health outcomes. Most studies (75%) addressing the relation between AAP and disease were cross-sectional. In general, exposure data were only obtained for selected cities in the framework of temporary international collaborative research initiatives without structural long-term continuation. Measurements of AAP revealed 10–20 fold higher levels than WHO standards. Of the 23 studies reporting health effects, 14 originated from South Africa, and most countries within SSA contributed no data at all. No studies, except from South Africa, were based on reliable morbidity or mortality statistics at regional or country level. The majority of studies investigated self-reported respiratory symptoms. Children and the elderly were found to be more susceptible to AAP.

Conclusion: AAP and its negative health effects have been understudied in SSA compared with other continents. The limited direct measurements of air pollutants indicate that AAP in SAA cities is high compared with international standards. Efforts are needed to monitor AAP in African cities, to identify its main sources, and to reduce adverse health effects by enforcing legislation.

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Panel 1

Research in context

Evidence before this study

No systematic review has summarized the scientific literature on levels of ambient air pollution (AAP) and its adverse health effects in sub-Saharan African (SSA) countries. Using keywords related to "ambient air pollution", "health outcomes" and "Sub-Saharan Africa", we searched, without language restrictions, PubMed, Medline-OVID, EMBASE, SCOPUS and other databases (the WHO repository, national agencies. African Index Medicus, abstracts at scientific conferences, and other grey literature) for relevant information up to November 15, 2017. The search identified 1402 references, among which we found four reviews. One review (2013), reported on the concentrations of particulate matter (PM) in eight SSA countries and two reviews (2015, 2017) using satellite-based estimates of ground level pollution by fine particles and ozone to estimate the contribution of various sources of air pollution to under-five and maternal mortality in Africa. Finally, a review produced by a Lancet commission (2017) stressed the need of ascertaining and mapping exposure to pollution in low and middle-income countries (LMICs) to address pollution-related diseases and to drive change in pollution policy. None of these reviews included primary epidemiological studies on the air pollution - health/ diseases continuum.

Added value of this study

Our systematic search compiled data on the levels of exposure to various ambient pollutants in SSA and their health effects, as assessed by population based studies. A major finding is that specific information on AAP and its health impacts in SAA is sparse and even completely lacking for the majority of African countries. Nevertheless, the available evidence indicates that AAP is a serious issue in SAA, since urban pollutant levels are generally 10 to 20 times above WHO standards and levels currently found in industrialized regions from high-income countries.

Implications of all the available evidence

From our systematic review of the existing evidence and from previous reviews, it may be safely concluded that a substantial proportion of people residing in SSA are harmed by the currently prevailing high levels of AAP, and that this situation is likely to worsen with the rapidly increasing urbanization in Africa. However, the evidence is still limited and should be strengthened by primary studies with a much larger geographical coverage than has been hitherto the case. Such primary studies should prioritise longitudinal designs and rely on truly representative quantitative measurements of groundlevel air pollution. We listed modifiable and non-modifiable risk factors of high exposure to AAP and enumerated major gaps to support plans for primary epidemiological studies and to establish and reinforce strict regulation in the SSA region.

1. Introduction

Sub-Saharan African (SSA) is undergoing an epidemiological transition, characterized by a heavy burden of both infectious diseases and non-communicable diseases (NCDs)(Dagadu and Patterson, 2015; Mboera et al., 2014; Nyaaba et al., 2017; Nyirenda, 2016). The rise in NCDs can be explained, at least in part, by risk factors that accompany changes in lifestyle (e.g. switch from traditional to western diet) and increased urbanization, with ambient air pollution (AAP) as one consequence. The impact of exposure to AAP on human health has been well documented(Brunekreef and Holgate, 2002; Kampa and Castanas, 2008). This ranges from minor upper respiratory irritation to serious chronic respiratory and cardiac disorders, from aggravation of pre-existing heart and lung conditions to premature mortality, and reduced life expectancy. Moreover, a 2017 joint American Thoracic Society/ European Respiratory Society (ATS/ERS) statement expanded the evidence to include emerging effects of AAP on the central nervous system (CNS), reproduction and development and certain metabolic outcomes, as well as non-respiratory cancer including leukemia in children (Thurston et al., 2017).

In spite of the successful efforts to reduce air pollution in industrially developed countries, mortality and morbidity attributable to air pollution have not decreased on a global level. The 2015 Global Burden of Disease study (Cohen et al., 2017) revealed that exposure to particulate matter with a diameter less than $2\cdot5 \,\mu\text{m}$ (PM_{2:5}) caused $4\cdot2$ million deaths (95% uncertainty interval [UI] $3\cdot7$ to $4\cdot8$ million), representing 7·6% of total global deaths, and 103·1 million disabilityadjusted life-years (DALYs) (95% UI 90·8 to 115.1 million) in 2015. Rising levels of pollution and increasing numbers of deaths from NCDs in low- and middle-income countries (LMICs) are responsible for this. The Lancet commission on air pollution(Landrigan et al., 2017) recently stressed that nearly 92% of pollution-related deaths occurred in LMICs. However, this figure is almost entirely based on data obtained from LMICs *outside* Africa, and the magnitude of the risk attributable to AAP has not been well documented for the African continent.

The United Nations department of economic and social affairs recently provided important data on demographic trends and future prospects to help policies intending to achieve the new sustainable development goals.("World Population Prospects: The, 2017 Revision | Multimedia Library - United Nations Department of Economic and Social Affairs," n.d.) Overall, between 2017 and 2050, 26 African countries are projected to double their current population size, with six countries even undergoing a five-fold population growth by 2100. Half of Africa's population is expected to live in urban areas by 2035, and SSA will host five of the world's 41 megacities by 2030 (Lagos, Kinshasa, Johannesburg, Dar es Salaam, and Luanda). However, the urban population boom in SSA is occurring in a context of slow structural transformation, violence and poverty(http://issafrica.org/ ISSAfrica.org, 2016). Although rural areas are not necessarily free from air pollution, SSA's rapid urbanization is and will undoubtedly be associated with high AAP.

Our objective was to systematically review the literature on air pollution levels and, especially, epidemiological studies focusing on the association between AAP and human health in SSA. We combined and updated published data on AAP from primary studies, reviews(Petkova et al., 2013) and the 2016 WHO global urban AAP database ("WHO | WHO Global Urban Ambient Air Pollution Database (update 2016)," n.d.) to assess the situation in SSA and compare it with other regions. We summarized the published epidemiological evidence on the association between AAP exposure and health outcomes in SSA. We then synthesized and highlighted risk factors of exposure to AAP and classified them into modifiable and non-modifiable factors, which could be used when planning mitigation actions and/or calls to action.

2. Methods

2.1. Search strategy and selection criteria

We searched the following electronic databases: PubMed and Medline-OVID, EMBASE, and Scopus (see Appendix 1 for the full search strategy). In general, databases were searched with a combination of terms and derived keywords, including variations of the following terms: "air pollution", "outdoor air pollution", "health", "health effects", "respiratory disorders", "respiratory health", "respiratory



Fig. 1. PRISMA flow diagram for selection of studies. SSA: Sub-Saharan Africa.

symptoms"," cardiovascular" and "Sub-Saharan Africa". In the MED-LINE search, the names of each Sub-Saharan African country("United Nations - Sub-Saharan Africa," n.d.) as well as regions (e.g. East-Africa) were included to increase search sensitivity. Alternative sources were also searched for eligible studies: the WHO database, national agencies, African Index Medicus, abstracts at scientific conferences (e.g. Pan African Thoracic Society), and other relevant grey literature. No language restriction was applied; the timeframe of the search included all records from the electronic database inception to November 15, 2017.

Studies exploring levels of pollutants were included if the data came from one of the SSA countries (see box1b) and if results were quantifiable.

The criteria for selecting studies regarding health outcomes included:

- Study population: children, adults or elderly residing in SSA;
- Exposure of interest: any ambient air pollutant, whether measured directly or inferred (e.g., by proximity to roadways or mining dumps), with a focus on the pollutants considered as "criteria air pollutants" by the US Environment Protection Agency (CO, NO₂, SO₂, O₃, Pb, PM₁₀, and PM₂₅); (US EPA, n.d.)
- Study design: epidemiologic studies of any design, including crosssectional, case-control, case-crossover, and cohort studies;
- Outcomes: any health findings.

We excluded studies that only assessed tobacco smoking-related pollution, household air pollution, or occupational exposures (e.g. among mineworkers). We also excluded studies focusing on Saharan dust if they did not include measurements or effects in SSA countries. Biomonitoring studies assessing exposures to metallic agents (e.g. blood lead) were also excluded if no aerial measurements of these pollutants had been made. We also excluded experimental or purely methodological studies.

Two reviewers (PK and TN) evaluated the eligibility of studies. In cases of discrepancy a third reviewer (LB) provided arbitration. For the exposure studies, two reviewers (LB and AB) extracted data and this was checked by two others (PK and JM). For the studies reporting health effects, one reviewer (PK) extracted data and three others (LB, JM, and AB) checked the extracted data. Regarding exposure, we extracted the following information: reference, country/city/town, period, context, design (analytic method), type and level of pollutant and other relevant considerations (comparison with international standards, major sources of pollution etc.). For each health study meeting the selection criteria, we extracted the following data: first author's name, publication year, country and city where the study was performed, study design, year(s) of data collection, type of controls (population-based, village-based), sample size, type of pollutant exposure (PMx, NOx, proximity to roadway etc.), method to quantify exposure (quantitative methods or questionnaires), type of outcomes (cardiorespiratory, others), outcome measurement (spirometry, questionnaire), confounding variables adjusted for, and key conclusion of the authors.

We assessed the quality of the included studies using the Newcastle-Ottawa Scale (NOS)("Ottawa Hospital Research Institute," n.d.), as recommended by the Cochrane Non-Randomized Studies Methods Working Group.("Cochrane Handbook for Systematic Reviews of Interventions," n.d.) The NOS uses an eight-item rating system to evaluate the method of selection of participants, the exposure/outcome assessment, and comparability among study groups. It has specific formats for cohort and case-control studies. We used the modified form for cross-sectional studies and the case-control form for case-crossover studies. Two reviewers (LB and JM) independently performed the quality assessment and a third reviewer (PK) resolved disagreements.

2.2. Data analysis

We performed a narrative synthesis and produced summary tables of findings of included papers because methodological heterogeneity in exposure and outcome assessment precluded a meta-analysis. This systematic review was reported according to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guideline (Liberati et al., 2009).

3. Results

3.1. Search results

As shown in a PRISMA flow diagram (Fig. 1), the initial searches provided 1402 non-duplicate records, of which 139 full texts were assessed for eligibility. After exclusion of 79 records that did not meet the pre-established inclusion criteria (list of excluded studies available on request), 60 studies were retained for qualitative synthesis. Of these 60 studies, 37 dealt only with levels of exposure and 23 combined exposure and health outcomes; among the latter, seven studies reported levels of exposure estimated by questionnaires, and 16 studies reported levels measured by quantitative methods.

3.2. Risk of bias in studies assessing health outcomes

Fig. 2 summarizes our scoring of risk of bias and our total NOS score for each included study. The overall methodological quality assessment

of the studies was moderate, with a mean (\pm SD) NOS score of 7·4 \pm 1·3 (10 being the maximum achievable score). Although the selection of study groups and determination of exposure/outcomes were clearly defined in more than 70% of the included health studies, only half of them clearly compared the exposed group(s) to a well-defined control group (mostly lacking in cross-sectional studies).

3.3. Exposure measurement

eTable 1 summarizes the findings regarding exposure measurements and Fig. 3 represents SSA countries where "criteria air pollutants" (US EPA, n.d.) were measured. After updating different databases, (Petkova et al., 2013: "WHO | WHO Global Urban Ambient Air Pollution Database (update 2016)," n.d.) data were available from only 18 countries (out of potentially 47 SSA countries). The distribution was very uneven across political and linguistic regions, with six countries in Western Africa, six in Eastern Africa, four in Southern Africa and only one in Central Africa. Moreover, measurements were mostly performed in the country capitals or largest cities, and data were collected for brief periods only. Consequently, mean annual data were scarce, with only levels of 24 h (or less) obtained during brief campaigns being commonly reported. Only, SA and Senegal have a continuous and real-time air quality monitoring network coverage(project, n.d.) with data available publicly and freely on-line from www.saaqis.org.za and www.air-dakar.org, respectively. Analytical methods varied depending on study context. For measurements of PM concentrations, the gravimetric method was widely used, followed by optical methods. Studies investigated various conditions such as daily variation, seasonal variations, area (urban vs rural), type of activities, wind direction, atmosphere stability, roads proximity, spatial distribution, socio-economic status and population density.

In the 23 studies assessing health effects (eTable 1), only seven countries were represented, with SA covering more than half of these studies. Pollutants measured varied across studies: from PM, NOx, SO₂, CO, PAHs, or benzene(Ana et al., 2014; Anderson et al., 2018; Ayi-Fanou et al., 2011; Hamatui and Beynon, 2017; Lawin et al., 2016; Makamure et al., 2017, 2016; Mentz et al., 2018; Naidoo et al., 2013; Nkosi et al., 2017) to "traffic related air pollution" (TRAP, evaluated as proximity to road or number of trucks passing per day or per hour) (Shirinde et al., 2015, 2014; Venn et al., 2005), or MEE (meteorologically estimated exposure) in the context of proximity to an oil refinery(White et al., 2009), a cement plant(Nkhama et al., 2017) or mine dumps (Nkosi et al., 2017, 2016; 2015a).PM_{2:5}, PM₁₀, NOx, and SO₂ were mostly assessed in SA. Quantitative personal exposure was generally absent and only sporadically assessed (e.g. CO(Lawin et al., 2016) and PAHs(Ayi-Fanou et al., 2011) in Benin; lead(Rahama et al., 2011) in Sudan). Two studies(Anderson et al., 2018; Goodrich et al., 2016) applied land use regression to model individual exposure. Most studies included one or more control groups. For instance, urban/industrialized communities were compared to rural/non-industrialized communities, or living close to roads or mine dumps was compared to living far away from these pollution sources.

3.4. Exposure comparability

The included studies reported a high diversity of pollutant sources, from natural meteorological phenomena, such as Harmattan dust, to anthropogenic pollution, such as street food preparation. To inform policy and to plan for mitigation actions, we classified AAP sources into modifiable and non-modifiable risk factors (eTable 2). To ensure comparability between studies, we referred to the mean annual PM_{2:5} and PM₁₀ data from the WHO, 2015 database ("WHO | WHO Global Urban Ambient Air Pollution Database (update 2016)," n.d.). People from SSA appeared to be more exposed to hazardous levels of PM_{2:5} and PM₁₀ than their counterparts from high-income countries (Figs. 4 and 5). Of 39 cities with available mean annual data, five to eight were around the



Fig. 2. Quality of health studies using the New Castle Ottawa (NOS) method. Red, yellow and green circles indicate high, moderate and low to very low risk of bias, respectively. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

WHO limit for $PM_{2:5}$ and PM_{10} , respectively (eFig. 1a and 1b), while the others had 10–20-fold higher levels.

Characteristics of studies assessing health outcomes.

eTable 3 summarizes the main characteristics of the 23 included studies and highlights the heterogeneity in terms of study participants, exposure assessments and investigated outcomes. Overall, the articles covered discrete study locations (rather than a countrywide coverage) in seven countries across SSA, with more than half (13/23) the studies having been conducted in SA. The majority of studies (15/23) were cross-sectional surveys. Nine studies focused on children (mostly schoolchildren between 9 and 14 years old), two on older persons, and two on mother-child pairs. When exposure was not quantitatively measured, authors used questionnaires with various items (self-reported or via caregiver) to assess outdoor and, occasionally, indoor environment. Consequently, no studies provided effect estimates for health effects associated with specific pollutants.

With regard to health outcomes (Table 1 and eFig. 3), respiratory diseases (RD) were most frequently investigated, mainly using symptoms obtained by questionnaires (nasal symptoms, wheeze, cough, phlegm, rhinitis, asthma, and bronchial hyperactivity) or pulmonary function tests.

RD, atopy and gene effects were mostly assessed in children. Three studies(Makamure et al., 2017, 2016; Reddy et al., 2012) investigated gene-pollutants interaction on respiratory parameters (such as intraday variation of FEV1) following exposition to PMx, NOx and SO₂. Co-morbidities associating RD (such as asthma, and emphysema) and cardiovascular diseases (CVD) (such as hypertension and myocardial

infarction) were especially investigated among older persons. One study from Cape Town(Wichmann and Voyi, 2012) reported on mortality resulting from RD, CVD and cerebrovascular diseases (CBD).

Other conditions such as skin diseases, atopy, gastro-intestinal diseases, malaria, and cancer were also investigated.(Ana et al., 2009). Two studies evaluated the effect of exposure to pollutant on the change of DNA methylation in general throughout the epigenome, *in utero* (Goodrich et al., 2016) and in adults (Ayi-Fanou et al., 2011; Goodrich et al., 2016) respectively.

Most studies were community-based, and two studies(Ana et al., 2009; Wichmann and Voyi, 2012) used medical or other health records. Known confounders such as age, gender, race, biomass smoke exposure, tobacco smoke exposure, and household income were adjusted for in the majority of studies.

3.5. Ambient air pollutants and respiratory health

3.5.1. In children

Four studies evaluated the effect of AAP on wheeze among schoolchildren(Naidoo et al., 2013; Nkosi et al., 2017; Shirinde et al., 2014; Venn et al., 2005). In Ekurhuleni (SA), heavy truck traffic significantly increased the risk of ever, current, and especially severe wheeze [OR 2·22 (95%CI 1·28-3·77)](Shirinde et al., 2014). In Durban (SA), no significant association was found between the prevalence of wheeze and mean PM_{10} or SO₂ measured in primary schools(Naidoo et al., 2013). This is contrary to the findings for adults from Gauteng (SA), in whom living close to mine dumps was strongly associated with chronic



Fig. 3. Map of sub-Saharan Africa indicating countries with at least one report or study on ambient air pollution. Full symbols indicate that at least one "criteria air pollutant" was measured (e.g. PM_{10} , $PM_{2.5}$, CO, SO_2 , NO_2), open symbols indicate health outcomes were also assessed. The location of the symbols within a country does not correspond to the exact location of the study; most studies were done in the country capitals or major cities.

cough [OR 2.02 (95% CI 1.58-2.57)]. (Nkosi et al., 2015a).

The covariate-adjusted prevalences of persistent asthma and bronchial hyperreactivity (BHR)(Naidoo et al., 2013) were higher among children attending schools in an industrial area compared to a residential area, with the risk of BHR being associated with mean school levels of SO₂ [OR 2·14, (95%CI 0·98-4·66)]. A study from Ethiopia(Venn et al., 2005) showed that AAP did not significantly increase the prevalence of wheezing. In SA, children living around mine dumps were 1.38 times more likely to have wheeze than controls (Nkosi et al., 2015a) and children living close to a refinery were more likely to report asthma, waking up with wheeze and carry an inhaler when going to school (White et al., 2009). Counterintuitively, living close to mine dumps(Nkosi et al., 2015a) was associated with less asthma among adolescents [OR 0.29, (95%CI 0.23-0.35)]. These findings contradict results from SA, which showed that children in industrial communities were more likely to have diagnosed asthma (Naidoo et al., 2013), and that living close to mine dumps was associated with asthma, albeit in the elderly (Nkosi et al., 2015a). Naidoo et al., (2013) also reported that FEV1% did not differ significantly with different PM10 and SO2 levels in Durban (SA).

Rhinitis (ever and current) and rhino-conjunctivitis were significantly associated with both exposure to TRAP(Shirinde et al., 2015) and proximity to mine dumps(Nkosi et al., 2015a) among schoolchildren. Bronchitis, shortness of breath, phlegm, but not cough, were more likely to be present in children living in industrial areas(Naidoo et al., 2013), but the associations with mean PM_{10} and SO_2 were not statistically significant across schools. Atopy and allergy to cockroach, house-dust-mite and mould were more prevalent in children from industrialized areas compared to cat, dog and grass allergy, which was more prevalent in non-industrialized areas(Naidoo et al., 2013).

Modification of pollutant effects by genes in relation to RD has been explored among African schoolchildren in the KwaZulu-Natal province of SA. (Makamure et al., 2016; Reddy et al., 2012) (Makamure et al., 2017). TNF- α 308 G/A polymorphism (known to be pro-inflammatory in the pathogenesis of airways disorders) alone was not significantly associated with intraday variation of FEV1. However, carriers of TNF- α 308 An allele had increased deterioration of lung function following exposure to NO and SO₂. Similarly, glutathione-S-transferase M1 gene (GSTM1) and glutathione-S-transferase P1 gene (GSTTP1), both known to increase susceptibility to asthma, alone was not associated with FEV1 variation. However, interacting with pollutants, GSTP1 (AG/GG) genotype modified the effect of three days prior 24-hr average PM₁₀ and SO₂ and increased FEV1 variability. In addition, CD14 CT/TT genotype has also showed a reverse effect of respiratory phenotype (symptoms



Fig. 4. Mean annual $PM_{2.5}$ and PM_{10} (µg/m³) across global regions as defined by WHO, between 2011 and 2016. Box plots represent medians with 25th-75th percentiles and 10th-90th percentiles for whiskers; dots are outliers. Red and blue vertical dotted lines at 10 and 20 µg/m³ correspond to WHO annual standards for annual PM_{2.5} and PM₁₀, of respectively Afr: Africa; Amr: America; Emr: Eastern Mediterranean; Eur: Europe; Sear: South-East Asia; Wpr: Western Pacific; LMI: Low and middle-income; HI: high-income. Source: WHO, 2015 database: WHO_AAP_database_May2016_v3web. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

based) following exposure to NO₂ and NO.

3.6. In adults

In adults, cough (43%), shortness of breath (25%), and asthma (11.2%) were more prevalent in a region with high PM in Windhoek (Namibia), and high PM exposure led to an increased OR for episodes of phlegm and cough(Hamatui and Beynon, 2017). Counterintuitively, motorcyclists in Benin were not at significantly higher risk of cough, phlegm or cough and phlegm when compared to neighborhood controls (Lawin et al., 2016).In a community study in Ibadan (Nigeria)(Ana et al., 2014), a dose-response relationship was observed between PM_{10} and lung function impairment. In that study, PM₁₀ was five-fold higher than the WHO standard $(20 \,\mu g/m^3)$ in a quiet academic area, six-fold higher in a high traffic area, seven-fold higher in an industrial area and eight-fold higher in a commercial area, and FEV1 and PM₁₀ were negatively correlated across the study locations (r = -0.37, p < 0.05). This contradicts findings in Benin, where no difference in FEV1 was observed between motorcyclists and controls (Lawin et al., 2016). In this study, neitherrespiratory symptoms nor lung function were significantly associated with 8-h average CO levels. A survey in the Niger delta (Nigeria)(Ana et al., 2009) showed a higher prevalence of respiratory diseases (based on five-year medical records) in an industrial community compared to a residential community.

A study comparing DNA adducts in urban and villages/suburbs in Benin (Ayi-Fanou et al., 2011) in association with exposure to PAHs found that taxi-motorbike drivers, roadside residents, street vendors, taxi-motor-bike drivers and gasoline sellers had significantly higher levels of DNA-adducts (suggesting an excess risk for chronic diseases) than suburban and village inhabitants.

3.6.1. In older adults

In SA, living close to mine dumps for older adults (\geq 55y)(Nkosi et al., 2015b) was significantly associated with bronchitis, asthma, pneumonia and emphysema among older persons and even doubled the odds of wheeze, and cough. In a cross-sectional study of 11 communities in SA(Nkosi et al., 2016), cardiorespiratory comorbidities were significantly more prevalent among 1499 older persons living near mine dumps than among 898 controls living further away (asthma + hypertension [OR 1.67 (1.22 - 2.28], emphysema + arrhythmia [OR 1·38 (1·07-1·77)], emphysema + myocardial infarction [OR 2·01(1·73-2·54)] and hypertension + pneumonia [OR 1·34 (1.05 - 1.93)]). In a case-crossover study in Cape Town (SA), the risk of dying from RD, CBD and CVD was associated with 24-h average levels of AAP with even higher estimates than those reported in developed countries (IQR increase in yearly PM_{10} [12 µg/m³] and NO_2 [12 µg/m³] increased CBD mortality by 4% (0.4-8%) and 8% (3-13%), respectively. IQR increase in yearly NO₂ $[12 \mu g/m^3]$ and SO₂ $[8 \mu g/m^3]$ increased CVD mortality by 3% (0·3–7%) and 3% (0·1–5%), respectively) (Wichmann and Voyi, 2012). However, the association was only seen during the warm (summer) period. The larger number of associations of AAP and poor health among the elderly is disheartening but not unexpected, given the increased propensity for the development of health problems in old age.



Fig. 5. Mean annual $PM_{2:5}$ and PM_{10} (µg/m³) in sub-Saharan African countries, between 2011 and 2016. Box plots represent median with 25th-75th percentiles and 10th-90th percentiles for whiskers; dots are outliers. Red and blue vertical dotted lines at 10 and 20 µg/m³ correspond to WHO annual standards for annual PM_{2:5} and PM₁₀, of respectively Source: WHO, 2015 database: WHO_AAP_database_May2016_v3web. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

4. Discussion

To our knowledge, this is the first systematic review of published studies on ambient air pollution and its relation to health for the African continent south of the Sahara. We identified 53 studies in which the levels of outdoor exposure were quantified, and seven that estimated exposure via questionnaires. Twenty-three of these studies also assessed health outcomes. Based on the latter studies, we summarized the current evidence linking AAP to adverse health effects in people living in SSA. It is fair to conclude that this evidence base is very limited. No systematic AAP monitoring takes place in SSA, except for SA and Dakar in Senegal. Moreover, the limited exposure data that are available, have been obtained by independent research teams during brief campaigns covering small geographic areas. Consequently, we do not have reliable primary data on AAP for the vast majority of the people living in SSA. Nevertheless, the existing information indicates that the levels of exposure to pollutants exceed WHO standards and levels currently found in industrialized regions from high-income countries. However, as far as can be judged from the limited data available, African cities do not appear to have reached the extremely high levels of pollution found in some Chinese or Indian cities.("WHO Air pollution levels rising in many of the world's poorest cities," n.d.) With so many gaps in our knowledge of the prevailing AAP in SAA, it is obvious that we know very little about the true impact of AAP on the health of the African populations, since epidemiological studies on this subject have hardly been done in SAA, except for SA. If one assumes that poverty increases the harm caused by AAP (and vice versa), we may posit that air pollution must be especially damaging for the poorest

fraction of the world's population, i.e. the people living in Sub-Saharan Africa.

Our systematic review showed that the methodologies for assessing outcomes in relation to exposure in SAA were highly diverse and, generally, of a poorer standard than those conducted elsewhere (Kethireddy et al., 2014; Kumar et al., 2016). Thus, exposure was often assessed via questionnaires or estimated via aggregated data, and rarely by representative individual measurements or GIS-based models. No studies, except from SA, were based on reliable morbidity or mortality statistics at regional or national level. The majority of studies were cross-sectional surveys, thus precluding an assessment of temporality, let alone causality. In addition, health outcomes were mostly assessed by questionnaires. Consequently, even though associations were found between exposure to pollution and health outcomes, the current overall evidence cannot be considered sufficient to estimate the true impact of AAP on the health of African populations. For example, among children, few studies demonstrated increased risks of respiratory symptoms associated with measured levels of pollutants, although several studies did show associations between symptoms and other indices of AAP, such as TRAP. This may mean that, even when air pollution was measured quantitatively, these measurements failed to capture the exposure correctly. One explanation could be that the measurements were not performed at the appropriate location (e.g. at school and not at home) or, alternatively, at the right time (during the study instead of before the study) or possibly because these measurements did not measure the right pollutants. In SSA, the limited number of studies mostly reported on respiratory symptoms such as wheeze, and cough-phlegm. Associations between air pollution and conditions such as acute respiratory

Table 1 Summary of health outcomes findings.						
Health Outcome	Age Group	Study ID	Location and setting	Study design	Exposure	Summary of main conclusions [estimates of associations (95% confidence intervals)]
WHEEZE	CHILDREN	Naidoo et al., 2013(Naidoo et al., 2013)	South Africa, Durban, urban/industrial vs suburban/non-industrial	Cross-sectional	PM ₁₀ , SO ₂	Children from industrial communities more likely to report wheeze with shortness of breath during past 12 months [OR 1·12 (1·01 - 1·3·4)]
		White et al., 2009(White et al., 2009)	South Africa, Cape Town, proximity to oil refinery	Cross-sectional	MEE $< 4 \text{ km}$ vs $> 4 \text{ km}$	MEE second with recent waking with wheezing [OR 1:33 (1:06 -166)] and frequent wheezing at rest (OR 1:27 (1:05 -1.54)]; no association for distance to refinery [OR 0:98 (0:74 -1.24); 0:93
		Venn et al., 2005(Venn et al., 2005)	Ethiopia, Jimma: distance to nearest road	Cross-sectional	TRAP < 150 m vs 150 m	(071 – 1.21)]. Children living within 150 m from the road more likely to report wheeze [OR 1.27 (0-93 – 1.73]
		Nkosi et al., 2015b (Vusumuzi Nkosi, Wichmann, and Vovi 2015a)	South Africa, Gauteng/North west, urban communities, proximity to mine dumps	Cross-sectional	1–2 km vs 5 km	Adolescents near mine dumps more likely to have current wheeze [OR 1:38 (1:10-1:71)]; higher risk if close to road with truck traffic [OR 1:33 (1:28-1:471)]
		Shirinde et al., 2014(Shirinde et al., 2014)	South Africa, Ekurhuleni Metropolitan, urban vs township	Cross-sectional	TRAP [truck traffic]	Truck traffic near home for almost the whole day during weekdays increased the likelihood of wheeze ever [OR 1:32 (1.01 – 1.73)], current wheeze [1.61 (1.15 – 2.24)] and current evere wheeze [OR
	ADULTS AND ELDERLY	Venn et al., 2005(Venn et al., 2005)	Ethiopia, Jimma: distance to nearest road	Cross-sectional	TRAP < 150 m vs	2.22 (1.28–377)]. Adults living within 150 m from the road more likely to report wheeze [1.14 (0.97 – 1.33)]
		Nkosi et al., 2015a (Vusumuzi Nkosi, Wichmann and Vovi 2015h)	South Africa, Gauteng/North west, urban communities, proximity to mine dumps	Cross-sectional	1–2 km vs 5 km	Living close to mine dumps associated with wheeze [OR 2.01 (1.73-2.54)].
	TIV	Venn et al., 2005(Venn et al., 2005)	Ethiopia, Jimma: distance to nearest road	Cross-sectional	TRAP < 150 m vs	Living within 150 m from the road more likely to report wheeze [OR $1.17 (1.01 - 1.36]$ (adjusted OR per 30 m increasing proximity).
соиен	CHILDREN ADULTS AND ELDERLY	Naidoo et al., 2013(Naidoo et al., 2013) Lawin et al., 2016(Lawin et al., 2016) Nkosi et al., 2015a (Vusumuzi Nkosi,	South Africa, Durban, urban/industrial vs suburban/non-industrial Benin, Cotonou, urban motorcyclist vs neighborhood controls (sex/aged matched) South Africa, Gauteng/North west, urban communities, proxinity to mine dumps	Cross-sectional Cross-sectional Cross-sectional	PM ₁₀ , SO ₂ CO 1–2 km vs 5 km	No differences in risk of cough when adjusting for school mean PM_{10} and SO_{2a} . [OR 1-03 (0-41-2:58); 1-02 (0-47-2:19)]. Motoryclists at non-significantly higher risk of cough [OR 1-42 (0-17 - 409)] Living close to mine dumps associated with chronic cough [OR 2-02 (1:58-2:57)].
PHLEGM	CHILDREN	Wichmann, and Voyi, 2015b) Naidoo et al., 2013(Naidoo et al., 2013) Lawin et al., 2016(Lawin	South Africa, Durban, urban/industrial vs suburban/non-industrial Benin, Cotonou, urban motorcyclist vs	Cross-sectional Cross-sectional	PM ₁₀ , SO ₂ CO	Children from industrial communities more likely to have phlegm during past 12 months [OR 1:55 (0.76–3:15)] Motorcyclists at non-significantly higher risk of phlegm [OR 2:96
COUGH + PHLEGM	STJUA ADULTS	et al., 2010) Lawin et al., 2016(Lawin et al., 2016) (Hamatui and Beynon, 2017)	neignoornood controis (sex/aged matched) Benin, Cotonou, urban motorcyclist vs neighborhood controls (sex/aged matched) Namibia, urban/suburbs/rural	Cross-sectional Cross-sectional	CO PM	(U-35 - 0:40.)] Motorcyclists at non-significantly higher risk of cough + phlegm [OR 1:57 (051 - 484)] Higher risk of episodes of cough and phlegm with higher PM
BREATHLESS/DYSPNOEA BRONCHITIS	CHILDREN	Naidoo et al., 2013(Naidoo et al., 2013) Naidoo et al., 2013(Naidoo	South Africa, Durban, urban/industrial vs suburban/non-industrial South Africa, Durban, urban/industrial vs	Cross-sectional Cross-sectional	PM ₁₀ , SO ₂ PM ₁₀ , SO ₂	exposure category LOK 25 (0.8-90)]. Children from industrial communities more likely to have breathlessness during past 12 months [OR 1-12 (1-01-1-24)] Children from industrial communities more likely to have chronic
	ELDERLY	et al., 2013) Nkosi et al., 2015a (Vusumuzi Nkosi,	suburban/non-industrial South Africa, Gauteng/North west, urban communities, proximity to mine dumps	Cross-sectional	1–2 km vs 5 km	bronchitis [OR 3-53 (1:1355)], risk attenuated after adjusting for school mean PM ₁₀ and SO ₂ [OR 1:02 (0:21 – 5:01); 1:34 (0:34 – 5:22)] Living close to mine dumps associated with chronic bronchitis [OR 1:74 (1:25-2:39)]
AIRWAY HYPER-RESPONSIVENESS	CHILDREN	Wichmann, and Voyi, 2015b) Naidoo et al., 2013(Naidoo et al., 2013)	South Africa, Durban, urban/industrial vs suburban/non-industrial	Cross-sectional	PM10, SO2	Children from industrial communities more likely to have AHR [OR $2\cdot49$ (1:30–9:55)], risk attenuated after adjusting for school mean PM ₁₀ and SO ₂ [OR 1.08 (0:44 – 2:66); 2:14 (0:98–4:66)]

(continued on next page)

Health Outcome	Age Group	Study ID	Location and setting	Study design	Exposure	Summary of main conclusions [estimates of associations (95% confidence intervals)]
ASTHMA	CHILDREN	White et al., 2009(White et al., 2009) Nkosi et al., 2015b (Vusumuzi Nkosi, Wichmann, and Voyi, 2015a)	South Africa, Cape Town, proximity to oil refinery South Africa, Gauteng/North west, urban communities, proximity to mine dumps	Cross-sectional Cross-sectional	MEE < 4 km $vs > 4 km$ $1-2 km vs$ $5 km$	MME associated with having to take an inhaler to school [OR 1:22 (1:06 - 1:40)], no association for distance to oil refinery. Proximity to mine dumps showed a protective association with asthma [OR 0:29 [0:23–0:35)].
	BLDERLY	Naidoo et al., 2013(Naidoo et al., 2013) Nkosi et al., 2015a (Vussimuzi Nkosi,	South Africa, Durban, urban/industrial vs suburban/non-industrial South Africa Gauteng/North west, urban communities, proximity to mine dumps	Cross-sectional Cross-sectional	PM ₁₀ , SO ₂ 1–2 km vs 5 km	Children from industrial communities more likely to have doctor- diagnosed asthma [OR 1:33 (not shown)] and symptoms based -persistent asthma (1:14 (not shown) Living close to mine dumps associated with asthma [OR 1:57 (1:20-2:05)].
RHINTIS/RHINOCONJUNTIVITIS	CHILDREN	Nuclearly, 2014) Shirinde et al., 2014(Shirinde et al., 2014) Nkosi et al., 2015b	South Africa, Ekurhuleni Metropolitan, urban vs township South Africa, Gauteng/North west, urban	Cross-sectional Cross-sectional	TRAP [truck traffic] 1–2 km vs	Truck traffic near home for almost the whole day during weekdays increased the likelihood of rhinitis ever [OR 1-46 (1.16 - 1.84)], current rhinitis [1-60 (1:24-2.02)] and current rhinoconjunctivitis [OR 1-42 (1:09-1:84)]. Adolescents near mine dumps more likely to have
EMPHYSEMA	ELDERLY	(Wusumuzi Nkosi, Wichmann, and Voyi, 2015a) Nkosi et al., 2015a (Vusumuzi Nkosi, Wichmann, and Voyi, 2015b)	communities, proximity to mine dumps South Africa, Gauteng/North west, urban communities, proximity to mine dumps	Cross-sectional	5 km 1–2 km vs 5 km	rhinoconjunctivitis [OR 1:54 (1:29 - 1:82)]; higher risk if close to road with truck traffic [OR 1:32 (1:07 - 1:75)] Living close to mine dumps associated with emphysema [OR 1:75 (1:11-2:77)].
PNEUMONIA Spirometry	ELDERLY CHILDREN	Nkosi et al., 2015a (Vusumuzi Nkosi, Wichmann, and Voyi, 2015b) Naidoo et al., 2013(Naidoo	South Africa, Gauteng/North west, urban communities, proximity to mine dumps South Africa, Durban, urban/industrial vs	Cross-sectional Cross-sectional	1–2 km vs 5 km PM ₁₀ , SO ₂	Living close to mine dumps associated with pneumonia [OR 1:38 (1:07–1:77)]. No significant difference in FEV1%, data not shown
	ADULTS	et al., 2016 Lawin et al., 2016(Lawin et al., 2016)	stour dary not annuastrat Benin, Cotonou, urban motorcyclist vs neighborhood controls (sex/aged matched)	Cross-sectional	CO	No differences between motorcyclists and controls for FEV1 [Mean difference (MD) 0.12L (-0.16 - 0.22)], FVC [MD 0.11L [-0.14 - 0.37)], FEV1/FVC [MD 0.78% (-0.23 - 1.17)] and PEF [MD 0.45 L/s (-0.26 - 0.81)]
COMORBIDITY [CARDIO-RESPIRATORY]	ELDERLY	Nkosi et al., 2016(V. Nkosi, Wichmann, and Voyi, 2016)	South Africa, Gauteng/North west, urban communities, proximity to mine dumps	Cross-sectional	1–2 km vs 5 km	Living close to mine dumps associated with: asthma + hypertension [OR 167 (1.22 – 228], asthma + pneumonia [OR 1.86 (1.14 – 304)], emphysema + arrhythmia [OR 1.38 (1.07 – 1.77)], emphysema + myocardial infarction IOR – (1.73 – 254)], emphysema + pneumonia [OR 3.36 (1.41 – 7.98)], hypertension + myocardial infarction [OR 1.60 (1.04 – 2.44)] and hypertension + nneumonia [OR 1.34 (1.05 – 1.931).
MORTALITY	ALL AGES	(Wichmann and Voyi, 2012)	South Africa, Cape Town	Case- Crossover Study	PM ₁₀ , SO ₂ , NO ₂	IQR increase in yearly PM_{10} [12 $\mu g/m^3$] and NO_2 [12 $\mu g/m^3$] increased CBD mortality by 4% (04–8%) and 8% (3–13%), respectively. Increase in yearly NO_2 [12 $\mu g/m^3$] and SO_2 [8 $\mu g/m^3$] increased IQR increase in yearly NO_2 [12 $\mu g/m^3$] and SO_2 [8 $\mu g/m^3$] increased CVD mortality by 3% (03–7%) and 3% (01–5%), respectively.

Table 1 (continued)

TRAP: Traffic Related Air Pollution, MEE: meteorologically estimated exposure, AHR: airway hyperreactivity, CBD: cerebrovascular diseases, CVD: cardiovascular diseases, IQR: interquartile range.

illness, pneumonia in infants, COPD(Berend, 2016; Dadvand et al., 2014; Garshick, 2014; Peacock et al., 2011; Schikowski et al., 2014), CVD(Atkinson et al., 2013; Chen, 2010; Forbes et al., 2009; Ljungman and Mittleman, 2014; Maheswaran et al., 2014; Nuvolone et al., 2011; Tsai et al., 2012), mental health(Cho et al., 2014; Genc et al., 2012; Power et al., 2011; Tzivian et al., 2015; Zijlema et al., 2016), type 1 and 2 diabetes(Balti et al., 2014; Beyerlein et al., 2015; Eze et al., 2014; Frampton et al., 2012; Park and Wang, 2014; Rao et al., 2015; Thiering and Heinrich, 2015), and tuberculosis(Álvaro-Meca et al., 2016; Blount et al., 2017; Chen et al., 2016; Kim, 2014; Smith et al., 2016, 2014) have been extensively documented in other continents. However, robust epidemiological information on these conditions is lacking for most African countries, and, no primary data (even from cross-sectional surveys) exist for the vast majority of African countries, especially those in Central-Africa. Based on estimates from satellite observations and other assumptions, it was concluded that (Owili et al., 2017) under-five and maternal mortality at country level were significantly associated with the levels of four types of PM2.5 (biomass, anthropogenic, dust, and mixture). They showed positive dose-response relationships between under-five or maternal deaths and types of PM_{2.5} or chronic exposure to $PM_{2:5}$ above 30 µg/m³. In Central Africa, $PM_{2:5}$ originating from biomass burning was estimated to increase under-five and maternal mortality by 2% and 19%, respectively, and PM2.5 from dust increased maternal mortality by 10%. Moreover, Heft-Neal et al., 2018) recently found excess children mortality of 449,000 (95% CI: 194,000-709,000) in 30 African countries that was attributed to air pollution (satellite-based). This estimate is more than three times higher than existing estimates that associate children mortality to poor air quality in this region.

Some studies described associations between living in urban/industrialized areas and adverse health effects. Economic growth is important and rapidly increasing, and SSA is becoming increasingly industrialized, with cities steadily expanding and traffic intensity mounting(Kinney et al., 2011; Petkova et al., 2013; "World Urbanization Prospects - Population Division - United Nations," n.d.). More than in high-income countries, socio-economic status (SES) plays a disproportionate role in air pollution exposure and health outcomes in Africa(Boadi et al., 2005). Studies in Ghana(Dionisio et al., 2010a, 2010b) and SA(Worobiec et al., 2011) found associations between low SES and high exposure to AAP. Similarly, studies in Kenya(Egondi et al., 2016) and Namibia(Hamatui and Beynon, 2017) found a higher health hazard associated with AAP in urban slums and in highly populated urban suburbs, respectively. Indeed, most risk factors such as unpaved roads, biomass combustion, street food preparation, public transport (taxi-motorbike, bus), use of old cars and proximity to mine dumps are related to poverty(GBD, 2013 Risk Factors Collaborators et al., 2015; Gordon et al., 2014; Jary et al., 2016; Kinney et al., 2011; Petkova et al., 2013; Rodriguez-Villamizar et al., 2012). Lelieveld and colleagues (Lelieveld et al., 2015) recently estimated, using satellite estimations of air pollution and pollutant-mortality risk models, the numbers of premature deaths attributable to air pollution. They suggested that ambient PM_{2.5} from commercial and domestic energy generation, agriculture and traffic sources contribute the most to premature deaths worldwide. They calculated that premature mortality could be reduced by 4.54 million each year by mitigating both ambient and household air pollution, mainly through changes in commercial and domestic energy use, especially in Africa where local energy mostly relies on solid fuels. Without concrete and appropriate mitigation plans and policies, the authors expect a doubling of mortality from air pollution by 2050 considering the projected rates of increase in population and air pollution levels. Commenting on this finding, the accompanying editorial raised the question of which sources should be reduced in Africa (Jerrett, 2015). This question is in line with the conclusions of the present systematic review and invites leaders at the African union and their partners to prioritise a high-level meeting on air pollution for a common agenda to achieve a tangible change in the region.

Strengths of our review include a detailed search of not only several databases of scientific publications, but also of national and agency reports and other grey literature to identify eligible studies, and an evaluation of all included studies for risk of bias using a rigorous method to quantify biases per study and per items. The present work provides environmental health authorities, clinicians and researchers with a current summary of the burden of AAP and evidence, however limited, of its adverse health effects on people living in SSA. We listed modifiable and non-modifiable risk factors of high exposure to help policies towards appropriate mitigation actions in the SSA region. A limitation of this review is that we were not able of quantitatively assess the risk of publication due to the paucity of evidence. **Panel 2**

Current Challenges and Suggested Future Directions to Address the Burden of AAP in SSA.

Challenges Recommendations (research and policy/management) Based on existing (limited) evidence, 1-Some factors related to high pollution, evels of exposure in most urban and such as biomass burning, unpaved industrial cities in SSA appear to be roads, poorly regulated industrial and exceeding WHO standards commercial activities, are modifiable Economic and demographic growth raand can be reduced. tes are increasing in SSA countries. The urbanization process is accelerating, and cities are increasingly agglomerating. Most African countries, except for Sen-Implementation plans to ensure contiegal and South Africa, do not have nuity in pollutant measurement should an air pollution monitoring system be elaborated. This could enable the establishment of air pollution moniin place. In most studies, air pollution was measured through brief toring networks to ensure availability of collaborative projects led by overdata for future studies. "African-friendly devices", i.e. equipseas laboratories without follow-up. ment that is adapted to local conditions and challenges unique to sub-Saharan Africa (low-cost, long-life batteries), should be developed and favored. Publishing data from national agencies should enhance gathering information concerning health risks. Establishing fixed monitoring stations for ambient air pollution is a priority in many SSA countries Future studies should consider assessing Very few studies assessed the health impact of PMx, NOx, SO₂, CO and these cardinal pollutants in other parts O3 in SSA and most of them were of SSA countries to ensure standardization and comparability of findings. from South Africa (Kwazulu-Natal province). Ad Hoc Ouestionnaires were mostly u-Standardized questionnaires such as the sed to estimate exposures. IMPALA questionnaire adapted for Africa(Saleh et al., 2018) should be preferred to ensure repeatability. Future studies should focus on developing standardized questionnaires to assess levels of exposure while considering factors associated with ambient air pollutant in this region. Studies should consider using quantitative methods to assess exposure to air pollution to reduce ascertainment bias that could dilute the true association. Cross-sectional designs were mostly us-Future studies should have prospective longitudinal designs. Continuous or reed. peated exposure measurement models

Household air pollution contribution was weakly reported in health studies and long-term exposure. Future research should account for this confounder that could bias the effect estimate towards the null. HAP caused

should be preferred to account for sea-

between regions. This would also allow assessing dose-response relationships

and quantifying the burden due to short

sonality and other factors that vary

	by biomass smoke does not only affect people through indoor pollution, but also through outdoor pollution.
Pollutants were separately included in multivariate analysis model.	After modelling each pollutant, future studies should consider evaluating the effect of mutual adjustment of other air pollutants in multivariate models
Outcomes relied on questionnaires.	As for exposure, future studies should use quantitative determinations of ef- fects. Where doctor-diagnosed out- comes are available, future studies should consider such an approach to avoid misclassifications of the outcomes due to recall bias, self-report responses, interviewer variability etc. In addition, to facilitate epidemiolo- gical studies, reliable registration sys- tems and databases of morbidity and mortality need to be in place at the level of health centres, hospitals, regions and countries
Respiratory morbidity related to expo- sure to ambient air pollution was modestly documented and data on other conditions were absents	Future studies should consider ex- tending the research to perinatal mor- bidity, pneumonia in children, COPD, TB, exacerbations of asthma, metabolic diseases, effect on major infectious dis- eases in the region (HIV status, malaria

5. Conclusion

SSA is facing environmental challenges due, largely, to its fast urbanization and economic transformation. Further exacerbated by poverty, these two factors contribute to rising pollution levels. Deleterious effects of AAP undoubtedly contribute to the gradually observed epidemiological transition, but we do not know to what extent this is the case. Although there is a vast amount of evidence on pollution-induced health effects in developed regions and developing regions such as China and India, virtually no quantitative data is available for the African continent. The paucity of data and methodological heterogeneity in exposure and outcomes assessments led us to propose that more primary studies must be done to fill knowledge and methodological gaps and thus strengthen the current evidence to inform and support African policy makers. Such primary studies should preferably have prospective designs and rely on exposure measurements made with "Africa-friendly devices" (i.e. low cost and lasting battery autonomy), for both large monitoring programmes and detailed personalized exposure assessments. Finally, studies should also focus on a deeper understanding of the vulnerability of people with low SES and on the combined effects of Africa-specific pollutants.

Conflicts of interest

The authors declare that they have no competing interests.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors contributions

Conceptualization, PK, TN and BN; Data curation PK, TN, BN, LB, AB and JM; Formal analysis, PK, BN and TN; Methodology, PK, TN, BN, LB, AB; Project administration, PK and TN; Resources, PB, HS and NG; Software, PK and LB; Supervision, BN and TN; Visualization, PB, HS, NG; Writing—original draft, PK; Writing—review & editing, PK, TN, BN, LB, AB, JM, PB, HS and NG.

Ethics approval, consent to participate

Not applicable.

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List of abbreviations

AAP	ambient air pollution
SSA	Sub-Saharan Africa
PM	Particulate matter
WHO	World health organisation
LMICs	Low and middle-income countries
NCDs	Non-communicable diseases
ATS/ERS	American thoracic society/European respiratory society
CNS	Central nervous system
UI	Uncertainty interval
NOS	Newcastle-Ottawa scale
PRISMA	Preferred reporting items for systematic reviews and meta-
	analyses
TRAP	Traffic related air pollution
MEE	Meteorologically estimated exposure
RD	Respiratory diseases
CVD	Cardiovascular diseases
CBD	cerebrovascular diseases
SA	South Africa
BHR	bronchial hyperreactivity
SES	Socio-economic status
COPD	Chronic obstructive pulmonary disease

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2019.03.029.

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