Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Association of asthma control with household particulate matter less than 2.5 microns, carbon monoxide and second-hand-smoking among children with asthma in Lagos, Nigeria: protocol of an ongoing observational cross-sectional study

Peter Odion Ubuane

peterubuane@gmail.com

Lagos State University Teaching Hospital

Olufemi Ademokoya

Lagos State University Teaching Hospital

Babatunde I. Awokola

Medical Research Council Gambia at London School of Hygiene & Tropical Medicine

Olufemi Tunde Ojo

Lagos State University College of Medicine

Temitope Victoria Fapohunda-Ayanda

Lagos State University Teaching Hospital

Abass Adigun

Nigerian Airspace Agency

Oluwajimi Sodipo

Lagos State University Teaching Hospital

Olatunde Adegboyega Odusote

Lagos State University Teaching Hospital

Study protocol

Keywords: PM2.5, indoor air pollution, sub-Saharan Africa, urinary cotinine, CO, air quality monitoring, PM2.5 sensor, CO logger, FEV1, PEF

Posted Date: November 6th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9634309/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

TITLE PAGE

Association of asthma control with household particulate matter less than 2.5 microns, carbon monoxide and second-hand-smoking among children with asthma in Lagos, Nigeria: protocol of an ongoing observational cross-sectional study

AUTHORS/AFFILIATIONS

Dr Peter Odion Ubuane, MBChB, FWACPaed;[1,2] **Dr Olufemi Ademokoya,** MBBS, MWACPaed;[1] **Dr Babatunde I. Awokola**, FWACP, ATSF; [3] **Dr Olufemi Tunde Ojo**, FWACP;[4] **Dr Temitope Victoria Fapohunda-Ayanda,** MBBS, FWACP;[5] **Dr Abass Adigun**, BSc (Statistics), PhD;[6] **Dr Oluwajimi Sodipo**, MBBS, FWACP, FMCFM;[7] **Dr Olatunde Adegboyega Odusote,** FMCPaed[2]

Affiliation

[1]Department of Paediatrics, Lagos State University Teaching Hospital (LASUTH), Ikeja, Lagos, Nigeria

[2]Department of Paediatrics & Child Health, Lagos State University College of Medicine (LASUCOM), Ikeja, Lagos, Nigeria

[3]Medical Research Council Gambia at London School of Hygiene & Tropical Medicine Fajara, The Gambia

[4]Respiratory Medicine Unit, Department of Internal Medicine, Lagos State University College of Medicine (LASUCOM)

[5]Respiratory Medicine Unit, Department of Internal Medicine Lagos State University Teaching Hospital (LASUTH), Ikeja, Lagos, Nigeria, Ikeja

[6]Department of Statistics, Nigerian Airspace Agency, Jos, Plateau, Nigeria

[7]Department of Family Medicine, Lagos State University Teaching Hospital (LASUTH), Ikeja, Lagos, Nigeria.

Corresponding Author

Dr Peter Odion Ubuane, MBChB, FWACPaed

Dept of Paediatrics, Lagos State University Teaching Hospital (LASUTH)/Lagos State University College of Medicine (LASUCOM), Nigeria, Ikeja, Lagos, Nigeria; peterubuane@gmail.com

ABSTRACT

Background

Childhood asthma is increasing in developing countries, partly due to household air pollution (HAP). Asthma care aims for optimal symptoms control and minimal future risk of exacerbations, known as asthma control (AC). There is a high prevalence of suboptimal AC among Nigerian children and adolescents with asthma, with consequent increased risk of psychosocial, academic and financial burden and, sometimes, fatality. The adverse role of HAP on AC may be significant because of high levels of air pollutants like particulate matter less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$) and carbon monoxide (CO) in Nigerian homes, and prevalent exposures to second-hand smokes (SHS). Previous studies mostly evaluated the association between HAP and asthma with questionnaires, with no detectable association, possibly because of recall and social desirability biases. Thus, this study plans to determine the association between AC and objectively-quantified household $PM_{2.5}$ and *CO* and SHS (urinary cotinine) among children with asthma accessing routine outpatient care at the Asthma Clinics of the Paediatric Allergology, Dermatology & Respiratory Medicine Unit, and the Adult Respiratory Clinic of LASUTH.

Methods

Eligible children and adolescents (up to age 18 years) are being consecutively recruited into this observational cross-sectional study in which the following data are obtained: socio-demographics, medical history, AC, medication adherence, inhalation technique, parental asthma-related knowledge, lung function (peak flow, FEV_1, home personal PEF variability), quality of life, urine cotinine level, household $PM_{2.5}$ and CO. Hierarchical multivariable logistic regression will be used to quantify the association of AC with $PM_{2.5}$, CO and urinary cotinine while controlling for potential confounders.

46 **Discussion**

47 The findings from this study may be useful for education of families of children with asthma, advocacy with
48 policymakers on air pollution control, as well as the design or strengthening of educational interventions to
49 mitigate air pollution in households of children with asthma, for example counselling on cessation of smoking or
50 use of cleaner cooking fuels.

51 **Trial Registration**:

52 Research project was registered at OSF with the project DOI: https://doi.org/10.17605/OSF.IO/2JS7P.

53 **Keywords:**

54 $PM_{2.5}$, indoor air pollution, sub-Saharan Africa, urinary cotinine, CO, air quality monitoring, $PM_{2.5}$ sensor, CO
55 logger, FEV_1, PEF

BACKGROUND

Asthma, "*a heterogeneous disease, usually characterized by chronic airway inflammation…* [and] *history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation…*,"[1] is the commonest chronic respiratory disease of childhood and adulthood.[2] Its incidence and prevalence is increasing in developing countries like Nigeria, partly due to increasing air pollution from urbanisation-related activities, traffic-related pollution, unregulated cigarette-smoking, industrial emissions, and poor housing.[3–10] Across Africa, asthma prevalence ranges from 4·2% in Malawi to 15% in Nigeria and 24% in South Africa.[7] Childhood asthma exerts enormous psychosocial, academic, physical and financial burden on children, their parents/caregivers, the healthcare system and the society.[1, 2, 6, 7] Thus, in the absence of known cure, the goal of asthma management is the optimal control of asthma symptoms and exacerbation risks, to ensure normal or near-normal quality of life.[1, 11]

Asthma control (AC) is the degree to which treatment or care has minimised the symptomatic manifestations of asthma, including the frequency of acute exacerbations.[1, 12] AC is related to health-related quality of life; children with poor or suboptimal AC, and their caregivers (parents/caregivers), have poorer quality of life, increased exacerbations, morbidity and mortality, compared with those with better control.[1, 7, 13–16] Poorly controlled asthma may result in airway remodelling and impaired lung growth.[17, 18] Thus, the objective evaluation of AC is an integral aspect of routine asthma management,[1] underscored by the fact that patient's or parent's perception of AC is often discordant with physician's objective assessment.[1, 7] There is a very high burden of sub-optimal AC among children with asthma (CwA) in developing countries largely due to potentially modifiable risk factors like air pollution, poor access to controller medications, poor inhaler techniques, poor adherence to medications, poor knowledge on asthma and psychosocial distresses.[1, 7, 11, 19] In Nigeria, about 18-84% of CwA have sub-optimal AC.[13, 15, 16, 20, 21]

Household air pollution and asthma control

Air pollution refers to the presence of harmful particulate and gaseous substances in the air, ambient (outdoor) or house-hold (indoor).[22] House-hold air pollution (HAP) is defined "as indoor air pollution from domestic solid-fuel combustion for cooking and/or heating (wood, charcoal, kerosene, animal dung, crop residues, pellets, coke, and coal)."[23] HAP is worse in developing countries[24, 25] like Nigeria,[8, 26, 27] contributing significantly to mortality and disability-adjusted life year.[2] Children, compared to adults, are particularly vulnerable to the cellular and molecular harmful effects of these pollutants because children are still growing and developing physiologically; they have higher physiological respiratory rate and smaller body mass resulting in the inhalation of larger amounts per given body size and time; and they are more likely to be around their mothers who spend considerable time cooking.[23, 28–31] HAP is associated with impaired lung growth and function in children.[28] HAP is even more devastating on CwA, worsening existing chronic lung inflammation, thus hindering optimal control resulting in more frequent symptoms, exacerbation, hospitalisation, resort to ineffective alternative remedies, school absenteeism and risk of fatalities.[23, 32–34] Since children spend a large part of their lives at home, HAP may exert more negative impact on respiratory health than outdoor AP.[31, 35–37] Incompletely combusted 'unclean' fuels like kerosene, charcoal and others emit hazardous particulate matter (PM), including fine respirable particulate matters that are less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$) and noxious gases like carbon monoxide (CO), nitrous dioxide and sulphur dioxide.[23, 32, 38, 39] Another important source of both PM, CO and other harmful substances is cigarette smoking, notably second-hand smoking (SHS) in children.[10, 23] Environmental control, including of HAP and SHS, thus remains a cornerstone of asthma management for optimal AC, quality of life and lung development.[1, 8, 9, 15]

Household $PM_{2.5}$ level is associated with asthma incidence, poor AC, exacerbation, emergency visits and use of reliever medications in a dose-dependent fashion.[35, 40–44] In Nigeria, Kuti *et al*[15] reported that the use of unclean fuels was not associated with poorer asthma control, perhaps because HAP was assessed with just questionnaire rather than objective measurements of particulate or gaseous pollutants. Subsequently, few studies have studied the association of objectively-measured $PM_{2.5}$ with asthma incidence in the general population, but

not with AC among CwA.[44–46] We hypothesise that there would be an adverse association between asthma control and objectively-measured HAP and SHS among Nigerian children with asthma because of higher burden of HAP in Nigeria compared to high-income settings.[47] Although the effect of ambient air pollution (AAP) on asthma is known, less is known about the effect of HAP on asthma outcomes in sub-Saharan Africa and Nigeria.[34] Moreover, HAP may be more amenable to low-cost interventions compared to AAP which requires large scale interventions. For example, households may change the source of domestic cooking or energy fuels to address HAP but it might be more difficult to get rid of old cars in a city in order to control AAP.[25, 34, 48]

Carbon monoxide (*CO*) level is correlated with both $PM_{2.5}$ and SHS, and it also exerts adverse effect on AC.[49, 50] Carbon monoxide, like $PM_{2.5}$, is produced from incomplete combustion from vehicles, generators and cooking fuels, including kerosene- the commonest cooking fuel in Lagos.[47, 51] The ubiquitous use of gasoline generators in many homes in Nigeria due to long-standing electricity shortage is an important source of environmental *CO*. Apart from a possibly synergistic adverse direct effect on the airway along with other air pollutants like $PM_{2.5}$, CO binds strongly with haemoglobin to form carboxy-haemoglobin which impairs oxygen exchange which may impair AC, even at sub-fatal level. Eghomwanre and Oguntoke[46] recently reported that household *CO* was not associated with asthma incidence among children in Benin City, Nigeria, although its association with AC was not assessed.

SHS is the "involuntary or passive breathing of air contaminated with tobacco smoke by someone who is not smoking".[23] The smoke emanating from the smouldering cigarette contains over 4000 constituents including *CO*, *PM* and nicotine. Ultrafine particles (<0.1mm) from the smoke travel deep into the lungs inducing inflammatory and chemical injuries.[23] In the US, most SHS exposure occurs in the home, day-care setting and grandparents' home, and parents have poor perception of the degree of exposure and effect of SHS on CwA.[52] About 40% of Nigerian children and adolescents were exposed regularly to SHS, with 16% of them living with parents or family members who smoke indoors, associated with two-fold increased odds of chronic cough.[53] SHS is associated with onset of asthma, poorer asthma control, increased frequency of exacerbation and increased

healthcare utilisation and cost.[23, 54–56] In Nigeria, the association between SHS and asthma outcomes have mostly been assessed with questionnaires, risking misclassification due to recall bias or social desirability bias. The addition of biomarkers like urinary cotinine may limit the effects of these biases.[23] Thus there is need to quantitatively assay SHS to better investigate its association with asthma outcomes.[57]

RATIONALE

Summarily, there is scarcity of data on the association between objectively-measured HAP/SHS and asthma control among Nigerian children living with asthma;[33] those published elsewhere may not extrapolate here because of differences in geographical, socioeconomic and air quality profiles. Studies on the association between HAP and asthma has largely been questionnaire-based with asthma outcomes being correlated with subjectively assessed exposures to different types of household cooking fuels or exposure to parental smoking.[9, 13, 15] Such questionnaire-based assessments are less sensitive compared to more objective measurements using air-quality monitors or biomarkers such as urine cotinine- a by-product of nicotine.[23, 32, 58–60] For example, smokers may skip questions about their smoking habit or altogether give false information to avoid perceived stigmatisation. Hence, the use of objective measures of HAP and SHS, additional to questionnaires, may provide a better assessment of the level of true exposures and the contributory magnitude of these factors.[35, 42] Also, previous studies on the association between AC and HAP/SHS did not account for known determinants of AC such as adherence, inhaler techniques, or asthma knowledge which are potential confounders of the association between AC and HAP/SHS. We are exploring the association between AC and HAP/SHS while controlling for potential confounding by medication adherence, inhaler technique, socioeconomic factors and comorbid allergies. The data on the impact of exposure to HAP and second-hand smoking may be useful in counselling parents of CwA on smoking cessation or change of household fuel types;[15, 48, 61] advocating with government on stricter control of public smoking and access to affordable clean cooking fuels.[62]

METHODS

Study aims and objectives

Overall, the study aims to determine the association of AC (using GINA assessment of asthma control, asthma control test (ACT), forced expiratory volume in 1 second (FEV_1) and peak expiratory flow (PEF)]) with household levels of $PM_{2.5}$ and *CO* averaged over 3 days and urinary cotinine among CwA at the paediatric asthma clinic of the Lagos State University Teaching Hospital (LASUTH).

The primary specific objectives are to determine the:

1. prevalence of suboptimal asthma control [using GINA and Asthma Control Test (ACT)];
2. factors associated with suboptimal AC;
3. mean 24-hour $PM_{2.5}$ and *CO* (averaged from 3 days measurement) in households of children with asthma;
4. proportion of households with mean 24-hour $PM_{2.5}$ and *CO* above WHO's target limit of 15µg/m^3;[39]
5. independent association between asthma control and household $PM_{2.5}$, *CO* and urinary cotinine in CwA;
6. independent association between $PM_{2.5}$, *CO* and cotinine and lung function (PEF, FEV_1, PEF variability).

Secondary objectives are to determine the:

1. correlation between questionnaire-based assessment of exposure to household pollution (types of household cooking fuel) and objectively-measured $PM_{2.5}$ and *CO*;
2. correlation between questionnaire-assessed exposure to SHS and urinary cotinine levels;
3. agreement between physician-assessed clinic-based PEF and personal PEF.

Study design and setting

The study is an ongoing observational cross-sectional study being conducted primarily at the Paediatric Asthma Clinic of LASUTH (previously described[63]) which provides outpatient care for CwA aged up to 12 years. Children older than 12 years are being recruited from the adult Respiratory Outpatient Clinic and the General

Outpatient Clinic of the Family Medicine Department of LASUTH. While the Paediatric Asthma clinic holds once weekly, the adult Respiratory Clinic holds twice weekly and the General Outpatient Clinic of the Family Medicine Department holds daily. Generally, in all the three clinics, asthma diagnosis is made mainly based on its characteristic symptoms and signs, supplemented with lung function testing. Asthma management utilises the step-wise guidelines of the Global Initiative for Asthma (GINA), adapted to local context.[1, 63, 64]

Study participants and eligibility

All children with physician-diagnosed asthma aged up to 18 years who have been attending the clinics and have been on daily controller medications for at least two months are eligible. We exclude: 1) neonates; 2) those with suspected but unconfirmed asthma diagnosis (including those on trial of controller medications); 3) those with acute asthma exacerbation; 4) acute/chronic cough or suspected acute respiratory tract infections or tuberculosis (excluded only from lung function testing); 5) those living far away outside urban Lagos (because of the logistics of retrieving air pollution devices from homes).

Sample size and sampling technique

Using EpiTools® online sample size calculator (https://epitools.ausvet.com.au/oneproportion), we needed a minimum of about 120 participants to estimate the proportion of CwA with poor asthma control within 5% at a 95% confidence level, using an assumed prevalence of poor asthma control among Nigerian children of 13%,[16] a finite population of 200 asthmatic children attending clinic over a year and an additional 10% to account for missing data, data error or drop-out. To minimise skewed age distribution, the sample size is stratified thus: 40 children aged 5 year and below; 40 children aged 6-10 years; and 40 above 10 years. Parents/guardians of eligible participants are consecutively approached and recruited into the study at each of the participating outpatient clinics after detailed information and written consent.

Data collection procedures

A data collection form (Additional File 1) is used to obtain socio-demographic (name, age, sex, asthma diagnosis date), anthropometric (weight, height) and clinical data (medications types, adherence) from the parents/caregivers, the child and their medical records. We also obtain and record data on study's outcome measures (asthma control, lung function), exposure measures (household air pollution, second-hand smoke) and confounder measures (medication adherence, inhaler technique, parental knowledge) (see individual participant's flowchart, Figure 1).

Outcome Measures: Asthma Control

The assessment of asthma control comprises the assessment of *symptom control* and *future risk.*[1]

A. Asthma symptom control is assessed using *GINA asthma control question*,[1] and *Asthma Control Test*- child and adult versions (ACT) for 4-11 and ≥12 year-olds, respectively.[65, 66] The GINA questions consists of 4 questions on the frequency of day- and night-time asthma symptoms, use of reliever medication and activity limitation in the preceding four weeks. The result is categorized as *well-controlled, partly controlled* or *uncontrolled*. The ACT questionnaire is a 5-item validated tool for the evaluation of asthma control: activity limitation, breathlessness, night-time symptoms and the use of reliever medications and the child's overall rating of control in the last four weeks. Scores are generated from each item and summed to yield a total of 25. The scores are graded as *well-controlled* (≥ 20), *partly controlled* (13-19) and *poorly controlled* (≤ 12). Overall, we define *suboptimal asthma control* asthma control level that is other than well-controlled based on either or both of the GINA or ACT.

B. Risk of future exacerbations is assessed with a question on the *frequency of exacerbation in last one year* and with objective *lung function testing* (Additional File 1). The lung function test is done at the clinic by the research team (using an electronic peak flow meter that measures PEF and FEV_1) and at home by the child (PEF, PEF variability).

Lung function at the clinic:

We use an electronic peak flow meter (Asma-1™ Asthma Monitor, Child®, Vitalograph, UK; https://vitalograph.com/de/produkt/asma-1-asthma-monitor-child) to measure PEF and FEV_1 in children aged 5 years and above according to the American Thoracic Society (ATS)/European Respiratory Society (ERS)'s guidelines.[67, 68] The test is done in standing position without nose clips. The child is taught to breathe in very deeply, put the mouthpiece in the mouth in-between the teeth with lips tightly closed over it, blow out the air into the device hard and fast and continue to blow it out without pausing. This is repeated at least two more times, for a minimum of three and maximum of eight times in total. The peak flow meter has an in-built quality check mechanism that rejects 'blows' characterise by poor efforts, cough or inadequate expiratory phase.[69] The best of at least three readings is recorded as the PEF and FEV_1.[67, 70]

Self-monitoring of peak expiratory flow at home

Each child will be given a mini-Wright peak flow meter. The child is taught to use the device thus: ensure the red marker is at the 0 level before use; attach the mouthpiece to the device; ensure there is no object obstructing the movable marker of the meter; breath in deeply, put the mouthpiece inside the mouth in-between the teeth with the lips closed around it and 'blow' out hard and fast into the device; read off the value and record it on the record paper diary provided (Additional File 2). The steps are repeated at least 2 more times. The child is requested to do this for 5 days and record the readings on the diary. Peak flow variability (PFV) will be calculated as (difference between highest & lowest PEF)/100% (mean of highest & lowest PEF) for each day and the average over 3 days derived as the PFV.

Exposure Measures: HAP and SHS

Measurement of household air $PM_{2.5}$ and CO

Household air pollution (HAP) is quantitatively measured with air pollution/quality monitors for $PM_{2.5}$ and *CO* (Figure 2), and qualitatively by questions on cooking fuel types used at home, frequency of use of generators and distance of generator from bedroom (Additional File 1).[71]

Household $PM_{2.5}$ levels: is measured with a $PM_{2.5}$ sensor (PurpleAir PA-11-SD® monitor, PurpleAir, Inc. Utah, USA; https://www2.purpleair.com/collections/air-quality-sensors/products/purpleair-pa-ii-sd; Figure 2). The purple air sensor is a laboratory- and field-validated device that measures outdoor and indoor particulate matter including $PM_{2.5}$. The device has a removable memory card where the $PM_{2.5}$ data is continuously logged and from here the data can be retrieved when inserted into to a computer.[59, 60, 72]

CO Measurement: *CO* is measured real-time by a CO logger (Easy Log EL-USB-CO® carbon monoxide logger, Lascer Electronics, Salisbury, UK; https://www.lascarelectronics.com/easylog-el-usb-co), a small hand-held pen-like pocket device. It measures CO between 3 – 1,000 parts per million CO with an accuracy of ±7 parts per million CO. It can take up to 32,510 readings logged every 5 seconds to 5 minutes as determined by the user. Its data is retrieved by inserting the USB end into a computer on which the devices' software has already been stored.

Installation and retrieval of air pollution monitors

Both devices – purple air $PM_{2.5}$ monitor and EL-USB-CO logger- are set-up at the clinic, placed in a leather carriage bag with vents (to allow air flow freely through the bag; Figure 2) and handed over to the child's parents. The $PM_{2.5}$ device is connected with a USB cord to a power-bank of 40,000-60,000mAh which can power it for at least 5 days. The *CO* logger has a battery which can last three months. Parents are only required to take the bag home and place it in the room where the child sleeps, assumed to be where the child spends most time at home.[42] The devices commence logging data from the day of enrolment to the fifth day after enrolment when they will be retrieved from the participant's home by a research assistant. After retrieving the air pollution monitors, the logged data are retrieved. For the $PM_{2.5}$ monitors, the SD memory card at the base is removed and inserted into a laptop. The $PM_{2.5}$ data, in micrograms/m^3, are logged by the device in form of CSV files. The $PM_{2.5}$ device logs data every 2 minute continuously. For the *CO* logger, the USB end of the device is connected directly to the laptop and the data extracted with the computer. The *CO* logger measures *CO* as *parts per million* (to be converted to microgram/m^3 by multiplication factor of 1.25*)*.[39] Data obtained from the logged data will include: average 24-hour $PM_{2.5}$ and *CO* obtained from 3-day readings (the readings of the first and last days are discarded during

data retrieval and the readings of the full 3 days in-between are used); peak $PM_{2.5}$ and *CO*over the 3-day period; and the proportion of time the concentration exceeded the current WHO daily threshold of 15 ug/m^3 for $PM_{2.5}$ and 4 mg/m^3 for *CO* .[39]

Household activities diary: To possibly correlate peaks in $PM_{2.5}$ and *CO* levels with indoor activities, parents are asked to fill a daily activity diary during the 5 days of date-logging (Additional File 3). They record the time of common home activities like cooking, use of generator, sweeping, etc (see supplementary file). The diary helps provide feedbacks to the families.

Second-hand smoking (SHS)

SHS is quantitatively assessed by measurement of urine cotinine in *ng/ml* and qualitatively by questions on exposure of the child to smokers in and outside the home. Urine cotinine test strips (*AllTest^{TM} Cotinine test strips, Hangzhou AllTest Biotech Co., Ltd., China*; https://en.alltests.com.cn/Home/ProductInfo/98) which measures urine cotinine semi-quantitatively will be used to measure cotinine in the children's urine. The child's caregiver is given a clean universal plain bottle into which urine of the child is collected. The test strip is dipped into the urine per manufacturer's instruction. Any urine sample positive with the 10 ng/ml strip will be tested with higher concentration strip(s) (20, 50, 100, 200 and 500 ng/ml strips) until a negative result is obtained. The cotinine level will be assumed to be less than the level at which a negative result is obtained, namely *negative or < 10 ng/ml* (negative with 10 ng/ml strip), *10-20 ng/ml* (positive with 10 ng/ml strip but not with 20 ng/ml strip), *20-50 ng/ml* (positive with 20 ng/ml strip but not with 50 ng/ml strip), *50-100 ng/ml* (positive with 50 ng/ml strip but not with 100 ng/ml strip) *100-200* ng/ml (positive with 100 ng/ml strip but not with 200 ng/ml strip), *200-500 ng/ml* (positive with 200 ng/ml strip but not with 500 ng/ml strip) and > *500 ng/ml* (positive with all strips including 500 ng/ml strip).

Confounders Measurements

Potential confounding factors of AC are assessed including medication adherence, inhaler technique, socioeconomic status, anthropometric/nutritional status, personal/family history of allergic comorbidities[15,

21], parental knowledge of asthma[73][15], age at asthma diagnosis, sex, overcrowding, place of sleep (floor, mat, raised bed); exposure to pets; exacerbation triggers[15]] and obesity (BMI).

Inhaler Technique Assessment

The Inhaler Device Assessment Tool (IDAT)[74] is used to assess the inhalation technique of participants. The IDAT was designed for assessing inhalation technique among healthcare workers, CwA and their caregivers. The tool consists of separate checklists for assessing the use of various forms of inhalers – metered-dose inhaler (MDI) alone, MDI with spacer, MDI with spacer and mask and dry-powder inhalers. Each form consists of 5 steps which are scored as 0 or 1 where 0 is incorrect and 1 is correct. The minimum and maximum scores are 0 and 5, respectively. The baseline scores for each device type in use by the child is recorded. For those using more than one device, the average score is obtained.

Assessment of controller medication adherence

This is done by the question "*in the last 4 weeks, how many days in a week did you forget to take your inhaler?*" (Additional File 1). This is scored as *poor* adherence (>5), *moderate* adherence (3-5), *good* adherence (<3).

Socioeconomic status index

A validated socioeconomic scale developed for Nigerian children and household by Ibadin & Akpede[75] (modified from a pre-existing classification by Oyedeji[76]) will be used to assess the socioeconomic status of the parents/caregivers (both or one parents/guardian). The scale uses the occupation and education of the parents to classify them into classes 1-6, with classes 1 and 6 being highest and lowest, respectively.

Nutritional status: BMI

BMI will be derived from height and weight (weight/height^2) obtained from the medical records or measured directly per standard procedures.[77]

Allergic comorbidities

Parents and/or children are asked about symptoms suggestive of and treatment for allergic rhinitis, conjunctivitis, dermatitis or food allergy.

Asthma knowledge

Parental asthma–related knowledge is assessed with a validated 10-item Asthma Knowledge Questionnaire, adapted by Franken *et al*[78] from previously published questions. The question is reliable with a Cronbach's alpha of 0.72.

Quality of life

The Asthma Module of the Paediatric Quality of Life (PedsQL^{TM}) questionnaire[79] will be used to determine the psychosocial burden of asthma on the child. The Asthma module tool consists of four scales: "problems with asthma" (11 items), "problems with treatment" (11 items), "problems with worry" (3 items), and "problems with communication."

Feedbacks to study participants

Since most of the assessments in this study are part of standard asthma care, caregivers and children with asthma are provided feedbacks for improved care during contact, for example on corrective steps on inhaler technique; education on importance and strategies for medication adherence; and corrective feedbacks on parental knowledge. After data retrieval and extraction, attempt is made to identify any potential sources of observable spikes in $PM_{2.5}$ and *CO*, and SHS in those with positive urine cotinine. Results of $PM_{2.5}$ and *CO* and measures to mitigate further/future exposures are suggested through a written report sent to parents. Additionally, at the conclusion of the project, the team hopes to design context-specific general educational interventions for all patients/parents and asthma providers (doctors and nurses) to address all factors identified as associated with suboptimal AC.

330 ***Quality control and safety***

331 The PI is trained and experienced in the set-up and use of the air pollution monitors having been part of a
332 multinational study group that longitudinally measured ambient $PM_{2.5}$ in sub-Saharan African countries including
333 Nigeria.[59, 80][59] Each *$PM_{2.5}$* has two in-built sensors allowing for inter-rater comparison of the readings of
334 these sensors. Standard pulmonary function infection control procedures is followed during lung function testing:
335 routine cleaning of the electronic peak flow meter and the mini-Wright peak flow meters in-between participants'
336 use as per manufacturer's instructions; use of single-user sterilised disposable mouthpiece with one-way filter
337 (Safe T-way^{TM} mouthpiece, Vitalograph, Ireland); use of a well-ventilated non-crowded room at the clinic;
338 exclusion of children with current coughs from lung function measurement.

339 ***Analysis plan***

340 Data will be deidentified and anonymised and then analysed with R-based graphical user's interphase softwares:
341 Bluesky statistics (https://www.blueskystatistics.com) and JASP statistics (https://jasp-stats.org). After data
342 cleaning and assessment for normality of distribution (with Shapiro-Wilk test), continuous variables (e.g. age,
343 age at diagnosis, $PM_{2.5}$, CO level) will be summarized with mean ± SD [or median (IQR) if skewed], while
344 categorical variables (prevalence of the levels of asthma control) will be summarized with frequencies,
345 percentages with confidence intervals. One-way ANOVA (or Kruskal-Wallis test if skewed) and Chi-square test
346 will be used to compare continuous variables (e.g. *$PM_{2.5}$*, *CO*) and categorical variables (e.g. sex, age-group,
347 socioeconomic status) among the levels of asthma control, respectively. Pearson's correlation (or Spearman's if
348 skewed) will be used to compare two continuous variables (e.g. between *$PM_{2.5}$* and each of PEF, PFV and FEV_1;
349 between in-clinic PEF and home personal PEF). Bland-Altman analysis will also be used to explore the level of
350 agreement between in-clinic PEF and home-measured PEF. We will use hierarchical multivariable multinomial
351 logistic regression to determine the independent association between AC and each of *$PM_{2.5}$* , *CO* and urinary
352 cotinine while controlling for potential confounders such as age, sex, ethnicity, residence area, season, family
353 history of asthma/atopy, comorbid atopy, adherence, inhaler technique, asthma knowledge, psychosocial burden
354 and socioeconomic level. Effect sizes of statistical associations will be quantified with adjusted and unadjusted

355 odds ratio at significant levels of <0.05 and 95% confidence intervals. Missing data will be handled on case-by-
356 case basis with respect to each affected variable.

357

358

DISCUSSION

There is need for more studies on air pollution from Africa because, generally, air pollution appears to be of little priority to governments across Africa compared to other parts of the world.[8, 25, 39, 59, 60, 81–83] Studies on the association of air pollution with health outcomes such as asthma incidence, morbidity or control are important, not only for characterising the burden and determinants of such outcomes to enhance clinical care, but also for reinforcing education and advocacy for improved environmental control at all levels- personal, household, community, national, regional and global.[48] For example, profiling of the pattern of household $PM_{2.5}$ and CO along with household activities may inform personalised education and counselling of cooking fuel choices, practices and placement within the home.[48] These may be followed by advocacy for proven interventions like home air purifiers for cleaner air and better asthma control in children.[48, 84] Additionally, identifying the burden or presence of SHS may enhance counselling for smoking cessation among parents/caregivers.[85] Whereas few studies in Nigeria have found household $PM_{2.5}$ and CO levels to be associated with asthma incidence,[44–46] and questionnaire-assessed SHS to be associated with increased respiratory symptoms in the general adolescent population,[53] no study has specifically explored their influences on asthma control among children with asthma. Hence, the ongoing study would provide important data along this line. Although a recent multinational study conducted among children with asthma across sub-Saharan Africa including Nigeria provided valuable implementable insights into their personal $PM_{2.5}$ exposures in different micro-environments (school, home and commuting), the association of $PM_{2.5}$ with their symptom control or lung function was not reported.[8]

AC is an interplay between several factors within and without the affected person, including the individual's genetic background, medications, socio-environmental and psychosocial factors.[1, 86] Achieving optimal asthma control (AC) requires identifying, quantifying and ameliorating these modifiable risk factors like access to medications, medication use technique, adherence and air pollution.[1, 9, 30, 38, 87] Standardised assessment of asthma control consists of two domains: the assessment of control of asthma symptoms like cough, wheezing, breathlessness and chest tightness, and the assessment of the future risk of adverse outcomes like exacerbations, persistent loss of lung function and medication side-effects.[1] The former is assessed by determining the

frequency of day- and night-time symptoms, activity limitation and exacerbation over the preceding four weeks while the latter is assessed by determining, amongst others, frequency of exacerbation in last one year, medication adherence, inhaler technique, psychosocial burden and lung function. Examples of standardised validated and reliable tools for assessing asthma symptom control includes the GINA questionnaire,[1] Asthma Control Test (ACT) and Asthma Control Questionnaire (ACQ).[1, 66] Some studies suggest that AC may be better assessed with combination of GINA and ACT questionnaires, rather than just either of them alone.[88, 89] Thus, the assessment of asthma control encompasses questionnaires-based assessment of symptom control and medication adherence; assessment of inhaler technique; and measurement of lung function in the clinic and at home.

Ishaq *et al*[16] reported non-correlation between asthma symptoms and objectively-measured lung function among asthmatic children in Sokoto, Nigeria, presumably because the two parameters measure different domains of asthma control; they are complementary to each other in assessing the overall respiratory health of children with asthma, or because one precedes the other in detecting airway abnormalities. It is known that poor lung function is more predictive of future adverse event rather than current control. The risk of future adverse outcomes like exacerbation is assessed with lung function parameters such as forced expiratory volume in 1 second (FEV_1), peak expiratory flow (PEF) or its variability. Low predicted FEV_1, especially when lower than 60% predicted, and exaggerated day-to-day diurnal PEF variability are associated with poor AC and increased risk of exacerbation even when symptom control is optimal.[90] Hence, the optimal assessment of AC should consist of the assessment of these two domains- symptom assessment with AC questionnaires and objective measurement of lung function with spirometry or peak flow meters-as both domains are essentially complementary in assessing control of asthma.[1]

Although the main harmful product of tobacco smoking is nicotine, it is not usually measured as an indicator of exposure to SHS because of its very short half-life (about 2 hours). On the other hand, cotinine- a by-product of nicotine- has a long half-life and can detect nicotine exposure in the last 3-4 days. Cotinine can be measured in the blood, urine or saliva; however, urine cotinine is a more sensitive and specific marker of SHS compared to

serum or saliva cotinine. [58, 91, 92] Exposure to SHS, assessed with cotinine, is strongly correlated with systemic inflammation.[93] Although self-reported SHS using questionnaire is less reliable than urine cotinine,[58] the use of the two provides a better assessment of SHS rather than either. While biomarkers such as cotinine measure short-term exposures to SHS, questionnaires measure long-term exposures, albeit imprecisely.[23] Thus, in this study, the assessment of SHS is being done by both questionnaire and a semi-quantitative assay with a urine test strip. A urinary cotinine cut-off point of 18ng/ml has been proposed as cut-off level for the detection of SHS;[94] however there is no universally agreed cut-off.

A potential limitation of our approach to air quality measurement is our use of household rather than personal measurement; personal measurement is a better reflection of true exposure compared to household measurement, allowing for delineating sources, context and cumulative exposures.[8, 82] However, personal exposure may be cumbersome for participants as they are required to carry the devices about. Also, merely measuring a 3-day averaged exposure may be inadequate to reflect true exposure over time.[72] Likewise, one-time cross-sectional measurement of outcomes (AC, lung function), rather than serially/longitudinally, may be inadequate to capture a potentially complex biological inter-relationship as that between pollutants and health outcomes.[82] Nonetheless, compared to previous studies, this study may provide valuable data for reinforcing personalised asthma care with respect to asthma care.

LIST OF ABBREVIATION

AC	Asthma Control
ACT	Asthma Control Test
AP	Air Pollution
CO	Carbon Monoxide
CwA	Children with Asthma
FEV_1	Forced Expiratory Volume in 1 Second
GINA	Global Initiative for Asthma
HAP	Household Air Pollution
PEF	Peak Expiratory Flow
PFV	Peak Flow Variability
$PM_{2.5}$	Particulate Matter less than 2.5 microns in Aerodynamic Diameter
SHS	Second Hand Smoking

DECLARATIONS

Ethical approval and consent to participate

The study was approved by the Health Research & Ethics Committee (HREC) of LASUTH (LHREC/06/10/1900, version 1c). Written informed parental consent is obtained from parents/guardians, as well as assent from children aged 6 years and above, after explaining the study to the parent and child.

Consent for publication

Not applicable

Availability of data and materials

Data generated from this study would be available from the authors upon reasonable request. Also, data sharing may be through Open Science Framework (OSF): https://doi.org/10.17605/OSF.IO/2JS7P

Competing interests

The authors declare that they have no competing interests

Funding:

Pan African Thoracic Society (PATS) Methods in Epidemiologic, Clinical & Operations Research (MECOR) Small Grants. Although funder suggested some modifications to study scope, the funder largely had no role in study conception, design, data collection, analysis and publication.

Authors' contributions

POU conceptualized, designed, drafted, reviewed and revised the protocol. OA contributed to study conceptualisation and design. BA contributed to study design. OO contributed to study design. All authors read, reviewed and approved the final manuscript.

Acknowledgements

The authors thank the participating children and their parents/caregivers, as well as the nursing and medical staff in the participating outpatient clinics. Of note the authors thank the various sets of residents who have been supporting data collection. The lead author is grateful to Engineer Chris Ubuane for helping to procure some of the devices from the Uk. Participants or the public were not involved in study design. A conference abstract has been presented and published in a local journal from this research, comprising the first 10 enrolled participants (https://dx.doi.org/10.4314/njp.v51i2.08).

REFERENCES

1. Global Initiative for Asthma (GINA). Global strategy for asthma management and prevention, updated 2025. 2025.

2. Yuan L, Tao J, Wang J, She W, Zou Y, Li R, et al. Global, regional, national burden of asthma from 1990 to 2021, with projections of incidence to 2050: a systematic analysis of the global burden of disease study 2021. eClinicalMedicine. 2025;80:103051. https://doi.org/10.1016/j.eclinm.2024.103051.

3. Adeloye D, Chan KY, Rudan I, Campbell H. An estimate of asthma prevalence in Africa: A systematic analysis. Croat Med J. 2013;54:519–31. https://doi.org/10.3325/cmj.2013.54.519.

4. Ozoh OB, Aderibigbe SA, Ayuk AC, Desalu OO, Oridota OE, Olufemi O, et al. The prevalence of asthma and allergic rhinitis in Nigeria: A nationwide survey among children, adolescents and adults. PLoS One. 2019;14:e0222281. https://doi.org/10.1371/journal.pone.0222281.

5. Falade AG, Ige OM, Yusuf BO, Onadeko MO, Onadeko BO. Trends in the Prevalence and Severity of Symptoms of Asthma, Allergic Rhinoconjunctivitis, and Atopic Eczema. J Natl Med Assoc. 2009;101:414–8.

6. Kuti B. Childhood Asthma in Nigeria: A Review of the Burden and Challenges of Management. Ann Heal Res. 2020;6:351–69. https://doi.org/10.30442/ahr.0604-01-99.

7. Oyenuga VO, Mosler G, Addo-Yobo E, Adeyeye OO, Arhin B, Fortune F, et al. Asthma symptoms, severity, and control with and without a clinical diagnosis of asthma in early adolescence in sub-Saharan Africa: a multi-country, school-based, cross-sectional study. Lancet Child Adolesc Heal. 2024;8:859–71. https://doi.org/10.1016/S2352-4642(24)00232-3.

8. Lim S, Said B, Zurba L, Mosler G, Addo-Yobo E, Adeyeye OO, et al. Characterising sources of PM2·5 exposure for school children with asthma: a personal exposure study across six cities in sub-Saharan Africa.

491 Lancet Child Adolesc Heal. 2024;8:17–27. https://doi.org/10.1016/S2352-4642(23)00261-4.

492 9. Ayuk AC, Ramjith J, Zar HJ. Environmental risk factors for asthma in 13-14 year old African children.
493 Pediatr Pulmonol. 2018;:1–10. https://doi.org/10.1002/ppul.24162.

494 10. Pecoraro L, Lanzilotti AG, De Musso M, Di Muri E, Tramacere F, Altavilla E, et al. The Role of
495 Environmental Exposures in Pediatric Asthma Pathogenesis: A Contemporary Narrative Review. Children.
496 2025;12:1327. https://doi.org/10.3390/children12101327.

497 11. Global Asthma Network. The Global Asthma Report 2022. Auckland, New Zealand; 2022.

498 12. Taylor DR, Bateman ED, Boulet LP, Boushey HA, Busse WW, Casale TB, et al. A new perspective on
499 concepts of asthma severity and control. Eur Respir J. 2008;32:545–54.
500 https://doi.org/10.1183/09031936.00155307.

501 13. Ayuk AC, Oguonu T, Ikefuna AN, Ibe BC. Asthma control and quality of life in school- age children in
502 Enugu south east, Nigeria. Niger Postgrad Med J. 2014;21:160–4.

503 14. Oviawe O, Osarogiagbon WO. Trend in asthma severity in steroid naive asthmatic children in Benin city,
504 Nigeria. Niger J Clin Pract. 2013;16:371–4.

505 15. Kuti BP, Omole KO, Kuti DK. Factors associated with childhood asthma control in a resource-poor center. J
506 Fam Med Prim care. 2017;6:222–30. https://doi.org/10.4103/jfmpc.jfmpc_271_16.

507 16. Ishaq FA, Garba BI, Jiya NM, Hamidu A. Assessment of asthma control and lung function in asthmatic
508 children in Sokoto, North Western Nigeria. J Pan African Thorac Soc. 2021;2:148–53.
509 https://doi.org/10.25259/jpats_8_2021.

510 17. Fehrenbach H, Wagner C, Wegmann M. Airway remodeling in asthma: what really matters. Cell Tissue
511 Res. 2017;367:551. https://doi.org/10.1007/S00441-016-2566-8.

512 18. Huang Y, Qiu C. Research advances in airway remodeling in asthma: a narrative review. Ann Transl Med.

513 2022;10:1023–1023. https://doi.org/10.21037/ATM-22-2835.

514 19. Mosler G, Oyenuga V, Addo-Yobo E, Adeyeye OO, Masekela R, Mujuru HA, et al. Asthma Symptoms,
515 Knowledge, and Perception in Sub-Saharan African Adolescents - Preliminary Results of the Achieving Control
516 of Asthma in Children in Africa (ACACIA) Study. Am J Respir Crit Care Med. 2021;203:A1735.
517 https://doi.org/10.1164/ajrccm-conference.2021.203.1_meetingabstracts.a1735.

518 20. Ozoh OB, Ayuk AC, Ukwaja KN, Desalu OO, Olufemi O, Aderibigbe SA, et al. Asthma management and
519 control in Nigeria: the asthma insight and reality Nigeria (AIRNIG) study. Expert Rev Respir Med.
520 2019;13:917–27. https://doi.org/10.1080/17476348.2019.1651201.

521 21. Ayuk A, Eze J, Edelu B, Oguonu T. The prevalence of allergic diseases among children with asthma: What
522 is the impact on asthma control in South East Nigeria? Niger J Clin Pract. 2018;21:632.
523 https://doi.org/10.4103/njcp.njcp_343_17.

524 22. Fuller R, Landrigan PJ, Balakrishnan K, Bathan G, Bose-O'Reilly S, Brauer M, et al. Pollution and health: a
525 progress update. Lancet Planet Heal. 2022;6:e535–47. https://doi.org/10.1016/S2542-5196(22)00090-0.

526 23. Öberg M, Jaakkola MS, Prüss-Üstün A, Schweizer C, Woodward A. Second hand smoke: Assessing the
527 burden of disease at national and local levels. 2010.

528 24. Katoto PDMC, Byamungu L, Brand AS, Mokaya J, Strijdom H, Goswami N, et al. Ambient air pollution
529 and health in Sub-Saharan Africa: Current evidence, perspectives and a call to action. Environ Res.
530 2019;173:174–88. https://doi.org/10.1016/j.envres.2019.03.029.

531 25. Mead MI, Okello G, Mbandi AM, Pope FD. Spotlight on air pollution in Africa. Nat Geosci. 2023;16:930–
532 1. https://doi.org/10.1038/s41561-023-01311-2.

533 26. The National Population Commission (NPC) [Nigeria] and ICF. Nigeria Demographic and Health Survey
534 2018. Abuja, Nigeria and Rockville, Maryland, USA; 2019.

27. State of Global Air Report 2024 | Climate, Environment, Energy, and Disaster Risk Reduction. https://knowledge.unicef.org/CEED/resource/state-global-air-report-2024. Accessed 1 Jan 2026.

28. Zigabe SM, Tamuzi JL, Toelen J, Hoet PHM, Katoto PDMC. Impact of prenatal and postnatal household air pollution exposure on respiratory morbidity and lung function in sub-Saharan African children: a systematic review and meta-analysis. Environ Heal A Glob Access Sci Source. 2025;24:66. https://doi.org/10.1186/s12940-025-01216-0.

29. Rylance S, Masekela R, Banda NPK, Mortimer K. Determinants of lung health across the life course in sub-Saharan Africa. Int J Tuberc Lung Dis. 2020;24:892–901. https://doi.org/10.5588/ijtld.20.0083.

30. Meghji J, Mortimer K, Agusti A, Allwood BW, Asher I, Bateman ED, et al. Improving lung health in low-income and middle-income countries: from challenges to solutions. Lancet. 2021;397:928–40. https://doi.org/10.1016/S0140-6736(21)00458-X.

31. Okello G, Devereux G, Semple S. Women and girls in resource poor countries experience much greater exposure to household air pollutants than men: Results from Uganda and Ethiopia. Environ Int. 2018;119:429–37. https://doi.org/10.1016/J.ENVINT.2018.07.002.

32. Aithal SS, Gill S, Satia I, Tyagi SK, Bolton CE, Kurmi OP. The Effects of Household Air Pollution (HAP) on Lung Function in Children: A Systematic Review. Int J Environ Res Public Health. 2021;18:11973. https://doi.org/10.3390/ijerph182211973.

33. Oluwole O, Arinola GO, Huo D, Olopade CO. Household biomass fuel use, asthma symptoms severity, and asthma underdiagnosis in rural schoolchildren in Nigeria: a cross-sectional observational study. BMC Pulm Med. 2017;17:3. https://doi.org/10.1186/s12890-016-0352-8.

34. Masekela R, Vanker A. Lung Health in Children in Sub-Saharan Africa: Addressing the Need for Cleaner Air. Int J Environ Res Public Health. 2020;17:1–13. https://doi.org/10.3390/IJERPH17176178.

35. McCormack MC, Breysse PN, Matsui EC, Hansel NN, Williams D, Curtin-Brosnan J, et al. In-home particle concentrations and childhood asthma morbidity. Environ Health Perspect. 2009;117:294–8. https://doi.org/10.1289/ehp.11770.

36. Isiugo K, Jandarov R, Cox J, Ryan P, Newman N, Grinshpun SA, et al. Indoor particulate matter and lung function in children. Sci Total Environ. 2019;663:408–17. https://doi.org/10.1016/j.scitotenv.2019.01.309.

37. Puzzolo E, Fleeman N, Lorenzetti F, Rubinstein F, Li Y, Xing R, et al. Estimated health effects from domestic use of gaseous fuels for cooking and heating in high-income, middle-income, and low-income countries: a systematic review and meta-analyses. Lancet Respir Med. 2024;12:281–93. https://doi.org/10.1016/S2213-2600(23)00427-7.

38. Oluwole O, Ana GR, Arinola GO, Wiskel T, Falusi AG, Huo D, et al. Effect of stove intervention on household air pollution and the respiratory health of women and children in rural Nigeria. Air Qual Atmos Heal. 2013;6:553–61. https://doi.org/10.1007/s11869-013-0196-9.

39. World Health Organisation. WHO global air quality guidelines. Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Geneva; 2021.

40. Juskiene I, Prokopciuk N, Franck U, Valiulis A, Valskys V, Mesceriakova V, et al. Indoor air pollution effects on pediatric asthma are submicron aerosol particle–dependent. Eur J Pediatr 2022 1816. 2022;181:2469–80. https://doi.org/10.1007/S00431-022-04443-6.

41. Ni R, Su H, Burnett RT, Guo Y, Cheng Y. Long-term exposure to PM2.5 has significant adverse effects on childhood and adult asthma: A global meta-analysis and health impact assessment. One Earth. 2024;7:1953–69. https://doi.org/10.1016/j.oneear.2024.09.022.

42. Woods KE, Apsley A, Semple S, Turner SW. Domestic airborne fine particulate matter exposure and asthma control among children receiving inhaled steroid treatment: http://dx.doi.org/101177/1420326X14527301. 2014;23:497–503. https://doi.org/10.1177/1420326X14527301.

580 43. Zora JE, Sarnat SE, Raysoni AU, Johnson BA, Li WW, Greenwald R, et al. Associations between urban air
581 pollution and pediatric asthma control in El Paso, Texas. Sci Total Environ. 2013;448:56–65.
582 https://doi.org/10.1016/J.SCITOTENV.2012.11.067.

583 44. Eghomwanre AF, Oguntoke O, Taiwo AM. Levels of indoor particulate matter and association with asthma
584 in children in Benin City, Nigeria. Environ Monit Assess. 2022;194. https://doi.org/10.1007/s10661-022-10135-
585 3.

586 45. Eghomwanre AF, Imarhiagbe EE. Associations between Indoor Bioaerosol Concentrations and the
587 Prevalence of Childhood Asthma in Benin City , Southern Nigeria. J Mater Environ Sci. 2025;16:2076–90.

588 46. Eghomwanre AF, Oguntoke O. Concentrations of indoor gaseous air pollutants and risk factors associated
589 with childhood asthma in Benin City, Nigeria. Environ Monit Assess. 2022;194.
590 https://doi.org/10.1007/s10661-022-10026-7.

591 47. Ozoh OB, Okwor TJ, Adetona O, Akinkugbe AO, Amadi CE, Esezobor C, et al. Cooking Fuels in Lagos,
592 Nigeria: Factors Associated with Household Choice of Kerosene or Liquefied Petroleum Gas (LPG). Int J
593 Environ Res Public Heal 2018, Vol 15, Page 641. 2018;15:641. https://doi.org/10.3390/IJERPH15040641.

594 48. Robertson NM, Qiu A, Raju S, McCormack MC, Koehler K. Cleaning indoor air—what works for
595 respiratory health: An updated literature review and recommendations. J Allergy Clin Immunol. 2024;154:847–
596 60. https://doi.org/10.1016/j.jaci.2024.08.011.

597 49. Yu O, Sheppard L, Lumley T, Koenig JQ, Shapiro GG. Effects of Ambient Air Pollution on Symptoms of
598 Asthma in Seattle-Area Children Enrolled in the CAMP Study. Environ Heal Perspect •. 2000;108.

599 50. Pollock J, Shi L, Gimbel RW. Outdoor Environment and Pediatric Asthma: An Update on the Evidence
600 from North America. Can Respir J. 2017;2017. https://doi.org/10.1155/2017/8921917.

601 51. Njoku KL, Rumide TJ, Akinola MO, Adesuyi AA, Jolaoso AO. Ambient Air Quality Monitoring in

602 Metropolitan City of Lagos, Nigeria. J Appl Sci Environ Manag. 2016;20:178.
603 https://doi.org/10.4314/JASEM.V20I1.21.

604 52. Farber HJ, Knowles SB, Brown NL, Caine L, Luna V, Qian Y, et al. Secondhand tobacco smoke in children
605 with asthma: Sources of and parental perceptions about exposure in children and parental readiness to change.
606 Chest. 2008;133:1367–74. https://doi.org/10.1378/chest.07-2369.

607 53. Oyapero A, Erinoso OA, Olatosi OO. Second-Hand Tobacco Smoke Exposure in Children: A Neglected
608 Driver of Health Disparities and Social Injustice in Nigeria. 2022; January.

609 54. Tiotiu AI, Novakova P, Nedeva D, Chong-Neto HJ, Novakova S, Steiropoulos P, et al. Impact of air
610 pollution on asthma outcomes. International Journal of Environmental Research and Public Health. 2020;17:1–
611 29. https://doi.org/10.3390/ijerph17176212.

612 55. He Z, Wu H, Zhang S, Lin Y, Li R, Xie L, et al. The association between secondhand smoke and childhood
613 asthma: A systematic review and meta-analysis. Pediatric Pulmonology. 2020;55:2518–31.
614 https://doi.org/10.1002/ppul.24961.

615 56. Hollenbach JP, Schifano ED, Hammel C, Cloutier MM. Exposure to secondhand smoke and asthma severity
616 among children in Connecticut. PLoS One. 2017;12:1–13. https://doi.org/10.1371/journal.pone.0174541.

617 57. Neophytou AM, Oh SS, White MJ, Mak ACY, Hu D, Huntsman S, et al. Secondhand smoke exposure and
618 asthma outcomes among African-American and Latino children with asthma. Thorax. 2018;73:1041–8.
619 https://doi.org/10.1136/thoraxjnl-2017-211383.

620 58. Ozoh OB, Dania MG, Irusen EM. The Prevalence of Self-Reported Smoking and Validation with Urinary
621 Cotinine Among Commercial Drivers in Major Parks in Lagos, Nigeria. J Public Health Africa. 2014;5:47–50.
622 https://doi.org/10.4081/JPHIA.2014.316.

623 59. Awokola BI, Okello G, Mortimer KJ, Jewell CP, Erhart A, Semple S. Measuring air quality for advocacy in

624 Africa (MA3): Feasibility and practicality of longitudinal ambient PM2.5 measurement using low-cost sensors.
625 Int J Environ Res Public Health. 2020;17:1–17. https://doi.org/10.3390/ijerph17197243.

626 60. Awokola B, Okello G, Johnson O, Dobson R, Ouédraogo AR, Dibba B, et al. Longitudinal Ambient PM2.5
627 Measurement at Fifteen Locations in Eight Sub-Saharan African Countries Using Low-Cost Sensors.
628 Atmosphere (Basel). 2022;13:1593. https://doi.org/10.3390/atmos13101593.

629 61. Batini C, Ahmed T, Ameer S, Kilonzo G, Ozoh OB, van Zyl-Smit RN. Smoking cessation on the African
630 continent: Challenges and opportunities. African J Thorac Crit care Med. 2019;25.
631 https://doi.org/10.7196/AJTCCM.2019.v25i2.015.

632 62. Mackay DF, Turner SW, Semple SE, Dick S, Pell JP. Associations between smoke-free vehicle legislation
633 and childhood admissions to hospital for asthma in Scotland: an interrupted time-series analysis of whole-
634 population data. Lancet Public Heal. 2021;6:e579–86. https://doi.org/10.1016/S2468-2667(21)00129-8.

635 63. Odusote OA, Ubuane PO, Akinola IJ. Allergic co-morbidities in childhood asthma in Lagos, Nigeria. Curr
636 Allergy Clin Immunol J. 2020;33:35. https://doi.org/10.1183/09031936.00070309.

637 64. Nigerian Thoracic Society. Guideline for Asthma Management in Nigeria. First. Nigerian Thoracic Society;
638 2017.

639 65. Liu AH, Zeiger R, Sorkness C, Mahr T, Ostrom N, Burgess S, et al. Development and cross-sectional
640 validation of the Childhood Asthma Control Test. J Allergy Clin Immunol. 2007;119:817–25.
641 https://doi.org/10.1016/j.jaci.2006.12.662.

642 66. Nathan RA, Sorkness CA, Kosinski M, Schatz M, Li JT, Marcus P, et al. Development of the Asthma
643 Control Test: A survey for assessing asthma control. J Allergy Clin Immunol. 2004;113:59–65.
644 https://doi.org/10.1016/j.jaci.2003.09.008.

645 67. Graham BL, Steenbruggen I, Barjaktarevic IZ, Cooper BG, Hall GL, Hallstrand TS, et al. Standardization of

spirometry 2019 update an official American Thoracic Society and European Respiratory Society technical statement. Am J Respir Crit Care Med. 2019;200:E70–88. https://doi.org/10.1164/rccm.201908-1590ST.

68. Ubuane PO, Ajiboye OA, Akinola AO, Adekunle MO, Akinyosoye G, Ajayi OA, et al. Development of reference values and equations for the pulmonary function of Nigerian children aged 6–11 years measured with digital peak flow meter and its validation against local and the Global Lung Function Initiative (GLI) equations. J Xiangya Med. 2024;9:1–21. https://doi.org/10.21037/jxym-23-29.

69. Vitalograph LTD. Vitalograph Asma-1 User Manual. Ennis, Ireland: Vitalograph; 2007.

70. Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R, et al. Interpretative strategies for lung function tests. Eur Respir J. 2005;26:948–68. https://doi.org/10.1183/09031936.05.00035205.

71. Saleh S, van Zyl-Smit R, Allwood B, Lawin H, Mbatchou Ngahane B, Ayakaka I, et al. Questionnaires for Lung Health in Africa across the Life Course. Int J Environ Res Public Health. 2018;15:1615. https://doi.org/10.3390/ijerph15081615.

72. Wang M, Chang D, Singh A, Wagner J, Wang ZM, Singer BC, et al. Practical Guidance for Using PurpleAir Particle Monitors for Indoor and Outdoor Measurements in Community Field Studies. Aerosol Air Qual Res. 2025;25:1–21. https://doi.org/10.1007/s44408-025-00048-4.

73. Ho J, Bender BG, Gavin LA, O'Connor SL, Wamboldt MZ, Wamboldt FS. Relations among asthma knowledge, treatment adherence, and outcome. J Allergy Clin Immunol. 2003;111:498–502. https://doi.org/10.1067/MAI.2003.160.

74. Davies B Cicutto L, Higuchi KS, McConnell H, Edwards N, MacPherson A & Clarke D DE. Nursing Best Practice Guidelines Evaluation User Guide: Inhaler Device Assessment Tool (IDAT) for Promoting Asthma Control in Children. Canada: Nursing Best Practice Research Unit, University of Ottawa; 2006.

75. Ibadin MO, Akpede GO. A revised scoring scheme for the classification of socio-economic status in

668 Nigeria. Niger J Paediatr. 2021;48:26–33. https://doi.org/10.4314/njp.v48i1.5.

669 76. Oyedeji EA. Socioeconomic and cultural background of hospitalized children in Ilesa. Niger J Paediatr.
670 1985;12:111–7.

671 77. Town C, Paediatric M, Group I. ANTHROPOMETRY GUIDELINE Paediatrics. 2009; April 2007.

672 78. Franken MMA, Veenstra–van Schie MTM, Ahmad YI, Koopman HM, Versteegh FGA. The presentation of
673 a short adapted questionnaire to measure asthma knowledge of parents. BMC Pediatr. 2018;18:14.
674 https://doi.org/10.1186/s12887-018-0991-4.

675 79. Varni JW, Burwinkle TM, Rapoff MA, Kamps JL, Olson N. The PedsQL^{TM}in pediatric asthma: Reliability
676 and validity of the Pediatric Quality of Life Inventory^{TM} Generic Core Scales and Asthma Module. J Behav
677 Med. 2004;27:297–318. https://doi.org/10.1023/B:JOBM.0000028500.53608.2c.

678 80. Awokola BI, Amusa GA, Okello G, Semple S, Jewell C, Erhart A, et al. Measuring Air Quality for
679 Advocacy in Africa (MA3): Can Longitudinal Ambient PM2.5 Measurement Be Done Satisfactorily Using
680 Low-Cost Sensors? In: A46. Health effects of air pollution and air toxics. American Thoracic Society; 2020. p.
681 A1780–A1780. https://doi.org/10.1164/ajrccm-conference.2020.201.1_MeetingAbstracts.A1780.

682 81. Makoni M. Air pollution in Africa. Lancet Respir Med. 2020;8:e60–1. https://doi.org/10.1016/S2213-
683 2600(20)30275-7.

684 82. Okello G, Mortimer K, Lawin H, Semple S. Quantifying exposure to respiratory hazards in sub-Saharan
685 Africa: planning your study. African J Respir Med. 2019;14:18-27 WE-Emerging Sources Citation Index
686 (ESCI).

687 83. Lim S, Bassey E, Bos B, Makacha L, Varaden D, Arku RE, et al. Comparing human exposure to fine
688 particulate matter in low and high-income countries: A systematic review of studies measuring personal PM2.5
689 exposure. Sci Total Environ. 2022;833 April:155207. https://doi.org/10.1016/j.scitotenv.2022.155207.

690 84. Cui X, Li Z, Teng Y, Barkjohn KK, Norris CL, Fang L, et al. Association between Bedroom Particulate
691 Matter Filtration and Changes in Airway Pathophysiology in Children with Asthma. JAMA Pediatr.
692 2020;174:533–42. https://doi.org/10.1001/jamapediatrics.2020.0140.

693 85. Osinibi M, Lawton A, Bossley C, Gupta A. Promoting smoking cessation in the paediatric respiratory clinic.
694 Eur J Pediatr. 2022;181:2863–5. https://doi.org/10.1007/s00431-022-04453-4.

695 86. Taylor DR, Bateman ED, Boulet LP, Boushey HA, Busse WW, Casale TB, et al. A new perspective on
696 concepts of asthma severity and control. Eur Respir J. 2008;32:545–54.
697 https://doi.org/10.1183/09031936.00155307.

698 87. Mortimer K, Nantanda R, Meghji J, Vanker A, Bush A, Ndimande N, et al. Africa's respiratory "Big Five."
699 J Pan African Thorac Soc. 2021;2:64–72. https://doi.org/10.25259/jpats_12_2021.

700 88. Akan A, Dibek Mlslrlloğlu E, Civelek E, Kocabaş CN. Determining the Best Tool Comparable with Global
701 Initiative for Asthma Criteria for Assessing Pediatric Asthma Control. https://home.liebertpub.com/ped.
702 2021;34:89–96. https://doi.org/10.1089/PED.2020.1334.

703 89. Deschildre A, Pin I, El Abd K, Belmin-Larrar S, El Mourad S, Thumerelle C, et al. Asthma control
704 assessment in a pediatric population: comparison between GINA/NAEPP guidelines, Childhood Asthma
705 Control Test (C-ACT), and physician's rating. Allergy. 2014;69:784–90. https://doi.org/10.1111/ALL.12402.

706 90. Buzoianu E, Moiceanu M, Doina ;, Plesca A. Asthma Control Assessment in Children: Correlation between
707 Asthma Control Test and Peak Expiratory Flow. Mædica. 2014;9:338.

708 91. Aina OO. Assessment of cotinine as a biomarker of cigarette smoking and self-reported smoking status
709 among undergraduate students of Ahmadu Bello University, Zaria. National Postgraduate Medical College of
710 Nigeria; 2017.

711 92. Abdullah N. Validation of Self-Reported Smoker and Second Hand Smoke Exposure by Urinary Cotinine

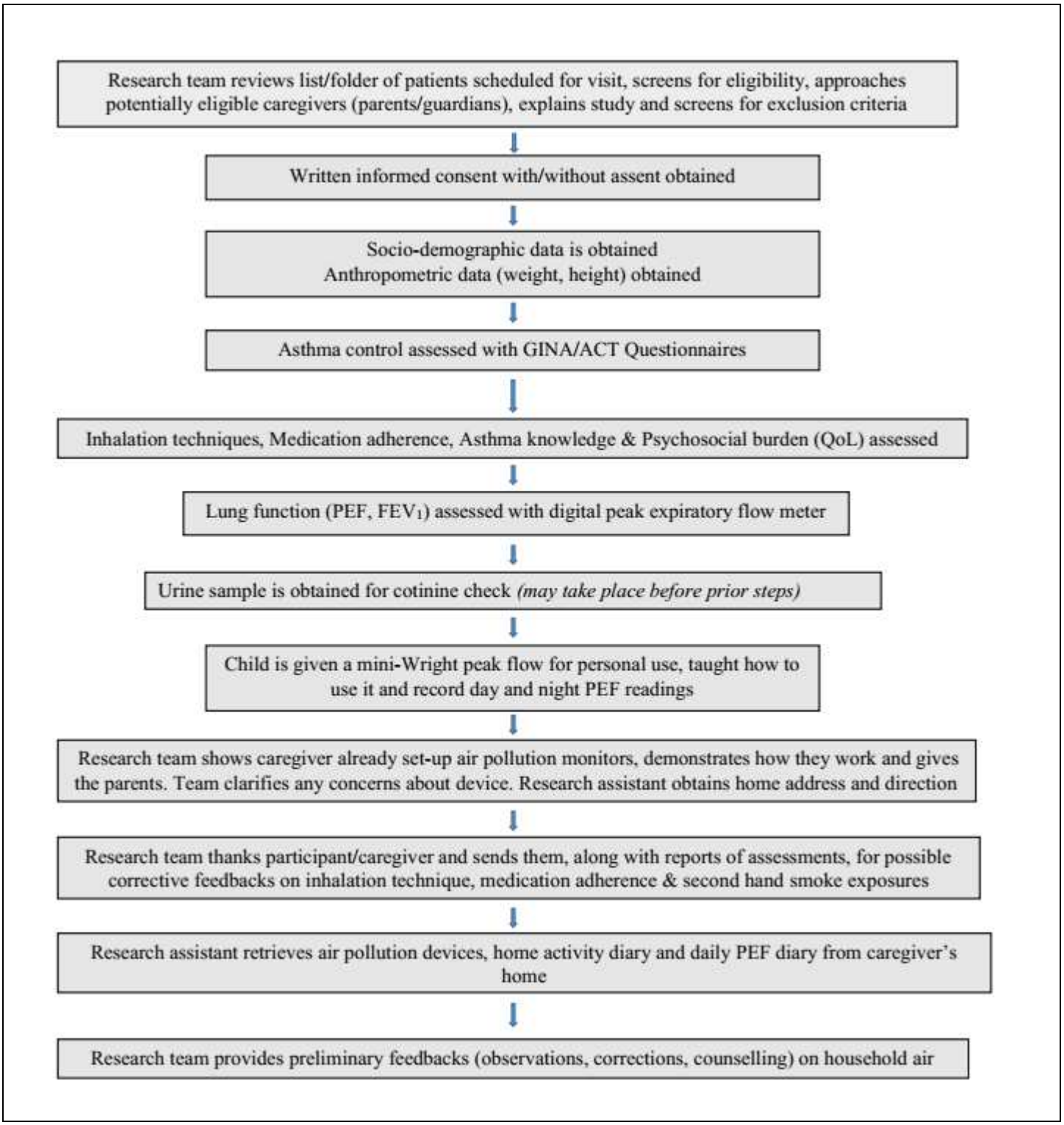
within The Malaysian Cohort Project. J Biomed Transl Res. 2019;5:15. https://doi.org/10.14710/jbtr.v5i1.3971.

93. Eworo ER, Okhormhe ZA, Offor BK, Nsonwu-anyanwu CA, Uduak IB, Atim E. Assessment of Serum Cotinine , C-Reactive Protein Levels and Body Mass Index in Smokers in Calabar , Nigeria. Ann Clin Case Stud. 2019;1:40–3.

94. Susanto AD, Putri PD, Hudoyo A, Taufik FF, Nurwidya F, Andarini S. Urinary cotinine level in Indonesian children exposed to domestic cigarette smoke. J Nat Sci Biol Med. 2018;9:77–81. https://doi.org/10.4103/jnsbm.JNSBM_119_17.

720 **FIGURES**

721



722 Figure 1. Individual participant's flow chart

723



A B

Figure 2. Carriage bag for devices: A shows an opened carriage bag containing a power bank (a), Easy Log EL-USB-CO® carbon monoxide logger (b) and a PurpleAir PA-11-SD® $PM_{2.5}$ sensor. B is a side-view of the carriage bag hanged on a wall, depicting the bag's air vents (d)

ADDITIONAL FILES

Additional File 1. docx. Data collection form

Additional File 2. docx. Personal peak flow meter record

Additional File 3. docx. Household activities diary

ADDITIONAL FILE 1

DATA COLLECTION FORM

	Date *(dd/mmm/yyyy)*	[__ __/__ __ __/__ __ __ __]				
	Hospital No (6 digits)	[__/__/__/__/__/__]	Research ID (3 digits)	[__/__/__]		
A.	**CHILD'S SOCIODEMOGRAPHICS**					
	Child's surname _____	First name _____	Middle name _____			
	Date of Birth *(dd/mmm/yyyy)*	[__ __/__ __ __/__ __ __ __]	Age in years at last birthday _____			
	Sex: M [__] F [__]	State of Origin _____	Nationality _____			
	Home address *(house no, street, community, area)*	_____				
	Tribe: Yoruba[__]	Igbo[__]	Hausa[__]	Others (specify)_____		
B.	**PARENTS/GUARDIAN'S SOCIO-DEMOGRAPHICS**					
	Relationship to child: Father _____	Mother _____	Others (sepcify) _____			
	Phone number (11 digits):	[__/__/__/__/__/__/__/__/__/__/__]	[__/__/__/__/__/__/__/__/__/__/__]			
	Father's level of education:	Primary[__]	Secondary[__]	Tertiary[__]	Postgraduate[__]	
	Mother's education:	Primary[__]	Secondary[__]	Tertiary[__]	Postgraduate[__]	
	Father's occupation: _____	Mother's occupation: _____				
C.	**CHILD'S ANTHROPOMETRY**					
	Height (cm) _____ Weight (kg)_____ BMI _____					
D.	**CHILD'S MEDICAL HISTORY**					
	When was child first diagnosed with asthma? *(mmm/yyyy)*	[__ __ __/__ __ __ __]				
	Where was child first diagnosed with asthma?					
	LASUTH[__] General Hospital[__] Private Hospital[__] PHC _____ Other (specify)_____					
	Since when has your child been attending LASUTH clinic? *(mmm/yyyy)*	[__ __ __/__ __ __ __]				
	Last clinic visit: *(dd/mmm/yyyy)*	[__ __/ __ __/__ __ __ __]				
	Last/current Asthma Therapy prescribed at the clinic:	Formulation/device type eg. MDI, DPI, tablet,	Dosage/frequency e.g. 200mcg bd			
	Salbutamol only					
	Salmeterol-Fluticasone					
	Fluticasone					
	Formoterol budesonide					
	Others (specify)					
	Other self-prescribed medications	_____				
	Oral salbutamol					
	Oral prednisolone					
	Others, specify _____					
	Allergic Co-Morbidities in child					
	Atopic dermatitis: Yes[__] No[__]	*If yes, is child on treatment for AD? Yes[__] No[__]*				
	Allergic rhinitis: Yes[__] No[__]	*If yes, is child on treatment for AR? Yes[__] No[__]*				
	Allergic conjunctivitis: Yes[__] No[__]	*If yes, is child on treatment for AC? Yes[__] No[__]*				
	Food allergy: Yes[__] No[__]	*If yes, is child on treatment for FA? Yes[__] No[__]*				
E.	**FAMILY & ENVIRONMENT**					
	Allergies in other family members:					
	Asthma: Yes[__] No[__]	*If yes, who? father[__] mother[__] sibling[__]*				
	Atopic dermatitis: Yes[__] No[__]	*If yes, who? father[__] mother[__] sibling[__]*				

		Allergic rhinitis: Yes[__] No[__]	*If yes, who? father[__] mother[__] sibling[__]*	
		Allergic conjunctivitis: Yes[__] No[__]	*If yes, who? father[__] mother[__] sibling[__]*	
		Food allergy: Yes[__] No[__]	*If yes, who? father[__] mother[__] sibling[__]*	
	Child's position in family: 1st[__] 2nd[__] 3rd[__] 4th[__] 5th[__] 6th[__] 7th[__] >7th[__]			
	Type of apartment:			
	1-2 room tenement[__] 1 bedroom flat[__] 2 BR flat[__] 3BR Flat[__] 4BR Flat/Duplex[__] Others _____			
	Family size (total number of occupants, apart from visitors on short stay) _____			
	Where does child usually sleep? Mat[__] Bare floor with clothe[__] Bare floor Sofa[__] Mattress[__]			
	Which of the following pets or domestic animals do you keep?			
	None[__] Dog[__] Poultry[__] Cat[__] Goats/Rams[__] Others (specify) _____			
F.	**ASSESSMENT OF ASTHMA CONTROL**			
	GINA ASTHMA CONTROL:	Well-controlled [__]	Partly controlled [__]	Uncontrolled [__]
	ASTHMA CONTROL TEST:	ACT Score: _____		
		ACT Category: Well-controlled [__]Poorly controlled [__]Very poorly controlled [__]		
	ASTHMA CONTROL GRADING (COMBINATION OF GINA & ACT):			
		Optimal control (well-controlled with BOTH GINA & ACT Scales) [__]		
		Sub-Optimal control (not well-controlled with EITHER GINA or ACT scale) [__]		
	ASSESSMENT OF FUTURE RISKS OF EXACERBATION			
	Exacerbation: In the last one year how many times had your child been hospitalised for asthma exacerbation (attacks)?			
	Adherence: In the last 4 weeks, on how many days of a week did you forget to use your controller medication (eg seretide)? _____			
	Do you have any concerns or fears about any of your inhalers or medications? Yes[__] No[__] If yes, specify _____			
	Inhaler technique	Number of devices:	0[__] 1[__] 2[__]	
		Device types:	None[__] MDI only[__] MDI + improvised Spacer[__] MDI + manufactured Spacer[__] MDI+Spacer+Facemask[__] DPI (discus) [__]	
		IDAT Score:	IDAT Score: _____	
	Asthma knowledge scores _____			
	Psychosocial burden (Quality of Life score) _____			
G	**HOUSEHOLD AIR POLLUTION PROFILE**			
	Where is cooking done?	Inside room[__] Separate kitchen[__] Others_____		
	Main cooking fuel	Firewood[__] Charcoal/Coal[__] Kerosene[__] Gas/LNG[__] Electric[__] Others (specify) _____		
	Secondary cooking fuel	Firewood[__] Charcoal/Coal[__] Kerosene[__] Gas/LNG[__] Electric[__] Others (specify) _____		
	How frequently do you use this			
	• Mosquito coils	Everyday[__] Most days[__] Sometimes [__] Rarely [__] Never [__]		
	• Incense	Everyday[__] Most days[__] Sometimes [__] Rarely [__] Never [__]		
	• Insecticides	Everyday[__] Most days[__] Sometimes [__] Rarely [__] Never [__]		
	• Air freshner	Everyday[__] Most days[__] Sometimes [__] Rarely [__] Never [__]		
	• Perfumes/deodorants	Everyday[__] Most days[__] Sometimes [__] Rarely [__] Never [__]		
	• Generator	Everyday[__] Most days[__] Sometimes [__] Rarely [__] Never [__]		
	How far is your generator from the room where child sleeps			
		< 2meter[__] 2-4meter[__] 4-6meter[__] 6-8meter[__] 8-10meter[__] 10meter[__]		
H.	**SECOND-HAND SMOKE EXPOSURE**			
	Does anyone smoke in your apartment?	yes[__] no[__]		

		if yes, who? Father[__] Mother[__] others (specify)_____				
	Does anyone smoke outside your apartment but inside your house building? yes[__] no[__]					
		if yes, who? Father[__] Mother[__] others (specify)_____				
	Is your child (are your) exposed to tobacco smoke outside the home (in school, daycare, etc) yes[__] no[__]					
		if yes, specify where _____, _____,				
	Prenatal exposure to tobacco smoke:					
	NB: exposure means being around people who smoke to the extent of perceiving the tobacco odour					
	• Was mother smoking during pregnancy? yes[__] no[__] • Was mother exposed to tobacco smoke during pregnancy? yes[__] no[__]					
I.	**LUNG FUNCTION TEST**					
		FEV_1 (L) PEF (ml/min)	Predicted _____ Predicted _____	%Predicted _____ %Predicted _____		
J.	**URINE COTININE TEST (*AllTest™* cotinine test strips)**					
	Negative[__] 10ng/mL[__] 20ng/mL[__] 50ng/mL[__] 100ng/mL[__] 200ng/mL[__] 500ng/mL[__]					
K.	**HOUSEHOLD AIR QUALITY MEASUREMENTS**					
		Daily average value (mean of 3 days readings)	Proportion of readings above WHO level	Household activity/time associated with peak level		
	$PM_{2.5}$ (ug/m^3)					
	CO (mg/m^3)					
L.	**PERSONAL PEF VARIABILITY**	Day 1	Day 2	Day 3	Average	
	Highest PEF:	_____	_____	_____	_____	
	Lowest PEF:	_____	_____	_____	_____	
	Difference (highest-lowest)	_____	_____	_____	_____	
	Mean [(highest+lowest)/2]	_____	_____	_____	_____	
	PEF Variability [100%(PEF difference/mean PEF)]	_____	_____	_____	_____	

ADDITIONAL FILE 2

PERSONAL PEAK FLOW METER RECOR

NAME _____

RESEARCH ID NO: _____

DAILY HOME PEAK FLOW MEASUREMENT RECORD

		DAY 1	DAY 2	DAY 3	DAY 4	DAY 5
Morning	*Reading 1*					
	Reading 2					
	Reading 3					
Evening	*Reading 1*					
	Reading 2					
	Reading 3					

INSTRUCTION ON HOW TO USE YOUR PEAK FLOW METER: Before using your personal peak flow meter, make sure there is no object inside the device. Attach the mouthpiece. Make sure the slider is at the zero mark. Get ready to breathe very deeply. With your head straight without bending your neck, breathe in deeply to fill your lungs with air until you can no longer do so. Put the mouthpiece in your mouth in-between your teeth and with your lips sealed around it. Make sure the meter is straight, without bending. Blow out the air in your lungs forcefully and suddenly, as fast as you can. Read of the point where the slider is and write on the form as reading 1. Move the slider back to the zero mark and repeat the steps to get reading 2. Repeat to get reading 3. You need at least 3 readings at each time. Repeat any of The reading if you make mistake at any of the steps.

745 **ADDITIONAL FILE 3**

746 **HOUSEHOLD ACTIVITIES DIARY**

747

CHILD'S NAME _____ **PARENT/GUARDIAN'S NAME** _____

HOME ACTIVITIES DIARY

DAY _____ **DATE** _____

INSTRUCTION: Tick (√) activities that occur in the home at the time periods listed

TIME PERIODS	cooking	sweeping	insecticide use	generator use	kerosene lamps/candles/lantern use	mosquito coils use	incense use	others (specify)
4-5 am								
5-6 am								
6-7 am								
7-8 am								
8 -9 am								
9-10 am								
10-11 am								
11-12 noon								
12-1 pm								
1-2 pm								
2-3 pm								
3-4 pm								
4-5 pm								
5-6 pm								
6-7 pm								
7-8 pm								
8-9 pm								
9-10 pm								
10-11 pm								
11-12 midnight								
12-2 am								
2-4am								

Note: Grey-shaded areas refers to times that most children are likely to be home

748