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Asthma as a Type I Hypersensitivity Reaction and Laboratory Diagnosis: A Review of Current Literature

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ABSTRACT: Asthma is a common chronic respiratory disease affecting people worldwide. Globally, over 260 million people were estimated to have asthma in 2019, with an incidence peak in children aged 5–9 years. Prevalence tends to be higher in high-income countries (~6–8%) than in low- and middle-income countries (~2–5%). In Nigeria, national surveys find that 2.5% of people report doctor-diagnosed asthma, while 6.4% have "clinical asthma" (symptoms suggestive of asthma) and 9.0% report wheezing in the past year. Rates rise with age: e.g. 10–17-year-olds have clinical asthma in ~3–10%. In Southeast Nigeria (Enugu State), school-based studies similarly show high prevalence: one recent survey found overall bronchial asthma in ~11.3% of children, with urban pupils (13.1%) slightly more affected than rural (11.2%). Asthma is an IgE-mediated type I hypersensitivity reaction characterized by an initial sensitization to inhaled allergens, followed by allergen challenge that triggers mast cell and basophil degranulation, resulting in bronchoconstriction, mucus hypersecretion, and airway inflammation. Understanding asthma immunopathology is essential for precision therapy. Therefore, a central objective is to precisely phenotype patients so that appropriate biologics can be selected. Standardized biomarkers are critical for this. For instance, serum total and specific IgE (or skin prick) assess atopy, blood eosinophil count and sputum eosinophils measure airway eosinophilia, and FeNO reflects IL-13–driven inflammation. International guidelines endorse such testing: NICE and ATS recommend FeNO for asthma diagnosis and monitoring, and ERS/ATS guidelines highlight sputum eosinophil counts and FeNO to guide therapy in severe casest. In practice, an elevated FeNO (>50 ppb) or blood eosinophils (≥150–300 cells/µL) suggests Type-2-high asthma responsive to steroids and biologics. Indeed, sputum-guided management (treating to maintain eosinophils <3%) reduces exacerbations, although its use is currently limited to specialty centres.

KEWORDS: Asthma, chronic respiratory disease, FeNO, IL-13, IgE-mediated, Type I hypersensitivity, Type-2-high asthma

INTRODUCTION

Asthma is a common chronic respiratory disease affecting people worldwide. Globally, over 260 million people were estimated to have asthma in 2019 (GBD 2019 Risk Factors Collaborators, 2020; Barne, 2023), with an incidence peak in children aged 5–9 years (Kim *et al*., 2025). Prevalence tends to be higher in high-income countries (~6–8%) than in low- and middle-income countries (~2–5%) (Barne, 2023). Indeed, global reviews and WHO data report that 96% of asthma-related deaths occur in LMICs, reflecting under-diagnosis and under-treatment in these regions (Meghji *et al*., 2021). In the WHO African Region, the burden is similarly high: population surveys estimate that roughly 7% of rural and 9–10% of urban dwellers have asthma (largely undiagnosed), and urbanization is consistently linked to higher asthma rates (Adiele *et al*., 2022; GBD 2019 Risk Factors Collaborators, 2020).

In Nigeria, national surveys find that 2.5% of people report doctor-diagnosed asthma, while 6.4% have "clinical asthma" (symptoms suggestive of asthma) and 9.0% report wheezing in the past year (Ozoh *et al*., 2019). Rates rise with age: e.g. 10–17-year-olds have clinical asthma in ~3–10% (Ozoh *et al*., 2019). In Southeast Nigeria (Enugu State), school-based studies similarly show high prevalence: one recent survey found overall bronchial asthma in ~11.3% of children, with urban pupils (13.1%) slightly more affected than rural (11.2%) (Adiele *et al*., 2022). Socioeconomic and environmental factors (e.g. urban living, indoor pollutants, family history) appear to drive these patterns (Adiele *et al*., 2022). Overall, asthma burdens are increasing and under-recognized in Nigeria and Africa. This context underscores the need for reliable, standardized biomarker-based diagnostics to confirm asthma diagnoses and guide management. The next sections review the immunopathology of allergic asthma and the laboratory tests available.

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Asthma is an IgE-mediated type I hypersensitivity reaction characterized by an initial sensitization to inhaled allergens, followed by allergen challenge that triggers mast cell and basophil degranulation, resulting in bronchoconstriction, mucus hypersecretion, and airway inflammation (Sinyor and Concepcion, 2023; Froidure *et al*., 2015). The immunopathology involves a Th2-skewed adaptive response, with IL-4 and IL-13 driving B-cell isotype switching to IgE, which binds high-affinity FcεRI receptors on mast cells and basophils during sensitization (Abbas *et al*., 2023; Feng *et al*., 2024). Upon re-exposure, allergen cross-links IgE on effector cells, causing immediate release of pre-formed mediators (e.g., histamine, tryptase) and newly synthesized lipid mediators (e.g., leukotrienes, prostaglandins) that mediate bronchospasm and vascular permeability (Shamji *et al*., 2021; Michel *et al*., 2023). A late-phase response, occurring 3–12 hours later, is driven by recruited eosinophils, Th2 cells, and basophils, perpetuating airway hyperresponsiveness and remodeling (Feng *et al*., 2024). Laboratory diagnosis combines clinical assessment with lung function tests (spirometry with bronchodilator reversibility), supplemented by immunologic assays such as skin prick testing, serum-specific IgE measurement, fractional exhaled nitric oxide (FeNO), and peripheral eosinophil counts to confirm atopic status and airway inflammation (Murugesan *et al*., 2023; NICE, 2024).

Understanding asthma immunopathology is essential for precision therapy. The recognition of Type-2-high versus low endotypes has led to targeted biologic treatments that improve outcomes for specific subgroups. For example, anti-IgE (omalizumab) benefits severe allergic asthma, and anti–IL-5/IL-5R agents (mepolizumab, reslizumab, benralizumab) are effective in eosinophilic asthma, as does anti–IL-4Rα (dupilumab) in broad Type-2 asthma (Global Initiative for Asthma (GINA), 2023). Global Initiative for Asthma (GINA) explicitly recommends these add-on biologics in severe asthma with corresponding phenotypes (GINA Committee, 2023). Such therapies would not exist without a mechanistic understanding of the underlying endotypes (GINA Committee, 2023; Robinson *et al*., 2017). Conversely, Type-2-low asthma lacks well-defined biologics, underscoring the need for further research. Therefore, a central objective is to precisely phenotype patients so that appropriate biologics can be selected. Standardized biomarkers are critical for this. For instance, serum total and specific IgE (or skin prick) assess atopy, blood eosinophil count and sputum eosinophils measure airway eosinophilia, and FeNO reflects IL-13–driven inflammation. International guidelines endorse such testing: NICE and ATS recommend FeNO for asthma diagnosis and monitoring (Robinson *et al*., 2017) and ERS/ATS guidelines highlight sputum eosinophil counts and FeNO to guide therapy in severe cases (Chung *et al*., 2014). In practice, an elevated FeNO (>50 ppb) or blood eosinophils (≥150–300 cells/μL) suggests Type-2-high asthma responsive to steroids and biologics (Menzies-Gow *et al*., 2020). Indeed, sputum-guided management (treating to maintain eosinophils <3%) reduces exacerbations (GINA committee, 2023), although its use is currently limited to specialty centres. Overall, advances in asthma care hinge on bridging immunologic insight with diagnostics: by systematically measuring IgE, FeNO, and eosinophils, clinicians can classify asthma endotypes and tailor biologic or targeted therapies to each patient (GINA Committee, 2023; Robinson *et al*., 2017).

This review study aims to critically examine asthma as an IgE-mediated (Type I) hypersensitivity reaction, focusing on Th2-driven immunopathology and evaluating standardized laboratory diagnostics (SPT, sIgE assays, FeNO, induced sputum eosinophils). The examination will be informed by global and Southeast Nigerian epidemiology to enable precise endotype-guided management in accordance with GINA recommendations.

Asthma Overview

Asthma is a heterogeneous chronic inflammatory disease of the airways, characterized by episodic respiratory symptoms (wheeze, breathlessness, chest tightness, cough) and variable airflow obstruction (Luo *et al*., 2022; Simpson and Price, 2023). It affects all ages worldwide, with recent estimates ranging from ~262 million people in 2019 (WHO) to >350 million in other reports (Menzies-Gow *et al*., 2020; Vos *et al*., 2020). Asthma causes substantial morbidity and mortality: WHO reported ~455,000 deaths in 2019 (Vos *et al*., 2020), and a global analysis found ~457,000 asthma deaths in 2017 (Cao *et al*., 2022). Global prevalence varies by region: one analysis estimated 3.33% worldwide (2017), while continental rates range from ~3–4% in Asia and Africa to ~8% in North America and Oceania (Cao *et al*., 2022; Rabe *et al*., 2023). Notably, adolescent prevalence (~11%) exceeds that in children or adults, and asthma burden is generally higher in high-income countries (Rabe *et al*., 2023). However, most asthma deaths occur in low- and middle-income countries (Vos *et al*., 2020). These data underscore asthma as a major global public-health problem with regional variation in prevalence and outcomes. In Nigeria, national surveys find that 2.5% of people report doctor-diagnosed asthma, while 6.4% have “clinical asthma” (symptoms suggestive of asthma) and 9.0% report wheezing in the past year (Ozoh *et al*., 2019). Rates rise with age: e.g. 10–17-year-olds have clinical asthma in ~3–10% (Ozoh *et al*., 2019). In Southeast Nigeria (Enugu State),

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school-based studies similarly show high prevalence: one recent survey found overall bronchial asthma in ~11.3% of children, with urban pupils (13.1%) slightly more affected than rural (11.2%; Adiele *et al*., 2022).

Asthma is also clinically heterogeneous. Distinct clinical phenotypes have been identified, often reflecting differences in age of onset, triggers, and inflammation. For example, allergic asthma (usually childhood onset, atopic, eosinophilic, steroid-responsive) and non-allergic asthma (often adult-onset, variable eosinophilic/neutrophilic inflammation, less ICS-responsive) represent major phenotypes (GINA Committee, 2023). Other recognized phenotypes include late-onset asthma (new-onset in adults, often female, less atopic, higher treatment needs), asthma with persistent airflow limitation (fixed obstruction from airway remodelling), and obesity-associated asthma (severe symptoms with minimal eosinophilia). These phenotypes demonstrate that asthma symptoms and triggers cluster in identifiable patterns (GINA Committee, 2023). Underlying these clinical phenotypes are endotypes – mechanistic subtypes defined by immunopathology (Robinson *et al*., 2017). A widely used endotype classification contrasts Type-2-high versus Type-2-low inflammation. Type-2-high (Th2-high) asthma involves helper T-cell type 2 (Th2) and innate lymphoid 2 (ILC2) pathways, with signature cytokines IL-4, IL-5, and IL-13 (Robinson *et al*., 2017). This endotype is associated with eosinophilic airway inflammation, elevated exhaled nitric oxide (FeNO), high IgE levels, and often allergic sensitization (GINA Committee, 2023; Robinson *et al*., 2017). In contrast, Type-2-low (non–Type-2) asthma includes neutrophilic or paucigranulocytic patterns driven by Th1/Th17 and innate responses, and accounts for ~20–30% of patients (Peri *et al*., 2023). Type-2-low asthma tends to be non-atopic and less steroid-responsive. Importantly, identifying Type-2-high versus low endotypes guides therapy: GINA notes Type-2 inflammation is found in the majority of severe asthma and correlates with eosinophilia/atopy, whereas Type-2-low patients often need alternative approaches. Overall, modern asthma classification integrates observable phenotypes (e.g. allergic vs obese asthma) with molecular endotypes (Type-2-high vs low) to capture the disease's heterogeneity (GINA Committee, 2023; Robinson *et al*., 2017).

Immunopathology of Type I Hypersensitivity in Asthma

Asthma caused by allergies is a classic Type I (IgE-mediated) hypersensitivity process. The sensitization (priming) and effector (response) phases involve several immune cell types and mediators:

Antigen Presentation and Th2 Polarization: In sensitization, inhaled allergens (e.g. pollen, dust-mite proteins) are captured by airway dendritic cells (DCs). These DCs migrate to regional lymph nodes and present allergen peptides to naïve CD4+ T cells (Vroman *et al*., 2017). In atopic individuals, cytokines in the environment (notably IL-4, often from innate cells or basophils) drive these naïve T cells to differentiate into Th2-type helper T cells (Ogulur *et al*., 2025). Other influences, such as epithelial "alarmins" like IL-33 and TSLP, also bias toward Th2; innate lymphoid type 2 cells (ILC2s) can produce Th2 cytokines independent of DCs.) Th2 cells secrete a characteristic profile of cytokines – primarily IL-4, IL-5, IL-9, and IL-13 – which orchestrate allergic immunity (Ogulur *et al*., 2025).

IgE Class Switching: IL-4 (together with IL-13) stimulates B cells to undergo immunoglobulin class switching from IgM to IgE (Ogulur *et al*., 2025). The B cells produce allergen-specific IgE antibodies that enter the bloodstream. These IgE molecules bind with high affinity to FcεRI receptors on mast cells and basophils, "arming" them for an allergic response (Ogulur *et al*., 2025). This completes the sensitization phase: the host carries memory of the allergen via antigen-specific IgE bound to effector cells.

Effector (Immediate) Phase: Upon re-exposure to the allergen, the allergen cross-links the IgE on mast cells and basophils. This triggers immediate degranulation of mast cells, releasing histamine, proteases (e.g. tryptase), heparin, and pre-formed mediators, as well as generating lipid mediators (cysteinyl leukotrienes CysLTs, prostaglandin D2). Histamine and leukotrienes cause acute bronchoconstriction, increased vascular permeability (leading to oedema), and mucus secretion in the airways. Clinically this causes wheezing, coughing, and chest tightness within minutes of allergen contact. These mediators also recruit additional cells. Notably, IL-5 secreted by Th2 (and ILC2) cells promotes growth and activation of eosinophils (Ogulur *et al*., 2025). Thus, a late-phase inflammatory response ensues over hours.

Late (Eosinophilic) Phase: Hours after the initial reaction, there is influx of eosinophils, basophils, and Th2 cells into the bronchial mucosa. Eosinophils release cytotoxic proteins (major basic protein, eosinophil cationic protein) and additional cytokines (IL-5, IL-13) that sustain airway inflammation and cause tissue damage/remodelling. This contributes to prolonged airway hyperresponsiveness and chronic changes. IL-13 from Th2 and ILC2 cells promotes goblet cell hyperplasia and mucus hypersecretion, while IL-4/IL-13 maintains high IgE production (Ogulur *et al*., 2025). Thus, the cytokine milieu perpetuates asthma symptoms.

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ILC2 and Innate Factors: Besides Th2 cells, group 2 innate lymphoid cells (ILC2s) play a key role. ILC2s, found in airway tissues, are triggered by epithelial "alarmins" (IL-33, IL-25, TSLP) to secrete large amounts of IL-5 and IL-13. In some studies, ILC2s produced more IL-5 than Th2 cells did, driving intense eosinophilia (Matsuda *et al*., 2022). Thus, ILC2s amplify allergic inflammation and airway hyperreactivity even without T-cell activation.

Th17 and Treg Cells: Emerging evidence shows that Th17 cells (producing IL-17A/F) contribute in some asthma phenotypes, especially severe or neutrophilic asthma. Patients with Th17-mediated asthma often have steroid-refractory airway neutrophilia (Lopes *et al*., 2022). An imbalance of Th17 vs T regulatory cells (Tregs) can worsen asthma. Tregs (via IL-10, TGF-β, IL-35) normally suppress Th2/Th17 responses; immunotherapy that increases Treg activity tends to improve asthma control (Lopes *et al*., 2022; Matsuda *et al*., 2022).

Altogether, type I hypersensitivity in asthma is driven by a Th2/ILC2 cytokine profile (IL-4, IL-5, IL-13) that causes IgE-mediated mast cell activation and eosinophilic airway inflammation (Matsuda *et al*., 2022; Ogulur *et al*., 2025). Acute bronchospasm is mediated by histamine and leukotrienes, and late-phase reactions by eosinophils and Th2 cytokines. Regulatory mechanisms (Tregs, IL-10) normally restrain these responses (Ogulur *et al*., 2025) but when dysregulated they underlie asthma pathogenesis.

Laboratory Diagnosis of Asthma

Since asthma is clinically heterogenous, objective biomarkers can improve diagnosis and phenotyping. The following tests are standard for IgE-mediated asthma:

Skin Prick Testing (SPT)

Principle: The patient's forearm (or back) is pricked through drops of standardized allergen extracts. Each allergen sample is a solution containing a protein extract (e.g. dust mite, pollen) (Bousquet *et al*., 2012). A positive result is a localized wheal (swelling) and flare (redness) at the site after 15 minutes.

Patient Preparation: Patients must stop antihistamines for several days prior. The skin should be free of lotions. Procedure: A drop of each allergen extract is placed on the skin. Using a single-use lancet, each drop is gently pricked into the superficial dermis. Sites are spaced ~2 cm apart. A positive control (histamine, ~10 mg/mL) and a negative control (saline diluent) are included (Bousquet *et al*., 2012). After 15 minutes, the wheal diameter is measured. A wheal diameter ≥3 mm greater than the negative control is usually considered positive.

Interpretation: The size of the wheal correlates with the degree of IgE sensitization. For example, a negative control ≥3 mm necessitates that only wheals >6 mm count as positive (Bousquet *et al*., 2012). A positive histamine control (wheal ~6 mm) confirms test validity; a flat histamine response suggests residual antihistamines or mast cell dysfunction (Bousquet *et al*., 2012).

Quality Control: Use of standardized allergen extracts (validated potency) is crucial. Testing should be done by trained personnel; results are recorded on a chart immediately. Regular calibration of lancets and periodic proficiency assessments (e.g. interobserver checks) are recommended. Overall, SPT has good sensitivity/specificity for IgE allergy when performed properly (Bousquet *et al*., 2012).

Serum-Specific IgE Testing (ImmunoCAP)

Principle: ImmunoCAP (Thermo Fisher) is a quantitative fluoroenzyme immunoassay for serum IgE. Each test uses a solid-phase allergen (purified or mixed) on a cellulose sponge (the "CAP"). The patient's serum is incubated so specific IgE binds the allergen. After washing, enzyme-labelled anti-IgE is added, producing a fluorescent signal proportional to the IgE bound. The instrument (e.g. Phadia 2500) computes IgE concentration (kUA/L) against a WHO-traceable IgE standard (Strip and Anti-IgE, 2025).

Procedure: Venous blood is drawn, serum separated, and 40 µL is incubated with allergen CAP. The automated system (Phadia) carries out all steps with built-in calibrations (Strip and Anti-IgE, 2025).

Interpretation: Results ≥0.35 kUA/L are conventionally taken as positive for sensitization. Lower bound of quantitation is ~0.1 kUA/L, so values <0.1 are considered negative. Laboratories should establish their own reference ranges, but multicenter studies show 0.35 kUA/L yields ~85–95% sensitivity/specificity for various allergens (Strip and Anti-IgE, 2025).

Quality Control: Each run includes calibrators (a multi-point curve) and controls (low/mid/high IgE sera). Inter-assay CV is typically <5% for moderate ranges (Strip and Anti-IgE, 2025). External quality assessment (e.g. through proficiency programs or EQAS) is recommended to ensure consistency. The ImmunoCAP system is regarded as a "gold standard" for in vitro IgE testing (Johansson, 2004; Strip and Anti-IgE, 2025).

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Fractional Exhaled Nitric Oxide (FeNO)

Principle: Airway epithelial cells produce nitric oxide (NO) at higher levels in Th2/eosinophilic inflammation. FeNO measurement is a noninvasive marker of Type 2 airway inflammation. It is now standardized by guidelines (Dweik *et al*., 2011).

Procedure: The patient exhales slowly at a constant flow (50 mL/s) into a NO analyser, following ATS/ERS guidelines. Typically, three reproducible exhalations (10 seconds each) are performed.

Interpretation: FeNO is reported in parts per billion (ppb). Interpretation is stratified: <25 ppb in adults (or <20 ppb in children) is considered low – eosinophilic inflammation is unlikely, and steroid response is less likely (Dweik *et al*., 2011). >50 ppb (adults) or >35 ppb (children) is high, indicating active eosinophilic inflammation and probable steroid responsiveness. Values in the intermediate range (25–50 ppb adults) must be interpreted in clinical context. FeNO correlates well with sputum eosinophilia and predicts inhaled corticosteroid benefit. According to ATS, FeNO should be used in asthma management to identify eosinophilic inflammation and guide treatment (Dweik *et al*., 2011).

Quality Control: The device is zeroed and standardized per manufacturer instructions. Exhalations must meet flow/volume criteria. ATS/ERS protocols specify calibration and quality checks. FeNO adds objective data, but can be affected by smoking and atopy, so results should be integrated with clinical findings.

Induced Sputum Eosinophils

Principle: Inducing sputum via inhaled saline allows direct sampling of airway inflammatory cells. A hypertonic (3–5%) saline aerosol is inhaled in 3-minute increments (total ~15–20 min), after bronchodilator pre-treatment (e.g. salbutamol) to prevent bronchospasm. The patient coughs and expectorates sputum into a container.

Laboratory Processing: Sputum plugs are selected (discard saliva), weighed, and treated with dithiothreitol (DTT) to dissolve mucus. After filtration, cells are spun onto slides for cytospin. Slides are stained (e.g. May-Grünwald-Giemsa). At least 300 non-squamous cells are counted to determine differential cell percentages (eosinophils, neutrophils, macrophages, lymphocytes).

Interpretation: Elevated sputum eosinophil fraction (commonly >2–3%) indicates eosinophilic airway inflammation typical of allergic asthma. Conversely, neutrophil-predominant sputum can indicate other asthma phenotypes or infection. Induced sputum is the reference standard for airway eosinophilia assessment.

Quality Control: Laboratorial protocol requires consistency. DTT neutralization, viability checks (>50% viable cells), and duplicate counts help ensure accuracy. A reference laboratory or proficiency scheme is ideal to harmonize sputum processing.

Component-Resolved Diagnostics (CRD) and Multiplex Assays: Recent advances use purified allergen molecules (components) on multiplex platforms. For example, the ImmunoCAP ISAC is a microarray chip containing ~100 allergen components. A few drops of serum are incubated on the chip, and specific IgE binding is detected via fluorescence.

Strengths: CRD can profile sensitization to many individual allergens at once, aiding precise allergen identification and cross-reactivity analysis (Keshavarz *et al*., 2021). Only a small volume of serum is needed (Keshavarz *et al*., 2021).

Limitations: These arrays have variable sensitivity for each allergen and are costly. Interpretation of complex results (often dozens of positives) requires specialist input (Keshavarz *et al*., 2021). Nevertheless, multiplex CRD is increasingly used in research and specialized clinics.

In practice, asthma diagnosis often combines history, pulmonary function, and one or more biomarker tests. For allergic asthma, a combination of SPT or specific-IgE plus FeNO or eosinophils provides objective evidence of atopy and Type 2 inflammation. Newer molecular diagnostics (CRD) can refine allergen identification. Each lab test requires rigorous SOPs (as above) and quality assurance to ensure reliability in any setting, including resource-limited regions.

CONCLUSION

Asthma imposes a significant health and economic burden globally and especially in Africa. Over 260 million people currently have asthma, and nearly half a million die yearly, mostly in LMICs due to under-diagnosis and under-treatment. In Nigeria, asthma symptoms are common (clinical asthma ~6–9%, higher in children), but lack of diagnostic infrastructure means many cases go unrecognized until severe. This epidemiologic picture underscores an urgent need for improved diagnostic tools. Standardized biomarker tests (IgE assays, FeNO, sputum eosinophils) can provide objective confirmation of asthma and guide therapy, but they are underused in Africa.

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In under-resourced areas like Southeast Nigeria, significant challenges exist. Laboratories may lack immunoassay analysers, quality reagents, and trained technicians. Even SPT supplies or reading expertise can be limited. There is also low awareness of allergy testing among healthcare providers. As Barne et al. note, LMICs face gaps in infrastructure, training and quality assurance that perpetuate poor asthma outcomes. Overcoming these gaps requires investment in basic laboratory capacity (electricity, equipment maintenance, reagent supply chains) and specialized training in allergy diagnostics.

Looking forward, several strategies could bolster asthma care in settings like Nigeria. First, expanding component-resolved diagnostics (CRD) and multiplex allergy testing could improve efficiency: a single multiplex assay might replace many individual tests, conserving scarce blood and reagents. Developing low-cost allergy panels tailored to local allergens would make CRD more practical. Secondly, implementing quality assurance (QA) networks is vital. Participating in international External Quality Assessment (EQA) schemes (e.g. WHO-AFRO or SLIPTA programs) and inter-laboratory comparisons would ensure test accuracy. National reference laboratories could oversee proficiency testing for allergy assays. Thirdly, capacity-building is key. Regional training centres and online courses can equip laboratory staff and with skills in SPT technique, spirometry, FeNO measurement, and laboratory immunology. For example, Australasian and European allergy societies provide freely accessible SOPs and training materials for tests like SPT. Adopting such protocols locally (with appropriate validation) can raise standards. Mobile health (mHealth) and telemedicine might support remote areas in reading SPT or FeNO results through expert networks.

In conclusion, integrating the epidemiologic understanding of asthma's burden with robust diagnostic methods is essential. Asthma in Nigeria and across Africa remains under-recognized; bridging this gap means deploying standardized biomarkers alongside clinical criteria. By improving lab infrastructure, expanding CRD and QA efforts, and investing in human capacity, we can move toward timely and accurate diagnosis. This will enable better-targeted therapy (e.g. anti-IgE or anti–IL-5 in severe cases) and ultimately reduce asthma morbidity and mortality. Such steps are in line with WHO's call for improved asthma diagnosis and management towards universal health coverage.

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