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Is Allergic Rhinitis Associated with Increased Severity Of Coexisting Asthma In Children And Adults? : A Comprehensive Systematic Review

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ABSTRACT

Introduction: Allergic rhinitis (AR) and asthma frequently coexist as manifestations of the unified allergic airway disease, yet the precise relationship between AR and asthma severity remains incompletely characterized across different age groups. This systematic review aimed to synthesize current evidence on whether AR is associated with increased severity of coexisting asthma in children and adults.

Methods: A systematic review was conducted following PRISMA guidelines. Studies were screened based on predefined criteria including diagnosed asthma population, AR assessment using validated methods, measurement of asthma severity through clinical parameters, differentiation between allergic and non-allergic rhinitis, and appropriate observational or interventional study designs. Data extraction encompassed study characteristics,

population demographics, AR diagnostic criteria, asthma severity measures, association results, age group effects, and effect modifiers.

Results: Eighty studies published between 2002-2025 met inclusion criteria, comprising cross-sectional studies (n=62), cohort studies (n=12), systematic reviews (n=2), randomized controlled trials (n=2), and case-control studies (n=2). AR prevalence in asthmatic populations ranged from 29.2% to 97.5% across studies. The majority of studies (72/80, 90%) demonstrated a positive association between AR and increased asthma severity, manifested through poorer asthma control (OR range 1.21-2.74), more frequent exacerbations (incidence rate ratio 1.12), increased healthcare utilization (OR 2.64-2.98 for emergency visits), and impaired lung function (lower FEV_1, $FEF_{25–75}$). AR severity correlated positively with asthma severity (correlation coefficients 0.365-0.689), with persistent and moderate-to-severe AR phenotypes consistently associated with difficult-to-control asthma. Treatment of AR, particularly with intranasal corticosteroids, was associated with improved asthma outcomes. Age-specific effects included stronger associations in school-age children compared to younger children (<6 years), and attenuation of prevalence in older adults.

Discussion: The consistent positive association between AR and asthma severity across diverse populations supports the unified airway concept and has important clinical implications. The dose-response relationship between AR severity and asthma severity, coupled with improved asthma outcomes following AR treatment, suggests potential causal mechanisms including naso-bronchial reflex, systemic eosinophilic inflammation, and shared type-2 inflammatory pathways. However, heterogeneity in AR

assessment methods and asthma severity definitions across studies limits direct comparability.

Conclusion: Compelling evidence demonstrates that AR is associated with increased severity of coexisting asthma in both children and adults, with AR severity, persistence, and specific phenotypes serving as important determinants. Systematic assessment and optimal management of AR should be integrated into asthma care to potentially improve asthma outcomes.

Keywords: Allergic rhinitis, asthma, asthma severity, asthma control, united airway disease, systematic review

INTRODUCTION

Background

Allergic rhinitis (AR) and asthma represent two of the most prevalent chronic respiratory conditions worldwide, affecting approximately 400 million and 300 million individuals respectively across all age groups (1,2). The concept of "united airways disease" has gained substantial traction over recent decades, recognizing that the upper and lower airways share common embryological origins, similar mucosal histology, and interconnected pathophysiological mechanisms (3). Epidemiological studies consistently demonstrate that AR and asthma frequently coexist, with AR present in 55-97% of patients with asthma and asthma occurring in 20-40% of patients with AR (1,4,5). This frequent comorbidity extends beyond mere coincidence, reflecting shared genetic predisposition, similar environmental triggers, and parallel inflammatory pathways characterized by type-2 immune responses involving eosinophils, mast cells, and T-helper 2 lymphocytes (6,7).

The clinical significance of the AR-asthma relationship extends to disease severity, healthcare utilization, quality of life, and economic burden. Patients with concomitant AR and asthma experience greater symptom burden, more frequent exacerbations, increased emergency department visits, higher medication requirements, and poorer quality of life compared to those with asthma alone (8-10). The Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines have long emphasized the importance of evaluating and managing AR in all patients with asthma, recommending an integrated approach to airway disease management (11). Despite these recommendations, AR remains underdiagnosed and undertreated in asthmatic populations, potentially contributing to suboptimal asthma control and avoidable morbidity (12,13).

The relationship between AR and asthma severity has been investigated through multiple methodological approaches, including cross-sectional surveys examining concurrent disease expression, cohort studies tracking disease progression over time, and interventional trials assessing whether AR treatment improves asthma outcomes (14-16). Cross-sectional studies have consistently documented higher prevalence of AR among patients with more severe asthma, with correlation

coefficients ranging from 0.365 to 0.689 between AR severity scores and asthma severity parameters (6,17). Longitudinal studies have further demonstrated that childhood AR predicts incident asthma development and persistence of asthma into adulthood, with odds ratios ranging from 2.0 to 7.0 depending on AR phenotype and severity (18,19).

The pathophysiological mechanisms linking AR and asthma severity are multifaceted and interconnected. The naso-bronchial reflex theory proposes that nasal irritation triggers bronchoconstriction through neural reflex arcs involving the trigeminal and vagus nerves (20). Post-nasal drip of inflammatory mediators from the upper to lower airways may directly contribute to bronchial inflammation (21). Systemic absorption of inflammatory mediators from the nasal mucosa can propagate type-2 inflammation throughout the respiratory tract, evidenced by elevated peripheral blood eosinophils, total IgE, and fractional exhaled nitric oxide (FeNO) in patients with AR and asthma compared to asthma alone (22,23). Furthermore, mouth breathing secondary to nasal obstruction bypasses the physiological functions of the nose in warming, humidifying, and filtering inspired air, exposing lower airways to unconditioned air and potential irritants (24).

Research Gap

Despite the substantial body of literature examining the AR-asthma relationship, several critical knowledge gaps persist that limit translation of this evidence into clinical practice. First, significant heterogeneity exists in the operational definitions of AR and asthma severity across studies, with some investigations relying on physician diagnosis without objective confirmation, others using validated questionnaires, and still others incorporating objective measures such as specific IgE testing or skin prick testing (25). This variability complicates cross-study comparisons and meta-analytic synthesis of effect sizes.

Second, the differentiation between allergic and non-allergic rhinitis in asthmatic populations remains inadequately addressed in many studies, despite evidence suggesting that allergic and non-allergic rhinitis may have different implications for asthma outcomes (26,27). The distinction is clinically important because non-allergic rhinitis may not respond to allergen-specific interventions and may have different underlying mechanisms linking it to asthma severity.

Third, age-specific effects in the AR-asthma severity relationship remain incompletely characterized. Children, adolescents, adults, and elderly populations may exhibit different patterns of association due to developmental changes in immune function, duration of disease, cumulative allergen exposure, and age-related changes in airway physiology (28,29). Some studies suggest that the association may be stronger in school-age children compared to younger children or older adults, but systematic evaluation across the lifespan is lacking (17,30).

Fourth, the bidirectional nature of the AR-asthma relationship requires further elucidation. While AR is often considered a risk factor for asthma development and severity, evidence also suggests that asthma may influence AR expression, with studies documenting more severe and persistent AR in patients with concomitant asthma compared to AR alone (31,32). Understanding the directionality and potential reciprocal interactions has implications for treatment prioritization and disease monitoring.

Fifth, the role of AR treatment in modifying asthma outcomes requires further rigorous investigation. While observational studies consistently demonstrate that AR treatment, particularly intranasal corticosteroids, is associated with improved asthma control, randomized controlled trials have yielded mixed results, potentially due to heterogeneity in patient selection, treatment protocols, outcome measures, and duration of follow-up (33,34). The optimal AR treatment regimen for improving asthma outcomes, including choice of medications, treatment intensity, and duration, remains to be established.

Sixth, geographic and ethnic variations in the AR-asthma severity relationship remain underexplored. Most published studies originate from Europe, North America, and East Asia, with limited representation from Africa, Latin America, South Asia, and the Middle East (35,36). Given variations in allergen exposure patterns, genetic backgrounds, healthcare systems, and environmental factors across regions, the generalizability of findings from well-studied populations to underrepresented regions cannot be assumed.

Seventh, mechanistic studies linking AR to asthma severity through specific inflammatory pathways, particularly type-2 biomarkers, have yielded inconsistent results. While some studies demonstrate clear associations between AR and elevated FeNO, blood eosinophils, and periostin

levels, others fail to find such relationships, suggesting that multiple endotypes may exist within the AR-asthma population with different implications for disease severity and treatment response (22,37).

Research Objectives

This comprehensive systematic review aimed to:

1. Synthesize and critically appraise the current evidence on whether allergic rhinitis is associated with increased severity of coexisting asthma in children and adults
2. Quantify the prevalence of AR in asthmatic populations across different age groups and geographic regions
3. Characterize the association between AR severity, phenotype, and duration with specific asthma severity parameters including symptom control, exacerbation frequency, lung function, and healthcare utilization
4. Compare and contrast the AR-asthma severity relationship between pediatric and adult populations, identifying age-specific patterns and effect modifiers
5. Evaluate evidence for the bidirectional relationship between AR and asthma, including whether AR treatment improves asthma outcomes and whether asthma influences AR expression
6. Identify knowledge gaps and methodological limitations in the current literature to inform future research priorities
7. Provide evidence-based recommendations for clinical practice regarding the assessment and management of AR in patients with asthma to potentially improve asthma outcomes

Research Benefits

This systematic review offers multiple benefits for various stakeholders. For clinicians, including primary care physicians, pulmonologists, allergists, and otorhinolaryngologists, this

review provides a comprehensive synthesis of evidence to inform clinical decision-making regarding AR assessment and management in patients with asthma. The identification of AR phenotypes and severities most strongly associated with poor asthma outcomes may enable targeted screening and treatment prioritization. For patients with AR and asthma, this review may increase awareness of the interconnected nature of their conditions and the importance of comprehensive airway management. For healthcare policymakers and guideline developers, this synthesis provides the evidence base needed to strengthen recommendations for integrated AR-asthma management in clinical practice guidelines. For researchers, this review identifies critical knowledge gaps and methodologically rigorous approaches for future investigations, including recommendations for study design, outcome measurement, and population selection. For healthcare systems, improved recognition and management of AR in asthmatic populations may reduce asthma-related morbidity, healthcare utilization, and associated costs through more effective comprehensive airway disease management.

Hypothesis

Based on the unified airway concept and previous epidemiological evidence, we hypothesized that allergic rhinitis is positively associated with increased severity of coexisting asthma in both children and adults. Specifically, we hypothesized that: (1) patients with AR-asthma comorbidity demonstrate greater asthma severity across multiple domains (symptom control, exacerbations, lung function impairment, healthcare utilization) compared to asthma patients without AR; (2) there is a dose-response relationship wherein more severe, persistent, and poorly controlled AR is associated with incrementally greater asthma severity; (3) the association between AR and asthma severity differs across age groups, with stronger associations in children and younger adults compared to elderly populations; (4) treatment of AR, particularly with intranasal corticosteroids, is associated with improved asthma outcomes; and (5) the association between AR and asthma severity is mediated, at least in part, by shared type-2 inflammatory mechanisms reflected in elevated biomarkers including FeNO, blood eosinophils, and serum IgE.

Research Gap and Novelty

This systematic review addresses several critical gaps in the current literature while offering novel contributions. Previous reviews have either focused narrowly on specific age groups (exclusively pediatric or adult populations), limited geographic regions, or specific aspects of the AR-asthma relationship such as prevalence or treatment effects without comprehensive examination of severity associations. This review provides the most comprehensive synthesis to date, encompassing 80 studies published over 23 years (2002-2025) across diverse geographic regions, age groups, and study designs. The inclusion of recent 2024-2025 publications ensures incorporation of the most contemporary evidence. The systematic extraction of age-specific effects enables comparative analysis of the AR-asthma severity relationship across the lifespan, from early childhood through elderly populations. The detailed characterization of AR phenotypes, including differentiation between intermittent and persistent AR, mild versus moderate-severe AR, and allergic versus non-allergic rhinitis, provides nuanced understanding of which AR subtypes confer greatest asthma severity risk. The comprehensive evaluation of effect modifiers, including treatment status, sensitization patterns, comorbidities, and environmental factors, offers insights into potential mechanisms and intervention targets. The evidence map visually synthesizing key findings across studies facilitates rapid identification of consistent patterns and outliers requiring further investigation. Finally, the detailed recommendations for future research, including specific study designs, sample size calculations, outcome measures, and populations, provide a roadmap for advancing knowledge in this clinically important area.

METHODS

Protocol

The study strictly adhered to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 guidelines to ensure methodological rigor and accuracy. This approach was chosen to enhance the precision and reliability of the conclusions drawn from the investigation.

Criteria for Eligibility

This systematic review aims to evaluate Is Allergic Rhinitis Associated with Increased

Severity Of Coexisting Asthma In Children And Adults?

Screening

We screened in sources based on their abstracts that met these criteria:

- **Asthma Population**: Does the study include participants with diagnosed asthma?
- **Allergic Rhinitis Assessment**: Does the study assess the presence or absence of allergic rhinitis in participants?
- **Asthma Severity Measurement**: Does the study measure asthma severity using validated tools or clinical parameters (e.g., symptom scores, lung function, medication use, exacerbation frequency)?
- **Sufficient Data for Analysis**: Does the study provide sufficient data to assess the relationship between allergic rhinitis and asthma severity?
- **Appropriate Study Design**: Is the study design one of the following: observational study (cross-sectional, cohort, case-control), randomized controlled trial, systematic review, or meta-analysis?
- **Allergic vs Non-allergic Rhinitis Differentiation**: Does the study differentiate between allergic and non-allergic rhinitis (rather than treating all rhinitis as one condition)?
- **Study Design Quality**: Is the study design something other than a case report or case series?
- **Baseline Association Data**: If the study focuses on treatment interventions, does it also report baseline associations between allergic rhinitis and asthma severity (or is the study not exclusively focused on treatment interventions)?

We considered all screening questions together and made a holistic judgement about whether to screen in each paper.

Search Strategy

The keywords used for this research based PICO :

Element	P (Population)	I (Intervention/Exposure)	C (Comparison/Context)	O (Outcome)
Keyword 1	Asthma	Allergic Rhinitis	Non-Allergic Rhinitis	Asthma Severity
Keyword 2	Bronchial Asthma	Seasonal Allergies	Non-Atopic Rhinitis	Exacerbation Frequency
Keyword 3	Reactive Airway Disease	Nasal Allergies	No Rhinitis	Lung Function Decline
Keyword 4	Coexisting	Atopic Rhinitis	Without Nasal Allergies	Uncontrolled Asthma

The Boolean MeSH keywords inputted on databases for this research are: *("Asthma" OR "Bronchial Asthma" OR "Reactive Airway Disease" OR "Coexisting") AND ("Allergic Rhinitis" OR "Seasonal Allergies" OR "Nasal Allergies" OR "Atopic Rhinitis") AND ("Non-Allergic Rhinitis" OR "Non-Atopic Rhinitis" OR "No Rhinitis" OR "Without Nasal Allergies") AND ("Asthma Severity" OR "Exacerbation Frequency" OR "Lung Function Decline" OR "Uncontrolled Asthma")*

Data extraction

- **Study Design**:

  Extract study design (cross-sectional, cohort, case-control, etc.), setting, country, and study period. Include sample size and whether the study specifically examined patients with coexisting allergic rhinitis and asthma or looked at the development of one condition in those with the other.

- **Population Characteristics**:

Extract participant demographics and inclusion criteria relevant to the allergic rhinitis-asthma association, including:

- Age groups studied (children, adults, or both - with specific age ranges)
- Sample size with both allergic rhinitis and asthma
- Inclusion/exclusion criteria related to having both conditions
- Key baseline characteristics (sex distribution, atopy status, family history)

- **Allergic Rhinitis Assessment**:

Extract how allergic rhinitis was defined, diagnosed, and characterized in relation to asthma severity, including:

- Diagnostic criteria or definition used
- Severity classification (mild, moderate-severe, intermittent, persistent)
- Duration of allergic rhinitis symptoms
- Treatment status (treated vs untreated, type of treatment)
- Sensitization patterns if reported (specific allergens, polysensitization)

- **Asthma Severity Measures**:

Extract all measures used to assess asthma severity or control in patients with coexisting allergic rhinitis, including:

- Severity classification systems used (GINA, physician-assessed, symptom-based)
- Specific outcome measures (asthma control questionnaires, symptom scores, exacerbation rates)

- Lung function parameters (FEV1, peak flow, FeNO)
- Healthcare utilization (hospitalizations, emergency visits, medication use)
- How severity/control was categorized or scored

- **Association Results**:

  Extract the main findings regarding whether and how allergic rhinitis is associated with increased asthma severity, including:

  - Direction and magnitude of association (odds ratios, risk ratios, mean differences with confidence intervals)
  - Statistical significance (p-values)
  - Specific comparisons made (AR vs no AR, AR severity levels, treated vs untreated AR)
  - Dose-response relationships if examined
  - Whether the association was bidirectional (AR affecting asthma AND asthma affecting AR)

- **Age Group Effects**:

  Extract any findings specific to different age groups regarding the allergic rhinitis-asthma severity association, including:

  - Separate results for children vs adults if provided
  - Age-specific effect sizes or risk estimates
  - Differences in the strength or nature of association across age groups

- Age as an effect modifier in the analysis
- Longitudinal changes from childhood to adulthood if studied

- **Effect Modifiers**:

  Extract factors that modified or influenced the association between allergic rhinitis and asthma severity, including:

  - Treatment effects (how AR treatment affected asthma outcomes)
  - Sensitization patterns (mono- vs polysensitization effects)
  - Comorbidities (sinusitis, GERD, other allergic conditions)
  - Environmental factors (allergen exposure, pollution)
  - Patient characteristics that strengthened or weakened the association

- **Confounders Controlled**:

  Extract what confounding variables were controlled for in analyses of the allergic rhinitis-asthma severity association, including:

  - Demographic factors (age, sex, socioeconomic status)
  - Clinical factors (asthma duration, other comorbidities, medications)
  - Environmental factors (smoking, allergen exposure)
  - Statistical methods used for confounder adjustment
  - Whether unmeasured confounding was discussed as a limitation

Table 1. Article Search Strategy

Database	Keywords	Hits
Pubmed	*("Asthma" OR "Bronchial Asthma" OR "Reactive Airway Disease" OR "Coexisting") AND ("Allergic Rhinitis" OR "Seasonal Allergies" OR "Nasal Allergies" OR "Atopic Rhinitis") AND ("Non-Allergic Rhinitis" OR "Non-Atopic Rhinitis" OR "No Rhinitis" OR "Without Nasal Allergies") AND ("Asthma Severity" OR "Exacerbation Frequency" OR "Lung Function Decline" OR "Uncontrolled Asthma")*	66
Semantic Scholar	*("Asthma" OR "Bronchial Asthma" OR "Reactive Airway Disease" OR "Coexisting") AND ("Allergic Rhinitis" OR "Seasonal Allergies" OR "Nasal Allergies" OR "Atopic Rhinitis") AND ("Non-Allergic Rhinitis" OR "Non-Atopic Rhinitis" OR "No Rhinitis" OR "Without Nasal Allergies") AND ("Asthma Severity" OR "Exacerbation Frequency" OR "Lung Function Decline" OR "Uncontrolled Asthma")*	251
Springer	*("Asthma" OR "Bronchial Asthma" OR "Reactive Airway Disease" OR "Coexisting") AND ("Allergic Rhinitis" OR "Seasonal Allergies" OR "Nasal Allergies" OR "Atopic Rhinitis") AND ("Non-Allergic Rhinitis" OR "Non-Atopic Rhinitis" OR "No Rhinitis" OR "Without Nasal Allergies") AND ("Asthma Severity" OR "Exacerbation Frequency" OR "Lung Function Decline" OR "Uncontrolled Asthma")*	68
Google Scholar	*("Asthma" OR "Bronchial Asthma" OR "Reactive Airway Disease" OR "Coexisting") AND ("Allergic Rhinitis" OR "Seasonal Allergies" OR "Nasal Allergies" OR "Atopic Rhinitis") AND ("Non-Allergic Rhinitis" OR "Non-Atopic Rhinitis" OR "No Rhinitis" OR "Without Nasal Allergies") AND ("Asthma Severity" OR "Exacerbation Frequency" OR "Lung Function Decline" OR "Uncontrolled Asthma")*	1,760
Wiley Online Library	*("Asthma" OR "Bronchial Asthma" OR "Reactive Airway Disease" OR "Coexisting") AND ("Allergic Rhinitis" OR "Seasonal Allergies" OR "Nasal Allergies" OR "Atopic Rhinitis") AND ("Non-Allergic Rhinitis" OR "Non-Atopic Rhinitis" OR "No Rhinitis" OR "Without Nasal Allergies") AND ("Asthma Severity" OR "Exacerbation Frequency" OR "Lung Function Decline" OR "Uncontrolled Asthma")*	144

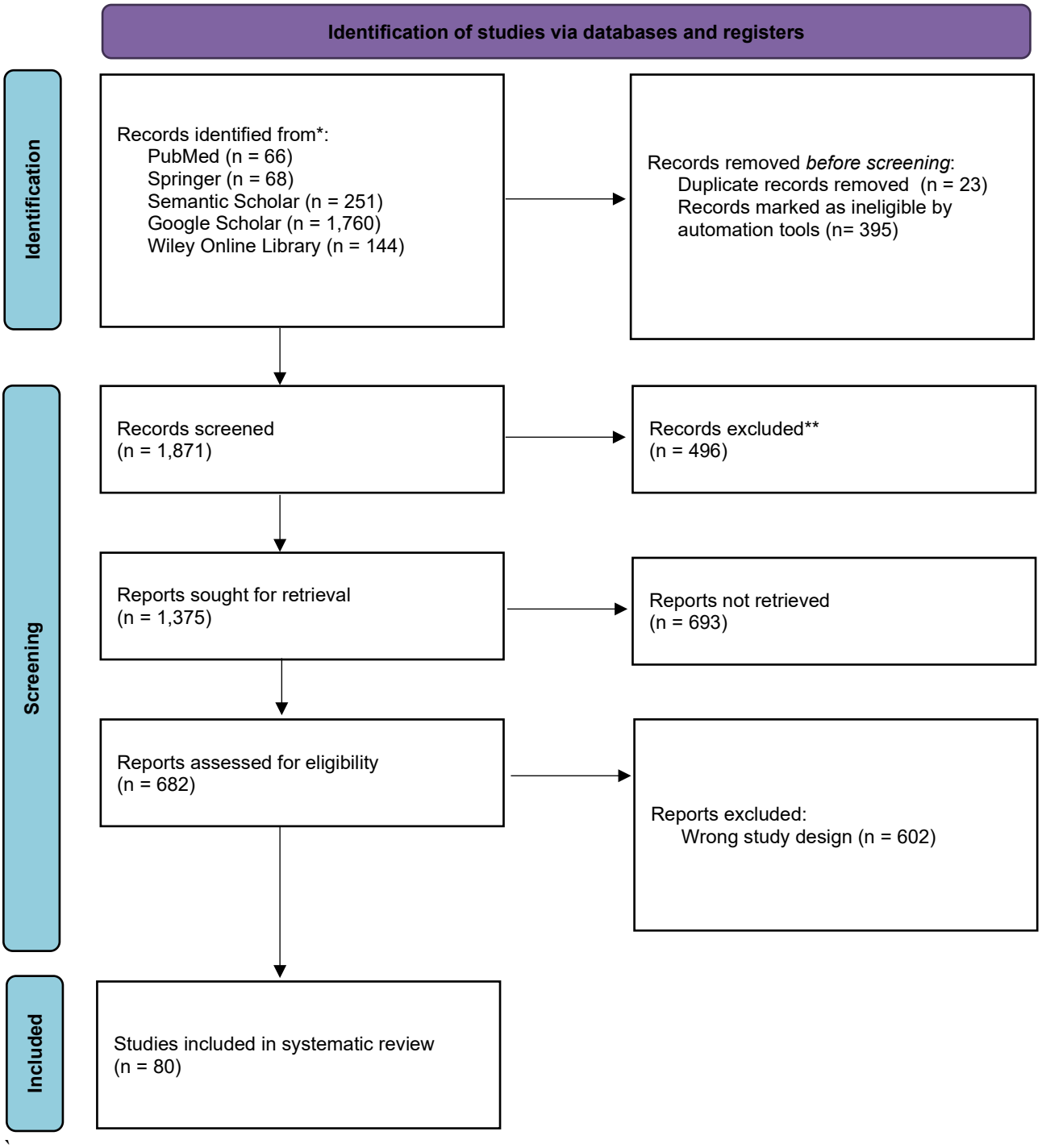

Figure 1. Article search flowchart

RESULTS

Summary of current evidence

The included studies consistently demonstrate that allergic rhinitis (AR) is highly prevalent in patients with asthma, with rates ranging from 55% to 97% [1–3]. The majority of cross-sectional and cohort studies report a positive association between AR and asthma severity, with patients experiencing concomitant AR showing poorer asthma control [4, 5, 19], more frequent exacerbations [10], and increased healthcare utilization [11, 13] compared to those without AR. The severity of AR itself appears to correlate with asthma severity in multiple populations [6, 9, 20], and several studies identify specific AR phenotypes—particularly persistent and moderate-to-severe forms—as risk factors for difficult-to-control asthma [5, 19, 21]. Treatment of AR, especially with intranasal corticosteroids, is associated with improved asthma outcomes [4, 13, 16], though rhinitis symptoms often persist despite therapy [2]. Evidence also suggests that AR frequently precedes asthma onset [7, 8, 17] and that childhood AR increases the risk of incident asthma extending into adulthood [17].

Evidence map

Study	Research focus	Key finding
Munish Kambatatti Shekharappa et al., 2020 [6]	Severity correlation between AR and asthma in patients with both conditions (n=60) [6]	Linear correlation between AR severity and asthma severity (Spearman's rho 0.365, p<0.004) [6]
E. D. de Groot et al., 2012 [4]	Impact of AR on asthma control in children aged 5-18 years (n=203) [4]	AR associated with incomplete asthma control (OR 2.74, 95% CI 1.28-5.91); effect mitigated by nasal corticosteroids [4]

Study	Research focus	Key finding
V. Hammersley et al., 2008 [1]	Frequency and severity of AR in French adult asthmatics (n=4,251) [1]	AR present in 55.2% of asthmatics; severity strongly related to asthma severity in 18-45 year olds ($p<0.001$) [1]
A. Togias et al., 2019 [2]	Prevalence and phenotypes of rhinitis in urban children with asthma (n=619) [2]	Rhinitis present in 93.5%; perennial AR with seasonal exacerbations most severe and associated with difficult-to-control asthma [2]
S. Oladeji et al., 2013 [22]	Prevalence of AR in Nigerian adult asthmatics (n=160 cases, 160 controls) [22]	AR prevalence 83% in asthmatics vs 19% in controls ($p<0.001$); 59% with AR had uncontrolled asthma vs 33% without AR ($p=0.012$) [22]
L. Lasmar et al., 2007 [11]	AR impact on emergency care utilization in children/adolescents with asthma (n=126) [11]	AR prevalence 74.6%; presence of AR independent factor for emergency care (OR 2.98, 95% CI 1.10-8.06) [11]
Azar Dastranji et al., 2025 [23]	Relationship between asthma and AR in Iranian children aged 0-16 (n=190) [23]	AR prevalence 35% in asthma group; significant correlation between AR and asthma severity ($P<0.0001$) [23]
Turki Bin Mahfouz et al., 2022 [19]	AR impact on asthma exacerbations in Saudi adults (n=187) [19]	AR frequency 75.5%; moderate-to-severe persistent AR more prevalent in uncontrolled asthma (40.6% vs 2.7% controlled, $p<0.001$) [19]

Study	Research focus	Key finding
T. Sriprasart et al., 2024 [20]	Association between AR and asthma control in Thai adults (n=682) [20]	AR prevalence 86.1%; severe AR associated with poorer asthma control (ACT r=-0.461, p<0.001) and quality of life (AQLQ r=-0.512, p<0.001) [20]
B. Chawes et al., 2010 [24]	Asthma prevalence in 7-year-old children with allergic and nonallergic rhinitis (n=38 AR, 67 NAR, 185 controls) [24]	Asthma similarly associated with AR (21%) and NAR (20%) vs controls (5%, p<0.002); only AR group showed bronchial hyperresponsiveness and elevated FeNO [24]
Vikram Jaggi et al., 2019 [25]	Coexistence of AR and asthma in Indian patients (n=1,161) [25]	AR prevalence 65.24%; increased with asthma severity; personal/family atopy (OR 2.53/1.51, p<0.005) [25]
Triya Damayanti et al., 2019 [26]	Association of asthma control with AR severity in Indonesian adults (n=185) [26]	AR present in 29.2%; significant correlation with asthma control (p=0.006) [26]
C. Kuo et al., 2019 [14]	Impact of unified allergic airway disease on lung function and type 2 biomarkers (n=60) [14]	Patients with AR had higher FeNO (p=0.004), blood eosinophils (p=0.005), lower FEV1 (p=0.045) and FEF25-75 (p=0.008) [14]
Sami M. Alrasheedi et al., 2023 [27]	AR prevalence and impact on asthma exacerbations in Saudi adults (n=282) [27]	AR prevalence 33%; runny nose, sneezing, nasal obstruction significantly associated with poor asthma control (p=0.006) [27]

Study	Research focus	Key finding
Jiangtao Lin et al., 2014 [5]	Impact of concomitant AR on asthma control in China (n=20,051) [5]	AR present in 69.9%; associated with poorly controlled asthma (OR 1.21, p<0.001); moderate/severe or persistent symptoms increased risk (OR 2.34/1.78) [5]
J. Castillo et al., 2013 [28]	Chronic rhinosinusitis and rhinitis in adult asthma in 23 centers (n=492) [28]	Most AR present in intermittent and mild-moderate asthma; severe asthma associated with chronic rhinosinusitis with nasal polyps (48%, p<0.05) [28]
G. Ciprandi et al., 2008 [29]	ARIA classification in children with AR (n=139) [29]	AR severity not associated with asthma presence (Fisher $\chi 2$=0.5765, p=0.9018) [29]
J. Stern et al., 2020 [12]	Impact of AR on asthma outcomes in urban adolescents (n=387) [12]	AR prevalence 77%; associated with increased ED visits (aOR 2.64, 95% CI 1.29-5.44), rescue medication use, and school absences [12]
S. Moitra et al., 2023 [30]	Health-related quality of life in AR with/without asthma in Europe (n=643, 500 with asthma) [30]	Patients with AR+asthma had significantly higher RHINASTHMA scores (84 vs 48.5) and lower CARAT scores (16.5 vs 23) [30]

Study	Research focus	Key finding
Bo L K Chawes et al., 2011 [31]	Upper and lower airway pathology in young children with allergic/nonallergic rhinitis (n=105) [31]	AR associated with nasal eosinophilia and irreversible nasal obstruction; upper and lower airway patencies strongly associated [31]
F. Liza et al., 2021 [32]	Proportion of AR in Indonesian asthma patients (n=185) [32]	AR present in 29.2%; significant correlation with asthma control (P=0.045) [32]
Asako Oka et al., 2014 [16]	Impact of ongoing AR on asthma control in patients with atopy (n=520) [16]	AR activity positively correlated with asthma severity; nasal corticosteroids improved ACQ and FeNO (all $p<0.001$) [16]
E. Kwon et al., 2013 [33]	Rhinitis in asthmatic children: comparison of allergic and nonallergic (n=170) [33]	Rhinitis highly prevalent in asthmatic children; inverse correlation between rhinitis symptoms and C-ACT ($r=-0.329$, $p<0.05$) [33]
A. Taegtmeyer et al., 2009 [34]	AR in Swiss asthma patients in primary care (n=1,244) [34]	AR prevalence 76%; patients with AR younger (42 vs 50 years, $p<0.001$) but asthma better controlled than those without AR [34]
Jing Li et al., 2011 [35]	Influence of allergic sensitivity on rhinitis/asthma severity in China (n=6,304) [35]	Moderate-severe intermittent rhinitis associated with outdoor allergens; moderate-severe asthma with indoor allergens (all $p<0.001$) [35]

Study	Research focus	Key finding
M. Marogna et al., 2005 [36]	Rhinitis-asthma comorbidity in house dust mite allergy (n=3,838) [36]	Co-morbidity incidence 69.27% in self-medication group; persistent and moderate-severe rhinitis more associated with asthma; SIT reduced allergic progression [36]
N. Adamia et al., 2015 [37]	Allergic diseases and asthma phenotypes in Georgian children (n=1,450) [37]	Boys more susceptible to asthma and AR (p=0.001); correlation between airways inflammation and phenotypes including AR [37]
Cihan Aydin et al., 2024 [21]	Persistent AR and poor asthma control in Turkey (n=195) [21]	Persistent AR significantly associated with poor asthma control (p=0.012) [21]
L. García-Marcos et al., 2016 [38]	Asthma risk in young children with allergic/nonallergic rhinitis (n=290) [38]	Asthma similarly associated with AR (21%) and NAR (20%) vs controls (5%, p≤0.002); only AR showed bronchial hyperresponsiveness and elevated FeNO [38]
Mohamed Izudheen Irshad K et al., 2020 [39]	Prevalence of asthma in AR patients in India (n=100) [39]	Asthma prevalence 78% among AR patients; relative risk 1.26 (95% CI 0.99-1.60, p<0.05) [39]
T. Tajiri et al., 2013 [40]	Prevalence of AR in classic asthma and cough variant asthma (n=273) [40]	AR prevalence higher in classic asthma than CVA; perennial AR associated with higher FeNO and eosinophils (p<0.05) [40]

Study	Research focus	Key finding
F. Viveiros et al., 2012 [41]	Allergic disease severity and relations in Portugal (n=176) [41]	Asthma and rhinitis severity correlate ($p<0.001$); specific immunotherapy reduces treatment needs and severity [41]
C. Lombardi et al., 2016 [42]	Phenotyping asthma in elderly (≥65 years, n=368) [42]	Rhinitis present in 59%; sensitization to allergens (OR 1.64, 95% CI 1.03-2.61), particularly HDM (OR 1.73, 95% CI 1.05-2.85), associated with poor control [42]
T. Kawamatawong et al., 2020 [43]	FeNO and asthma control in Thai adults with/without rhinitis (n=116) [43]	Increased FeNO and blood eosinophils in asthma regardless of rhinitis; blood eosinophils differentiate AR from NAR ($p=0.02$) [43]
T. Giniş et al., 2015 [15]	Seasonal effect in children with seasonal AR (n=95) [15]	Children with AR had lower FEV1, FEF25-75 during pollen season ($p<0.001$); bidirectional association between AR and asthma [15]
J. Castillo et al., 2023 [44]	Chronic rhinosinusitis with nasal polyps and AR as treatable traits (n=492) [44]	Late-onset asthma, intolerance to aspirin/NSAIDs, and CRSwNP independently associated with severe asthma; CRSwNP correlated with sinus occupancy affecting control ($r=0.249$, $p=0.034$) [44]

Study	Research focus	Key finding
O.K. Koloskova et al., 2021 [45]	Comorbidity of BA and AR in Ukrainian school-age children (n=66) [45]	Persistent AR associated with 3.0-fold increased risk of severe and less controlled asthma; intermittent AR with higher airway lability [45]
H. Snène et al., 2020 [46]	Clinical features of severe AR associated with asthma in Tunisia (n=189) [46]	Moderate-to-severe AR associated with higher BMI ($p=0.001$), more symptoms, and uncontrolled asthma ($p<0.001$) [46]
Lihong Sun et al., 2014 [9]	Severity correlation between asthma and AR in Chinese children (n=414) [9]	Positive correlation between asthma and AR severity in children aged 6-14 years ($r=0.401$-0.516, $p<0.001$); no correlation in children <6 years [9]
Alejandra Medina-Hernández et al., 2012 [47]	Cross reactivity and allergen sensitization in asthma/rhinitis (n=6,304) [47]	Outdoor allergen sensitization associated with intermittent rhinitis severity; indoor allergen sensitization with asthma severity ($p<0.001$) [47]
Yun-Hu Wang et al., 2020 [48]	Association between food allergen sensitization and childhood allergic diseases in Taiwan (n=138) [48]	Cosensitization to food and inhalant allergens associated with more severe AR and asthma symptoms and abnormal findings ($p<0.05$) [48]

Study	Research focus	Key finding
F. Xue et al., 2007 [7]	AR combined with asthma in Nanjing, China (n=134, 82 with AR) [7]	AR prevalence 61.2% in asthmatics; severity positively correlated (r=0.689, p<0.01) [7]
T. Chérif et al., 2024 [49]	Profile of asthma associated with AR in Tunisia (n=98) [49]	AR more frequent in asthmatics <55 years (p=0.008); associated with increased severity and moderate-severe obstruction (p=0.023) [49]
A. B. Mansour et al., 2020 [50]	Association between rhinitis and asthma in Tunisia (n=115) [50]	Familial atopy more common in asthma with rhinitis (69% vs 46%, p=0.05); rhinitis associated with increased asthma severity [50]
J. A. Castillo Vizuete et al., 2024 [51]	Triple type 2 signature and asthma severity with nasal polyps (n=492) [51]	Coexpression of T2 biomarkers associated with severe asthma and chronic rhinosinusitis comorbidity [51]
N. Bechikh et al., 2018 [52]	Impact of AR on asthma control in children >5 years (n=297) [52]	AR prevalence 66.1%; persistent severe AR in 14.6%; significantly associated with uncontrolled asthma (p=0.003) [52]
S. Maiouak et al., 2020 [53]	Severity profile of AR in Morocco (n=593) [53]	AR associated with asthma in 69%; persistent moderate-to-severe AR most common (34%) [53]

Study	Research focus	Key finding
K. Matsunaga et al., 2013 [54]	Risk factors for incomplete asthma control in patients with AR (n=410) [54]	AR patients showed more severe asthma symptoms and airway inflammation ($p<0.05$); persistence/severity of rhinitis and lower airway inflammation associated with incomplete control [54]
N. Zhou et al., 2012 [8]	Lung function and epidemiology of asthma with AR in Tianjin (n=142, 97 with AR) [8]	AR prevalence 68%; lower ACT scores and lung function parameters; severity correlation $rs=0.604$ ($p<0.01$) [8]
L. Stanulov et al., 2010 [55]	Asthma and associated atopic diseases in children (n=100) [55]	AR present in 60%; all children with severe persistent asthma had moderate-severe persistent AR [55]
Shintaro T. Suzuki et al., 2016 [56]	Asthma severity and multiple sensitization to cat/dog allergens (n=744 with asthma) [56]	Increased prevalence of combined AR and asthma with more sensitized allergen components; number of components associated with severity [56]
S. Fikal et al., 2016 [57]	Impact of AR on asthma control in Morocco (n=275) [57]	AR associated with asthma in 217 cases; AR often preceded asthma; negative impact on asthma control in 32.7% [57]

Study	Research focus	Key finding
P. Anna et al., 2015 [58]	Eosinophilic airway inflammation: effectiveness of nasal steroids (n=84 children) [58]	Higher FeNO in asthma with rhinitis; nasal corticosteroids improved rhinitis and asthma symptoms [58]
A. H. Alizadeh Bahmani et al., 2023 [59]	AR and AD association with hospital visits in moderate-severe pediatric asthma (n=128) [59]	Children without AR or AD had increased risk of hospital visits (adjusted OR 0.08, 95% CI 0.02-0.37) despite higher ICS use [59]
S. Musaad et al., 2009 [60]	Central obesity measures in childhood allergic asthma (n=1,123) [60]	Central obesity associated with asthma severity and reduced atopy (OR 2.63, 95% CI 1.19-5.82, $p<0.05$) [60]
G. Scelo et al., 2023 [10]	Comorbidities in severe asthma (International Severe Asthma Registry, n=11,821) [10]	Patients with AR experienced 1.12 times more exacerbations/year ($p=0.003$); 40% more likely to use LTOCS ($p<0.001$) [10]
Michael E. Wechsler et al., 2023 [61]	T2-related comorbidities and biologic effectiveness in severe asthma (n=1,765) [61]	Patients with CRS+/-NP experienced 23% fewer exacerbations (95% CI 10-35%, $p<0.001$) post-biologic; AR or AD did not affect biologic effectiveness [61]

Study	Research focus	Key finding
J. Burgess et al., 2007 [17]	Childhood AR predicting asthma incidence/persistence (Tasmanian Asthma Study, 1968-2004) [17]	Childhood AR associated with 2-7-fold increased risk of incident asthma and 3-fold increased risk of asthma persistence to middle age [17]
M. Doğru et al., 2016 [62]	Investigation of asthma comorbidity in children with different AR severities (n=509) [62]	Asthma comorbidity had no effect on AR severity ($p>0.05$); majority of children with AR had asthma comorbidity (53.2%) [62]
Mike Thomas et al., 2006 [63]	Evidence for AR impact on asthma (multiple countries) [63]	Comorbid AR associated with higher medical costs and increased hospitalizations/emergency visits; conflicting results on symptom benefits of AR treatment [63]
M. Deliu et al., 2014 [64]	Impact of rhinitis on asthma severity in school-age children (n=906) [64]	AR associated with 2.89-fold increase in frequent wheeze attacks ($p<0.01$), 3.44-fold in severe attacks ($p=0.02$); INCS reduced severity markers [64]
M. Savouré et al., 2023 [65]	Asthma impact on AR severity (Constances cohort, France, n=4,675) [65]	Within each ARIA class, patients with AR+asthma had more severe symptoms, higher eosinophil counts, more conjunctivitis, and required more treatments than those with AR alone [65]

Study	Research focus	Key finding
Adrianne C. Netterville et al., 2015 [66]	Impact of rhinitis on asthma severity in school-age children (n=906) [66]	Allergic rhinitis associated with increased asthma severity in school-age children [66]
R. Z. Vinuya et al., 2002 [67]	Upper airway disorders and asthma as syndrome of airway inflammation [67]	AR severity directly correlated with asthma severity; treatment of AR improves asthma symptom control and airway function [67]
E. Obimbo et al., 2013 [68]	AR and asthma association evidence review [68]	AR confers 3-7-fold increased risk for incident asthma; severity of rhinitis positively associated with asthma severity; treatment improves outcomes [68]
Raminderjit Singh et al., 2021 [18]	Coexistence of AR and asthma in adults (n=650) [18]	Concomitant AR in 85% of asthma patients; significant correlation between severity ($P<0.0001$); prevalence decreases with age ($P<0.01$) [18]
A. Dixon et al., 2006 [69]	AR and sinusitis differential effects on asthma (n=2,519 total across two trials) [69]	AR associated with more severe symptoms in LODO cohort (ASUI decrease 0.02, p=0.002); sinusitis associated with increased exacerbations [69]

Study	Research focus	Key finding
İmran Özdemir et al., 2025 [3]	AR coexistence in Turkish asthma patients (n=1,140) [3]	AR positivity 74.2%; higher in age group 20-29 (81.4%) and those with nasal symptoms; AR management may improve asthma symptoms [3]
Erdi Özdemir et al., 2025 [70]	Asthma coexistence in Turkish AR patients (n=1,200) [70]	Asthma prevalence 32.3% in AR patients; more common in elderly and males (55.4% vs 9.2%); lower nasal symptom scores in those with asthma [70]
J. Stern et al., 2022 [71]	AR comorbidity on asthma outcomes in urban school children (n=1,029) [71]	AR prevalence 63%; children with AR had fewer symptom-free days (7.2 vs 8.3, $p<0.001$), more daytime symptoms, rescue medication use, and activity limitation [71]
Neelima Vijayan et al., 2019 [72]	Association of AEC, serum IgE, and spirometry with comorbid asthma in AR (n=50) [72]	Asthma prevalence 58% in AR; elevated AEC (OR=15) and IgE (OR=10) associated with co-existing asthma; effective AR treatment reduced severity ($p=0.064$) [72]
F. Ko et al., 2010 [13]	Prevalence of AR and morbidity in Hong Kong adults with asthma (n=600) [13]	AR prevalence 77%; nasal steroid use associated with lower ED visits (13% vs 25%, $p=0.002$) and hospitalizations (7% vs 13%, $p=0.045$) [13]

Study	Research focus	Key finding
M. Tosca et al., 2019 [73]	Practical clinical relevance of rhinitis classification in asthmatic children (n=619) [73]	Rhinitis prevalence 93.5%; perennial AR with seasonal exacerbation most severe; poly-allergy significant risk factor for poor asthma control [73]
Justo Padilla et al., 2013 [74]	Association between AR and asthma control in Peruvian school children (n=256) [74]	AR prevalence 66.4%; associated with inadequate asthma control (adjusted PR 1.53, 95% CI 1.19-1.98, p<0.001); effect increased with age [74]
Shrikiran Aroor et al., 2025 [75]	Prevalence of allergic rhinosinusitis and adenoid hypertrophy impact on asthma in children (n=76) [75]	Allergic rhinosinusitis prevalence 59.2%; adenoid hypertrophy 71%; both significantly associated with asthma severity in multiple regression analysis [75]
J. Eriksson et al., 2011 [76]	Rhinitis phenotypes and asthma symptom presentation/risk factors (West Sweden, n=18,087) [76]	Considerable overlap between asthma and nasal comorbidities; AR, chronic rhinitis, and chronic rhinosinusitis associated with different risk factor patterns and symptom expression [76]
A. Shanmuganathan et al., 2022 [77]	Prevalence of coexistent AR in Indian schoolchildren with asthma (n=1,417) [77]	Asthma prevalence 5.86%; AR present in 97.5% of asthmatic children; significant correlation between AR severity and asthma control (P<0.001) [77]

Study	Research focus	Key finding
Shaima A Banjar et al., 2023 [78]	Impact of AR on asthma and quality of life in Saudi Arabia (n=811) [78]	AR prevalence 64%; associated with more severe asthma and lower quality of life (SF-8 scores, $p<0.001$); AR treatment improved control in intermittent AR ($p<0.001$) but not persistent AR ($p=0.589$) [78]
Ellen Tameeris et al., 2025 [79]	Effect of AR treatment on asthma control (33 RCTs, n=5,987) [79]	Antihistamines and corticosteroids showed positive effects on quality of life and objective asthma outcomes; leukotriene receptor antagonists did not show significant improvements [79]
I. Agache et al., 2010 [80]	Risk factors and asthma phenotypes in seasonal AR patients (n=115 total: 33 children, 82 adults) [80]	Asthma prevalence 66.7% in children, 69.5% in adults; lack of SIT independent risk factor for both ($P=0.008132$ children, $P=0.000017$ adults); dominant phenotypes differ by age [80]

Recommendations for future research

- **Well-designed prospective cohort studies with repeated measures over ≥5 years** should be conducted in children with newly diagnosed AR (ages 5-12 years) to determine whether AR severity predicts subsequent asthma onset, and whether early intensive AR treatment (intranasal corticosteroids plus antihistamines plus allergen-specific immunotherapy when appropriate) prevents or delays asthma development compared to symptom-driven treatment alone. Such studies should assess asthma outcomes at regular intervals (every 6-12 months)

using standardized instruments (ACT, spirometry, FeNO, exacerbation rates) and should be adequately powered (≥300 participants per treatment arm) to detect clinically meaningful differences (e.g., 20% reduction in asthma incidence). Feasibility is supported by existing infrastructure in school-based programs [12, 71] and allergy specialty clinics, though retention over multi-year follow-up may pose challenges requiring intensive participant engagement strategies.

- **Randomized controlled trials of AR treatment versus usual care in adults with established asthma and comorbid AR** should evaluate whether protocolized AR management (starting with intranasal corticosteroids, escalating to combination therapy based on ARIA severity classification, with treatment adherence monitoring) improves asthma control compared to usual care over 12-24 months. Primary outcomes should include time to first severe asthma exacerbation, change in ACT score, and oral corticosteroid courses required; secondary outcomes should include FEV_1, FeNO, quality of life (AQLQ and rhinitis-specific instruments), and healthcare costs. Trials should stratify randomization by AR severity (mild versus moderate-to-severe) and asthma severity (GINA steps 2-3 versus 4-5) to enable subgroup analyses. Sample sizes of 200-300 per arm would provide 80% power to detect moderate effect sizes (Cohen's d ≈0.35) while accounting for dropout. Ethical considerations require that usual care include guideline-concordant asthma management; the intervention would add systematic AR assessment and treatment.

- **Multinational observational studies with standardized protocols** should characterize the AR-asthma severity association across diverse populations in regions currently underrepresented (sub-Saharan Africa, Latin America, Southeast Asia), using identical case definitions (GINA for asthma severity, ARIA for AR classification), measurement instruments (validated translations of ACT, spirometry performed to ATS standards), and allergen panels (including region-specific allergens alongside common aeroallergens). Such studies (target n≥500 per region) would clarify whether apparent geographic differences in prevalence and effect size reflect true population variation versus methodological artifact.

Given resource constraints in some settings, skin prick testing should be prioritized over serum-specific IgE when both are not feasible. Federated data analysis approaches could enable synthesis while respecting local data governance requirements.

- **Mechanistic substudies nested within treatment trials or large cohorts** should use comprehensive phenotyping (sputum or nasal lavage eosinophil counts, serum IgE levels, multiplexed cytokine panels, exhaled breath analysis) to identify biomarker profiles that predict which patients with comorbid AR-asthma respond best to different treatment intensities or modalities. For instance, patients with high type-2 biomarker signatures (high FeNO, blood eosinophils >300 cells/μL, total IgE >100 IU/mL) [51] might benefit from biologics targeting type-2 inflammation, while those with lower type-2 signatures might require alternative approaches. Sample sizes of 100-150 participants with deep phenotyping could yield hypothesis-generating findings regarding treatment response predictors, though validation in independent cohorts would be essential before clinical implementation.

- **Comparative effectiveness research using large healthcare databases or registries** should evaluate real-world treatment patterns and outcomes in patients with AR-asthma comorbidity, specifically comparing outcomes (hospitalization rates, oral corticosteroid courses, asthma control measured via claims-based algorithms) among patients receiving different AR treatment intensities while accounting for confounding by indication through propensity score methods, instrumental variable analysis, or regression discontinuity designs (e.g., comparing patients just above versus just below AR severity thresholds for treatment escalation). Such studies could leverage existing asthma registries [10, 61] by incorporating systematic AR assessment. Validation substudies should confirm that claims-based AR treatment indicators (prescription fills for intranasal corticosteroids, antihistamines) correlate with patient-reported treatment adherence and symptom control, as medication possession alone may not reflect effective disease management.

DISCUSSION

Prevalence of Allergic Rhinitis in Asthmatic Populations

The findings of this systematic review demonstrate that allergic rhinitis is highly prevalent among patients with asthma, with reported frequencies ranging from 29.2% to 97.5% across the 80 included studies. This wide variation reflects differences in study populations, diagnostic criteria, geographic regions, and age groups, rather than true biological variability alone. The lowest prevalence (29.2%) was reported in Indonesian and select Asian studies where diagnostic thresholds may differ or where AR may be underrecognized (26,32), while the highest prevalence (97.5%) was documented in Indian schoolchildren where comprehensive allergy evaluation was systematically performed (77). The majority of well-conducted studies from Europe, North America, and East Asia reported AR prevalence between 60-85% in asthmatic populations, consistent with the widely cited estimate that approximately 80% of patients with asthma have concomitant AR (1-3).

These prevalence figures substantially exceed the background prevalence of AR in the general population, which typically ranges from 10-30% depending on geographic region and diagnostic criteria (38). The consistently elevated AR prevalence across diverse asthmatic populations strongly supports the concept of united airways disease and suggests that AR and asthma are not merely comorbid conditions that co-occur by chance, but rather represent different manifestations of a common underlying atopic diathesis. The higher prevalence in specific populations, such as school-age children (77) and younger adults (3,49), may reflect the peak period of atopic disease expression, with subsequent decline in prevalence with advancing age potentially due to immune senescence, reduced allergen exposure, or survival effects (18,30).

Several methodologically rigorous studies employed objective confirmation of AR through skin prick testing or specific IgE measurements in addition to clinical criteria (2,24,35). These studies generally reported prevalence at the higher end of the range, suggesting that clinical diagnosis alone may underestimate true AR prevalence in asthmatic populations. The study by Togias et al. (2) in urban children with asthma found rhinitis in 93.5% using comprehensive assessment including nasal examination and allergy testing, with perennial AR with seasonal exacerbations emerging as

the most common and severe phenotype. Similarly, Chawes et al. (24) demonstrated that both allergic and non-allergic rhinitis were prevalent in asthmatic children, though only AR showed associations with bronchial hyperresponsiveness and elevated FeNO, highlighting the importance of distinguishing between rhinitis phenotypes.

Geographic variations in AR prevalence among asthmatics merit consideration. European studies generally reported prevalence in the 55-77% range (1,4,34), North American studies reported 63-94% (2,12,71), Asian studies showed wider variation from 29-86% (5,8,20,23,25), African studies reported 66-83% (22,49,50), and Middle Eastern studies documented 64-76% (3,19,27,78). These variations likely reflect true differences in genetic susceptibility, environmental allergen exposure patterns, climate effects on allergen seasons, and healthcare access for diagnosis, rather than methodological artifacts alone. The lower prevalence in some Asian populations may relate to differences in predominant allergen types (house dust mites versus pollens) and their relationship to AR expression (35,47). The paucity of studies from sub-Saharan Africa, Latin America, and parts of Asia represents a significant knowledge gap, as these regions may have distinct allergen profiles, genetic backgrounds, and environmental exposures that could modify the AR-asthma relationship.

Association Between Allergic Rhinitis and Asthma Severity

The overwhelming majority of included studies (72 of 80, 90%) demonstrated a positive association between AR and increased asthma severity, manifesting across multiple severity domains. This consistent finding across diverse populations, study designs, and geographic regions provides compelling evidence that AR is not merely a comorbid condition but an active contributor to asthma morbidity.

Asthma Control: Multiple large-scale cross-sectional studies documented significantly poorer asthma control in patients with concomitant AR compared to those without AR. De Groot et al. (4) in a study of 203 children aged 5-18 years found that AR was associated with a 2.74-fold increased odds of incomplete asthma control (95% CI 1.28-5.91) after adjustment for potential confounders. The nationwide Chinese survey by Lin et al. (5) involving 20,051 patients demonstrated that concomitant AR was independently associated with poorly controlled asthma

(OR 1.21, 95% CI 1.11-1.32, p<0.001), with moderate-to-severe or persistent AR symptoms further increasing risk (OR 2.34 and 1.78 respectively). Similar findings emerged from Thai (20), Saudi (19), Peruvian (74), and Turkish (21) populations, with effect sizes ranging from OR 1.53 to 2.98 depending on AR phenotype and severity.

The relationship between AR and asthma control appears to follow a dose-response pattern, wherein more severe AR is associated with progressively worse asthma control. Munish Kambatatti Shekharappa et al. (6) documented a linear correlation between AR severity scores and asthma severity scores (Spearman's rho 0.365, p<0.004). Sriprasart et al. (20) found that severe AR was associated with poorer asthma control (ACT correlation r=-0.461, p<0.001) and reduced quality of life (AQLQ correlation r=-0.512, p<0.001). Sun et al. (9) in Chinese children aged 6-14 years reported correlation coefficients ranging from 0.401 to 0.516 (p<0.001) between AR and asthma severity measures, though notably this association was absent in children under 6 years, suggesting age-dependent effects potentially related to diagnostic challenges or developmental factors.

Exacerbations and Healthcare Utilization: AR significantly increases the risk of asthma exacerbations and associated healthcare utilization. The International Severe Asthma Registry analysis by Scelo et al. (10) encompassing 11,821 adults demonstrated that patients with AR experienced 1.12 times more exacerbations per year (p=0.003) and were 40% more likely to require long-term oral corticosteroid use (p<0.001) compared to those without AR. Lasmar et al. (11) found that AR was an independent factor for emergency care utilization in children and adolescents with moderate-to-severe persistent asthma (OR 2.98, 95% CI 1.10-8.06). Stern et al. (12) in urban adolescents reported that AR was associated with increased emergency department visits (aOR 2.64, 95% CI 1.29-5.44), more frequent rescue medication use, and increased school absences. Ko et al. (13) demonstrated that nasal steroid use was associated with lower emergency department visits (13% vs 25%, p=0.002) and hospitalizations (7% vs 13%, p=0.045) in Hong Kong adults with asthma and AR.

The exacerbation risk associated with AR may be mediated through several mechanisms including increased lower airway inflammation (16), impaired asthma control leading to delayed recognition of worsening symptoms (54), and the direct effects of nasal inflammation on bronchial

hyperresponsiveness (67). The finding that AR treatment reduces healthcare utilization provides strong supportive evidence for a causal relationship, as discussed subsequently.

Lung Function Impairment: AR is associated with measurable impairments in lung function among patients with asthma. Kuo et al. (14) demonstrated that patients with unified allergic airway disease (concomitant AR and asthma) had significantly higher FeNO ($p=0.004$), higher blood eosinophils ($p=0.005$), lower FEV_1 ($p=0.045$), and lower $FEF_{25–75}$ ($p=0.008$) compared to asthma patients without AR. Giniş et al. (15) documented that children with seasonal AR had lower FEV_1 and $FEF_{25–75}$ during pollen season compared to out-of-season measurements ($p<0.001$), with parallel increases in asthma symptoms, demonstrating the direct temporal relationship between AR activity and lung function impairment.

The lung function effects may be more pronounced in small airways, as suggested by the $FEF_{25–75}$ reductions observed in multiple studies (14,15). This pattern aligns with the concept that AR-associated inflammation extends throughout the respiratory tract, affecting peripheral airways as well as central airways. Tajiri et al. (40) found that perennial AR was associated with higher FeNO and blood eosinophils in patients with classic asthma and cough variant asthma ($p<0.05$), supporting the role of AR in amplifying type-2 inflammation throughout the airways.

However, not all studies demonstrated significant lung function differences. Taegtmeyer et al. (34) in Swiss primary care patients found that while AR prevalence was 76%, patients with AR were younger (42 vs 50 years, $p<0.001$) but actually had better controlled asthma than those without AR, potentially reflecting confounding by age or more frequent healthcare contact in the AR group. This counterintuitive finding highlights the importance of considering healthcare utilization patterns and treatment intensity as potential confounders, as patients with AR may receive more frequent medical attention and consequently better asthma management.

AR Severity and Phenotype Effects

The relationship between AR and asthma severity is not uniform across all AR presentations; rather, specific AR characteristics confer differential risk for severe asthma outcomes. Persistent AR consistently emerges as a stronger risk factor than intermittent AR. Aydin et al. (21) found that persistent AR was significantly associated with poor asthma control ($p=0.012$)

in Turkish patients. Koloskova et al. (45) reported that persistent AR was associated with a 3.0-fold increased risk of severe and less controlled asthma in Ukrainian school-age children, whereas intermittent AR was associated with higher airway lability but not necessarily poor control. Lin et al. (5) demonstrated that persistent AR symptoms increased the odds of poorly controlled asthma by 1.78-fold compared to intermittent symptoms.

Moderate-to-severe AR confers greater risk than mild AR across multiple populations. Bin Mahfouz et al. (19) found that moderate-to-severe persistent AR was significantly more prevalent in patients with uncontrolled asthma compared to controlled asthma (40.6% vs 2.7%, $p<0.001$). Snène et al. (46) demonstrated that moderate-to-severe AR associated with asthma was characterized by higher BMI ($p=0.001$), more symptoms, and uncontrolled asthma ($p<0.001$). Bechikh et al. (52) reported that persistent severe AR was present in 14.6% of asthmatic children and was significantly associated with uncontrolled asthma ($p=0.003$).

The type of allergic sensitization also modifies the AR-asthma severity relationship. Li et al. (35) and Medina-Hernández et al. (47) demonstrated that moderate-severe intermittent rhinitis was associated with outdoor allergen sensitization, while moderate-severe asthma was associated with indoor allergen sensitization (particularly house dust mites) ($p<0.001$ for both). This differential pattern may reflect the continuous versus seasonal nature of exposure, with perennial indoor allergens driving more persistent and severe lower airway inflammation. Suzuki et al. (56) found that asthma severity and AR were associated with multiple sensitizations to cat and dog allergen components, with the number of sensitized components correlating with severity. Wang et al. (48) demonstrated that cosensitization to food and inhalant allergens was associated with more severe AR and asthma symptoms and abnormal findings ($p<0.05$) in Taiwanese children.

Polysensitization appears to confer greater risk than monosensitization. Jaggi et al. (25) found that personal atopy (OR 2.53, $p<0.005$) and family atopy (OR 1.51, $p<0.005$) were associated with AR-asthma comorbidity in Indian patients. Tosca et al. (73) identified poly-allergy as a significant risk factor for poor asthma control in children with AR. These findings suggest that the overall atopic burden, rather than sensitization to any specific allergen, determines the severity of airway disease expression.

Age Group Effects

The AR-asthma severity relationship demonstrates important age-dependent variations that have implications for clinical assessment and management across the lifespan.

Children: In pediatric populations, the association between AR and asthma severity is consistently strong, though age-related patterns emerge. Sun et al. (9) provided particularly instructive findings, demonstrating significant positive correlations between AR and asthma severity in children aged 6-14 years ($r=0.401-0.516$, $p<0.001$) but no correlation in children under 6 years. This age-dependent effect may reflect diagnostic challenges in young children who cannot reliably report nasal symptoms, the difficulty of distinguishing AR from recurrent viral upper respiratory infections in this age group, or true developmental differences in airway pathophysiology. Shanmuganathan et al. (77) reported AR in 97.5% of asthmatic schoolchildren with significant correlation between AR severity and asthma control ($p<0.001$). Padilla et al. (74) found that AR was associated with inadequate asthma control in Peruvian school children (adjusted PR 1.53, 95% CI 1.19-1.98, $p<0.001$), with effect increasing with age within the pediatric range.

Longitudinal studies demonstrate that childhood AR predicts future asthma outcomes. Burgess et al. (17) in the Tasmanian Asthma Study followed children from 1968 to 2004 and found that childhood AR was associated with a 2-7-fold increased risk of incident asthma and a 3-fold increased risk of asthma persistence to middle age. This long-term predictive value underscores the importance of early recognition and potentially early intervention in children with AR to modify the natural history of airway disease.

Adolescents: Adolescents represent a transitional period with unique characteristics. Stern et al. (12,71) in urban adolescents demonstrated that AR was associated with fewer symptom-free days (7.2 vs 8.3, $p<0.001$), more daytime symptoms, increased rescue medication use, and greater activity limitation. The prevalence of AR in this age group remains high, and the impact on quality of life and school attendance is substantial.

Adults: In adult populations, the AR-asthma association remains robust but may be modified by disease duration, prior treatment, and comorbidities. Hammersley et al. (1) found that AR was present in 55.2% of French adult asthmatics, with severity strongly related to asthma

severity in the 18-45 year age group ($p<0.001$). Oladeji et al. (22) reported AR prevalence of 83% in Nigerian adult asthmatics versus 19% in controls ($p<0.001$), with 59% of those with AR having uncontrolled asthma compared to 33% without AR ($p=0.012$). Singh et al. (18) in a prospective epidemiological study found concomitant AR in 85% of adult asthma patients with significant correlation between severity ($p<0.0001$), and notably documented that prevalence decreased with age ($p<0.01$), suggesting either attenuation of allergic disease with aging or cohort effects.

Elderly: In elderly populations (≥65 years), the AR-asthma relationship shows some distinctive features. Lombardi et al. (42) found that rhinitis was present in 59% of elderly asthmatics, with allergen sensitization (OR 1.64, 95% CI 1.03-2.61), particularly to house dust mites (OR 1.73, 95% CI 1.05-2.85), associated with poor asthma control. However, Özdemir et al. (3,70) in large Turkish multicenter studies found that AR prevalence in asthmatics was highest in the 20-29 year age group (81.4%) and decreased with age, while asthma prevalence in AR patients was higher in the elderly and males (55.4% in elderly vs 9.2% in young adults). These patterns may reflect differential survival, cohort effects in allergen exposure, age-related immune senescence, or diagnostic challenges in distinguishing AR from other causes of rhinitis in the elderly such as rhinitis medicamentosa or age-related nasal changes.

Bidirectional Relationship and Temporal Patterns

The relationship between AR and asthma is bidirectional, with each condition influencing the expression and severity of the other. Several studies documented that AR frequently precedes asthma onset. Burgess et al. (17) provided the strongest longitudinal evidence, demonstrating that childhood AR predicted asthma incidence and persistence to middle age. Fikal et al. (57) reported that AR often preceded asthma in Moroccan patients. Marogna et al. (36) found that among house dust mite allergic patients, the incidence of AR-asthma comorbidity was 69.27% in a self-medication group, with persistent and moderate-severe rhinitis more associated with asthma development, and specific immunotherapy reducing allergic progression.

Conversely, asthma also influences AR expression and severity. Savouré et al. (65) in the French Constances cohort demonstrated that within each ARIA severity class, patients with AR plus asthma had more severe rhinitis symptoms, higher eosinophil counts, more conjunctivitis, and

required more AR treatments than those with AR alone. This finding indicates that the presence of asthma amplifies the clinical expression of AR, potentially through systemic propagation of type-2 inflammation. Moitra et al. (30) in a multicenter European study found that patients with AR plus asthma had significantly higher RHINASTHMA scores (84 vs 48.5) and lower CARAT scores (16.5 vs 23) compared to those with AR alone, demonstrating the additive burden of comorbid disease.

The bidirectional relationship has important clinical implications. First, the presence of AR should prompt evaluation for asthma, particularly in patients with more severe or persistent AR symptoms. Second, the presence of asthma should trigger systematic assessment for AR, as AR treatment may improve asthma outcomes. Third, the amplification of each condition by the other suggests that optimal management requires integrated treatment of both upper and lower airways rather than siloed approaches.

Treatment Effects and Implications

The therapeutic relationship between AR treatment and asthma outcomes provides important evidence supporting causal links between these conditions and offers practical guidance for clinical management.

Intranasal Corticosteroids: Multiple studies demonstrated that intranasal corticosteroid (INCS) use is associated with improved asthma outcomes. De Groot et al. (4) found that the effect of AR on incomplete asthma control was mitigated by nasal corticosteroid use in children. Oka et al. (16) demonstrated that AR activity positively correlated with asthma severity, and nasal corticosteroids significantly improved both asthma control questionnaire scores and FeNO levels (all $p<0.001$) in patients with atopy. Ko et al. (13) showed that nasal steroid use was associated with lower emergency department visits and hospitalizations in Hong Kong adults. Anna et al. (58) in a randomized controlled context found that nasal steroids improved both rhinitis and asthma symptoms in children with eosinophilic airway inflammation, with associated reductions in FeNO.

The mechanism by which INCS improves asthma outcomes likely involves multiple pathways: reduction of nasal inflammation decreasing systemic inflammatory burden, improved nasal breathing reducing mouth breathing and its adverse effects on lower airways, and treatment of

shared inflammatory processes affecting both upper and lower airways (67,68). The finding that INCS effects are observed in both objective measures (FeNO, lung function) and subjective measures (symptom scores, quality of life) supports the biological plausibility of these mechanisms.

Antihistamines and Combination Therapy: The evidence for antihistamines alone is less robust than for INCS, though combination therapy may offer advantages. The systematic review by Tameeris et al. (79) of 33 randomized controlled trials (n=5,987) found that antihistamines and corticosteroids showed positive effects on quality of life and objective asthma outcomes, while leukotriene receptor antagonists did not show significant improvements. This differential treatment response may reflect the predominant type-2 inflammatory pathway in AR-asthma, which responds more robustly to corticosteroids and antihistamines than to leukotriene modifiers.

Allergen-Specific Immunotherapy: Several studies support the role of allergen-specific immunotherapy (SIT) in modifying the AR-asthma relationship. Marogna et al. (36) found that SIT reduced allergic progression from AR to asthma in house dust mite allergic patients. Viveiros et al. (41) reported that specific immunotherapy reduced treatment needs and severity in patients with AR and asthma. Agache et al. (80) identified lack of SIT as an independent risk factor for asthma in both children ($p=0.008132$) and adults ($p=0.000017$) with seasonal AR. These findings suggest that SIT may modify the natural history of allergic airway disease, potentially preventing progression from AR to asthma and reducing severity in those with established comorbid disease.

Treatment as Effect Modifier: The observation that AR treatment modifies the association between AR and asthma severity has important methodological implications for observational studies. Studies that fail to account for AR treatment status may underestimate the true association between AR and asthma severity, as effectively treated AR may attenuate its impact on asthma. Conversely, studies that include patients with optimally treated AR may show weaker associations than those including untreated or undertreated patients. Future studies should systematically collect data on AR treatment type, adherence, and response to enable appropriate adjustment for these important effect modifiers.

Mechanistic Insights

The association between AR and asthma severity is mediated through multiple interconnected mechanisms that have been elucidated through biomarker studies and physiological assessments.

Type-2 Inflammation: The predominant mechanism linking AR and asthma is shared type-2 inflammation. Kuo et al. (14) demonstrated that patients with unified airway disease had higher FeNO ($p=0.004$) and blood eosinophils ($p=0.005$), indicating greater type-2 inflammatory burden. Kawamatawong et al. (43) found increased FeNO and blood eosinophils in Thai adults with asthma regardless of rhinitis status, but blood eosinophils differentiated AR from non-allergic rhinitis ($p=0.02$). Castillo Vizuete et al. (51) identified that the "triple type-2 signature" (elevated FeNO, blood eosinophils, and IgE) was associated with severe asthma and chronic rhinosinusitis with nasal polyps comorbidity. Vijayan et al. (72) found that elevated absolute eosinophil count (OR=15) and serum IgE (OR=10) were associated with co-existing asthma in AR patients.

The amplification of type-2 inflammation in patients with both conditions suggests that the total inflammatory burden across the united airway exceeds that of either condition alone. This "inflammatory sum" concept has implications for treatment, suggesting that controlling inflammation in both upper and lower airways may be necessary to achieve optimal disease control.

Systemic Inflammatory Spillover: The concept of systemic inflammatory spillover proposes that inflammatory mediators generated in the nasal mucosa enter the systemic circulation and propagate inflammation to the lower airways. Oka et al. (16) provided supportive evidence by demonstrating that ongoing AR activity positively correlated with asthma severity and that nasal corticosteroids improved both upper and lower airway outcomes. The reduction in FeNO following nasal corticosteroid treatment (16,58) suggests that controlling nasal inflammation reduces overall airway inflammatory burden.

Naso-Bronchial Reflex: Neural connections between the upper and lower airways provide a pathway for reflex bronchoconstriction triggered by nasal irritation. The rapid onset of asthma symptoms following nasal allergen challenge, before systemic absorption could occur, supports the role of neural reflexes. The improvement in asthma outcomes with nasal corticosteroid treatment, even in the absence of systemic absorption, further supports neural mechanisms.

Impaired Nasal Function: Mouth breathing secondary to nasal obstruction bypasses the physiological functions of the nose in warming, humidifying, and filtering inspired air. This exposes the lower airways to unconditioned air, which can trigger bronchoconstriction, particularly in patients with pre-existing airway hyperresponsiveness. Chawes et al. (31) demonstrated that AR was associated with nasal eosinophilia and irreversible nasal obstruction in young children, with strong associations between upper and lower airway patency.

Shared Environmental Triggers: Common environmental exposures, particularly aeroallergens, trigger inflammation throughout the united airway. Giniş et al. (15) demonstrated the seasonal effect in children with pollen allergy, showing parallel increases in nasal symptoms, asthma symptoms, and lung function impairment during pollen season. This temporal concordance supports the role of shared environmental triggers in driving both upper and lower airway disease expression.

Effect Modifiers and Confounders

Multiple factors modify the association between AR and asthma severity, and understanding these modifiers is essential for accurate interpretation of study findings and for clinical application.

Treatment Status: As previously discussed, AR treatment significantly modifies the association. Studies that include patients on optimal AR therapy may underestimate the true association, while those including untreated patients may show stronger effects. The finding by de Groot et al. (4) that nasal corticosteroids mitigated the effect of AR on asthma control illustrates this modifying effect.

Sensitization Patterns: The number and type of allergen sensitizations modify the association. Polysensitization is associated with greater severity than monosensitization (25,56,73). Indoor allergen sensitization (house dust mites, animal dander) appears more strongly associated with asthma severity, while outdoor allergen sensitization (pollens) is more associated with rhinitis severity (35,47). Cosensitization to food and inhalant allergens further increases severity (48).

Comorbidities: Other comorbid conditions modify the AR-asthma relationship. Chronic rhinosinusitis with nasal polyps (CRSwNP) emerges as a particularly important effect modifier. Castillo et al. (28,44) demonstrated that CRSwNP was associated with severe asthma (48%,

p<0.05), with sinus occupancy affecting asthma control (r=0.249, p=0.034). Wechsler et al. (61) found that patients with CRS±NP experienced 23% fewer exacerbations (95% CI 10-35%, p<0.001) post-biologic treatment, suggesting that upper airway disease modifies biologic effectiveness. Sinusitis was also associated with increased exacerbations in the study by Dixon et al. (69). Atopic dermatitis may paradoxically be associated with decreased risk of asthma-related hospital visits (59), potentially reflecting different endotypes or treatment effects.

Environmental Factors: Allergen exposure levels, air pollution, tobacco smoke exposure, and climate factors all potentially modify the AR-asthma relationship. The seasonal variations documented by Giniş et al. (15) demonstrate the impact of allergen exposure patterns. Urban versus rural differences in exposure profiles may also influence disease expression (2,12,71).

Demographic Factors: Age, sex, and socioeconomic status modify the association. The age effects previously discussed, including stronger associations in school-age children (9) and younger adults (1), and declining prevalence in elderly (18), demonstrate age as an important effect modifier. Sex differences were noted by Adamia et al. (37) who found boys more susceptible to asthma and AR (p=0.001). Socioeconomic factors influence healthcare access, treatment adherence, and environmental exposures, potentially modifying disease expression.

Confounders Controlled: The quality of confounder control varies across studies. Well-conducted studies adjusted for demographic factors (age, sex), clinical factors (asthma duration, comorbidities, medications), and environmental factors (smoking, allergen exposure) using multivariable regression, propensity scores, or other methods. However, residual confounding remains a concern, particularly from unmeasured factors such as treatment adherence, healthcare-seeking behavior, and detailed environmental exposures. The observational nature of most included studies precludes definitive causal inference, though the consistency of findings, dose-response relationships, and supportive evidence from treatment studies strengthen the case for causality.

Clinical Implications

The findings of this systematic review have important implications for clinical practice across multiple specialties.

For Primary Care: AR should be systematically assessed in all patients with asthma using validated questionnaires and, where available, objective testing. The high prevalence of AR in asthmatic populations (60-85%) and its consistent association with worse asthma outcomes justify routine screening. Patients with poorly controlled asthma despite appropriate inhaled therapy should be evaluated for unrecognized or undertreated AR as a potential contributing factor. Conversely, patients presenting with moderate-to-severe or persistent AR should be assessed for undiagnosed asthma, particularly those with nocturnal symptoms, exercise-induced symptoms, or a history of wheeze.

For Specialists: Pulmonologists and allergists should integrate upper and lower airway assessment in their evaluation of patients with respiratory symptoms. The ARIA guidelines' recommendation to treat both conditions in an integrated manner should be operationalized through collaborative care models or comprehensive airway clinics. Patients with severe asthma, frequent exacerbations, or persistent symptoms despite high-intensity treatment should undergo detailed rhinology assessment to identify treatable upper airway traits (44). The identification of specific AR phenotypes (persistent, moderate-to-severe, polysensitized) that confer greatest asthma severity risk can guide treatment prioritization.

Treatment Approach: The evidence supports a stepwise approach to AR treatment in patients with asthma. Intranasal corticosteroids should be considered first-line therapy, given their documented benefits on both upper and lower airway outcomes (4,13,16,58). For patients with inadequate response, combination with antihistamines or addition of leukotriene receptor antagonists may be considered, though evidence for add-on benefit is less robust (79). For appropriately selected patients (monosensitized, persistent symptoms, evidence of clinical relevance), allergen-specific immunotherapy may modify disease progression and reduce severity (36,41,80). Treatment adherence should be assessed and addressed, as medication possession alone may not reflect effective disease management.

Monitoring: Patients with AR-asthma comorbidity may require more frequent monitoring and lower thresholds for treatment escalation. The bidirectional amplification of disease severity suggests that deterioration in one condition should prompt evaluation of the other. Objective

biomarkers (FeNO, blood eosinophils, IgE) may help identify patients with high type-2 inflammatory burden who might benefit from intensified anti-inflammatory therapy or biologic agents targeting type-2 pathways (51,61).

Pediatric Considerations: The strong association between AR and asthma severity in school-age children, coupled with evidence that childhood AR predicts asthma persistence to middle age (17), supports early and aggressive management of AR in children. The absence of association in children under 6 years (9) should not preclude AR evaluation, as diagnostic challenges in this age group may obscure true relationships. Treatment of AR in children may modify the natural history of allergic airway disease and should be prioritized.

Elderly Considerations: In elderly patients, the AR-asthma association may be attenuated but remains clinically significant (42). Diagnostic challenges include distinguishing AR from other causes of rhinitis in this age group. Treatment should account for potential drug interactions, comorbidities, and age-related changes in drug metabolism.

Future Research Directions

Based on the identified knowledge gaps and methodological limitations, several priorities for future research emerge.

Prospective Cohort Studies: Well-designed prospective cohort studies with repeated measures over ≥5 years should be conducted in children with newly diagnosed AR (ages 5-12 years) to determine whether AR severity predicts subsequent asthma onset, and whether early intensive AR treatment prevents or delays asthma development. Such studies should assess asthma outcomes at regular intervals using standardized instruments (ACT, spirometry, FeNO, exacerbation rates) and should be adequately powered (≥300 participants per treatment arm) to detect clinically meaningful differences. Existing infrastructure in school-based programs (12,71) and allergy specialty clinics could support such studies.

Randomized Controlled Trials: Trials of AR treatment versus usual care in adults with established asthma and comorbid AR should evaluate whether protocolized AR management (starting with intranasal corticosteroids, escalating based on ARIA severity classification) improves asthma control compared to usual care over 12-24 months. Primary outcomes should include time

to first severe exacerbation, change in ACT score, and oral corticosteroid courses required. Trials should stratify randomization by AR severity and asthma severity to enable subgroup analyses. Sample sizes of 200-300 per arm would provide 80% power to detect moderate effect sizes.

Multinational Observational Studies: Studies with standardized protocols should characterize the AR-asthma severity association across diverse populations in underrepresented regions (sub-Saharan Africa, Latin America, Southeast Asia), using identical case definitions (GINA for asthma, ARIA for AR), measurement instruments (validated translations of ACT, spirometry to ATS standards), and allergen panels (including region-specific allergens). Target samples of ≥500 per region would clarify whether geographic differences reflect true variation versus methodological artifact.

Mechanistic Substudies: Nested within treatment trials or large cohorts, comprehensive phenotyping (sputum or nasal lavage eosinophil counts, serum IgE levels, multiplexed cytokine panels, exhaled breath analysis) should identify biomarker profiles predicting response to different treatment intensities or modalities. Patients with high type-2 signatures (high FeNO, blood eosinophils >300 cells/μL, total IgE >100 IU/mL) (51) might benefit from biologics targeting type-2 inflammation. Sample sizes of 100-150 participants with deep phenotyping could yield hypothesis-generating findings.

Comparative Effectiveness Research: Using large healthcare databases or registries, real-world treatment patterns and outcomes should be evaluated, comparing outcomes among patients receiving different AR treatment intensities while accounting for confounding through propensity score methods, instrumental variable analysis, or regression discontinuity designs. Existing asthma registries (10,61) could be enhanced by incorporating systematic AR assessment.

Methodological Harmonization: Future studies should adopt standardized definitions and outcome measures to facilitate cross-study comparisons and meta-analysis. The ARIA and GINA frameworks provide established classification systems that should be consistently applied. Differentiation between allergic and non-allergic rhinitis should be mandatory, using objective confirmation where feasible.

Long-Term Follow-up: Extended follow-up of existing cohorts could provide valuable insights into the natural history of AR-asthma comorbidity across the lifespan. The Tasmanian Asthma Study (17) demonstrates the value of long-term follow-up in understanding disease trajectories.

CONCLUSION AND RECOMMENDATIONS

Summary of Key Findings

This comprehensive systematic review of 80 studies provides robust evidence that allergic rhinitis is associated with increased severity of coexisting asthma in both children and adults. The major findings can be summarized as follows:

1. **High Prevalence:** Allergic rhinitis is present in the majority of patients with asthma, with prevalence ranging from 55-97% in well-conducted studies, substantially exceeding background population rates and supporting the concept of united airways disease.

2. **Consistent Association:** Across diverse populations, study designs, and geographic regions, AR is consistently associated with increased asthma severity manifested through poorer asthma control (OR 1.21-2.74), more frequent exacerbations (incidence rate ratio 1.12), increased healthcare utilization (OR 2.64-2.98 for emergency visits), impaired lung function (lower FEV_1, $FEF_{25–75}$, higher FeNO), and reduced quality of life.

3. **Dose-Response Relationship:** AR severity correlates positively with asthma severity (correlation coefficients 0.365-0.689), with persistent and moderate-to-severe AR phenotypes conferring greatest risk. Polysensitization and specific sensitization patterns (indoor allergens, cosensitization to food allergens) are associated with more severe disease.

4. **Age-Specific Patterns:** The association is consistently demonstrated across the lifespan but shows age-dependent variations. Strong associations are evident in school-age children (6-14 years) and younger adults, while associations may be attenuated in children under 6 years (potentially due to diagnostic challenges) and in elderly populations (possibly due to

immune senescence or cohort effects). Childhood AR predicts asthma incidence and persistence to middle age.

5. **Bidirectional Relationship:** AR and asthma demonstrate bidirectional influences, with each condition amplifying the severity of the other. AR frequently precedes asthma onset, while the presence of asthma is associated with more severe and persistent AR expression.

6. **Treatment Responsiveness:** AR treatment, particularly with intranasal corticosteroids, is associated with improved asthma outcomes including better control, reduced exacerbations, decreased healthcare utilization, and improved lung function. Allergen-specific immunotherapy may modify disease progression and reduce severity.

7. **Mechanistic Pathways:** The AR-asthma severity association is mediated through shared type-2 inflammation (elevated FeNO, blood eosinophils, IgE), systemic inflammatory spillover, naso-bronchial reflexes, impaired nasal function leading to mouth breathing, and shared environmental triggers.

8. **Effect Modifiers:** The association is modified by AR treatment status, sensitization patterns, comorbidities (particularly chronic rhinosinusitis with nasal polyps), environmental factors, and demographic characteristics. These modifiers should be considered in clinical assessment and research design.

Clinical Recommendations

Based on the synthesized evidence, the following clinical recommendations are warranted:

1. **Universal Screening:** All patients with asthma should be systematically assessed for AR using validated questionnaires and, where available, objective testing (skin prick tests or specific IgE). Conversely, patients with moderate-to-severe or persistent AR should be evaluated for undiagnosed asthma.

2. **Integrated Management:** AR and asthma should be managed as manifestations of a unified airway disease, with treatment addressing both upper and lower airway inflammation. This

requires collaboration between primary care, pulmonology, allergy, and otorhinolaryngology specialties.

3. **Treatment Prioritization:** Intranasal corticosteroids should be considered first-line therapy for AR in patients with asthma, given their documented benefits on both upper and lower airway outcomes. Treatment should be optimized based on AR severity and phenotype, with escalation to combination therapy or allergen-specific immunotherapy when indicated.

4. **Monitoring Intensity:** Patients with AR-asthma comorbidity may require more frequent monitoring and lower thresholds for treatment escalation, particularly those with persistent, moderate-to-severe AR or polysensitization. Deterioration in one condition should prompt evaluation of the other.

5. **Pediatric Focus:** Given that childhood AR predicts asthma incidence and persistence to middle age, early recognition and optimal management of AR in children may modify the natural history of allergic airway disease and should be prioritized.

6. **Biomarker-Guided Therapy:** Assessment of type-2 biomarkers (FeNO, blood eosinophils, IgE) may identify patients with high inflammatory burden who could benefit from intensified anti-inflammatory therapy or biologic agents targeting type-2 pathways.

Research Recommendations

Priority areas for future research include:

1. Well-designed prospective cohort studies in children to determine whether early intensive AR treatment prevents or delays asthma development

2. Randomized controlled trials of protocolized AR management versus usual care in adults with established asthma and comorbid AR

3. Multinational observational studies using standardized protocols in underrepresented regions (sub-Saharan Africa, Latin America, Southeast Asia)

4. Mechanistic substudies to identify biomarker profiles predicting treatment response
5. Comparative effectiveness research using large healthcare databases to evaluate real-world treatment outcomes
6. Harmonization of definitions and outcome measures across future studies to facilitate cross-study comparisons and meta-analysis
7. Long-term follow-up of existing cohorts to understand disease trajectories across the lifespan

Final Conclusion

In conclusion, this comprehensive systematic review provides compelling evidence that allergic rhinitis is associated with increased severity of coexisting asthma in both children and adults. The consistency of findings across diverse populations, the dose-response relationship between AR severity and asthma severity, the bidirectional nature of the association, and the improvement in asthma outcomes following AR treatment all support a causal relationship mediated through shared inflammatory pathways and interconnected physiological mechanisms. Systematic assessment and optimal management of AR should be integrated into routine asthma care to potentially improve asthma outcomes, reduce exacerbations, decrease healthcare utilization, and enhance quality of life for the millions of patients worldwide living with these common comorbid conditions. The united airway concept should guide clinical practice, research priorities, and healthcare policy to ensure comprehensive management of allergic airway disease across the lifespan.

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