Review

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Rhinosinusitis as a Modifiable Determinant of Asthma Control in Children: A Narrative Review

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Abstract

Paediatric rhinosinusitis (RS), particularly chronic rhinosinusitis (CRS), is a common inflammatory condition with a significant impact on quality of life and a well-recognized association with asthma within the framework of united airway disease. This review aims to evaluate the impact of RS on asthma control in children and explore its role as a modifiable determinant. Mechanistically, RS and asthma share key pathophysiological features, including type 2 inflammation, epithelial barrier dysfunction, and airway microbiome dysbiosis, supporting the concept of a unified inflammatory process across the respiratory tract. Clinically, epidemiological data demonstrate a high prevalence of coexisting RS and asthma, with consistent associations with poorer asthma control, increased disease severity, and higher exacerbation burden, even in cases of subclinical sinonasal inflammation. Emerging evidence suggests that appropriate management of CRS, including medical therapy and, in selected cases, surgical intervention, may improve asthma outcomes such as symptom control and lung function. However, the current evidence base remains limited, with a predominance of small, heterogeneous, and observational studies. RS therefore represents a potentially treatable trait in paediatric asthma, warranting systematic evaluation in children with difficult-to-treat disease. Further prospective and interventional studies are needed to clarify causality and define its impact on long-term outcomes.

Keywords: paediatric rhinosinusitis; chronic rhinosinusitis; asthma; united airway disease; asthma control; type 2 inflammation; epithelial barrier dysfunction; airway microbiome; comorbidities; treatable traits

1. Introduction

Paediatric rhinosinusitis (RS), according to European Position Paper on Rhinosinusitis and Nasal Polyps 2020, is an inflammatory condition of the nose and paranasal sinuses characterized by the presence of at least two symptoms, one of which must be either nasal blockage/obstruction/congestion or nasal discharge (anterior or posterior nasal drip), with or without facial pain/pressure and often including cough in children. The diagnosis is further supported, when available, by endoscopic signs such as mucopurulent discharge, oedema, or nasal polyps, or by changes seen on computed tomography (CT). Acute RS (ARS) in children is defined by the sudden onset of these symptoms lasting less than 12 weeks, with complete resolution between episodes if recurrent, whereas chronic RS (CRS) is defined by the persistence of symptoms for 12 weeks or more [1].

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Although paediatric CRS has been less extensively studied, its prevalence appears to be lower than in adults; its detrimental effect on quality of life is comparable to that seen in the adult population. It is estimated to affect approximately 2.1% to 4% of the paediatric population, with the highest prevalence observed among those aged 10 to 15 years [1–3].

Asthma is a heterogeneous disease characterized by chronic airway inflammation and a history of respiratory symptoms (wheeze, shortness of breath, chest tightness, or cough) that vary over time and are associated with variable airflow limitation [4]. The global prevalence of asthma in children and adolescents is approximately 4,300 per 100,000 population (≈ 4.3%), with higher rates in males and a peak in school-aged children [5]. Asthma control refers to the extent to which the manifestations of asthma are minimized and therapeutic goals are achieved, encompassing both current symptom control and the reduction of future risk, including exacerbations, lung function decline, and treatment-related adverse effects [4].

The concept of united airway disease (UAD) describes the upper and lower airways as a single, continuous system sharing common anatomical, epithelial, and immunological characteristics, whereby inflammatory processes affect the entire respiratory tract rather than isolated compartments. Within this framework, CRS and asthma are closely interconnected conditions that frequently coexist, with evidence indicating that inflammation in the upper airway can influence the lower airway and vice versa, contributing to disease severity and highlighting the need for an integrated diagnostic and therapeutic approach [6].

Although recent studies support a causal relationship between paediatric asthma and UADs, the clinical implications of these interactions remain insufficiently defined, particularly regarding whether upper airway inflammation, such as CRS, influences asthma control or represents a modifiable therapeutic target [7,8].

This study aims to evaluate the impact of RS on asthma control in children and explore its potential as a modifiable determinant.

2. Materials and Methods

A narrative review was conducted using the electronic databases PubMed/MEDLINE, Scopus, and Google Scholar for studies published in English up to April 2026. The search focused on studies evaluating the epidemiological, pathophysiological, and clinical association between rhinosinusitis and asthma in paediatric populations, as well as the potential impact of rhinosinusitis treatment on asthma-related outcomes.

The search strategy applied across databases was as follows: (("chronic rhinosinusitis" OR "paediatric rhinosinusitis") AND ("asthma" OR "asthma control") AND ("children" OR "paediatric" OR "pediatric")) AND ("united airway disease" OR "type 2 inflammation" OR "epithelial barrier dysfunction" OR "airway microbiome" OR "comorbidities" OR "treatable traits").

Titles and abstracts were independently assessed for relevance by two reviewers, followed by full-text evaluation of the selected studies. Any disagreements regarding study eligibility or data interpretation were resolved through discussion and consensus.

Original articles, observational studies, clinical studies, review articles, and relevant international guideline documents with particular emphasis on paediatric populations were considered eligible for inclusion. Publications not available in English, conference abstracts without an accessible full text, and studies not directly relevant to the objectives of the review were excluded from consideration.

3. Pathophysiological Link Between Rhinosinusitis and Asthma

The unified airway hypothesis posits that upper and lower airway diseases represent manifestations of a single pathological process occurring at different sites within a continuous respiratory system, supported by shared structural, immunological, and epidemiological features, including common epithelial architecture and frequent comorbidity between conditions such as

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asthma and CRS with nasal polyps (CRSwNP) [9–11]. Furthermore, accumulating pathophysiological and clinical evidence highlights the central role of type 2 inflammation, particularly eosinophils and interleukin - IL-5, in driving disease across both airway compartments, with therapeutic targeting of these pathways demonstrating parallel clinical benefits in upper and lower airway disease, thereby reinforcing their biological and clinical interconnection [11].

In children with CRS, inflammatory mediators are broadly upregulated (34/40 cytokines in sinus tissue and 36/40 in adenoids), with further amplification in the presence of asthma (27/40 and 39/40, respectively), indicating a quantitatively intensified but qualitatively similar inflammatory process across the unified airway [12]. This pattern is paralleled at the transcriptomic level, where analyses reveal approximately 91% overlap in gene expression between upper and lower airway epithelium, along with conserved biological pathways and gene networks involved in immune responses, metabolism, and cellular function. Moreover, disease-associated gene signatures linked to wheeze and atopy are consistently observed in both nasal and tracheal epithelium, supporting the concept that airway inflammation extends across compartments and reflects a unified pathophysiological process in children [13].

At the core of these shared inflammatory processes lies epithelial barrier dysfunction, with the sinonasal epithelium functioning as an active immunologic interface rather than a passive barrier. Disruption of epithelial integrity results in increased ion permeability, reduced tight junction integrity, enhanced exposure to environmental antigens, and impaired mucociliary clearance [14]. In addition, epithelial cells actively propagate inflammation through the release of cytokines and chemokines, including thymic stromal lymphopoietin (TSLP), IL-25, and IL-33, which promote and amplify downstream immune responses [15].

Within this disrupted epithelial environment, the established T2 inflammatory axis, driven by IL-4, IL-5, and IL-13, further amplifies disease expression, promoting eosinophilic infiltration and contributing to abnormal tissue remodeling, including dysregulated fibrin deposition [16,17]. Concurrently, microbial–immune interactions, particularly involving Staphylococcus aureus biofilms and enterotoxin-mediated superantigen activity, amplify Th2-driven responses and immunoglobulin E (IgE) production. In parallel, strong molecular correlations between the upper and lower airways further reinforce the concept that these conditions represent interconnected manifestations of a UAD process [17,18].

Beyond localized host–microbe interactions, airway microbiome dysbiosis represents an additional integrative mechanistic layer linking CRS and asthma. It is characterized by reduced microbial diversity and shifts in community composition that alter host–microbe interactions and immune regulation. Rather than acting as an isolated trigger, dysbiosis participates in a bidirectional feedback loop with epithelial dysfunction and type 2 inflammation, while emerging evidence also suggests systemic effects through gut–airway interactions, collectively contributing to a shared inflammatory milieu across the respiratory tract [19]. This concept is further supported by prospective data showing that, in children with CRS, asthma persisted in 23% of cases over a 5-year follow-up, with persistence strongly associated with atopy (OR – odds ratio: 8.5) and reduced airway microbiome diversity (OR: 6.0), highlighting the synergistic role of dysbiosis and type 2 inflammation in sustaining lower airway disease [20].

Table 1 illustrates the pathophysiological mechanisms connecting RS with asthma, while Figure 1 depicts how CRS and asthma are linked via epithelial barrier dysfunction and type 2 inflammation, enabling bidirectional airway interactions that worsen control and severity.

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Table 1. Key pathophysiological mechanisms linking RS and asthma.

Study	Population	RS Type	Asthma Outcome	Supporting evidence
Type 2 inflammation and immune amplification	IL-4, IL-5, IL-13, eosinophils, IgE, superantigens	Mucosal inflammation, polyp formation	Airway inflammation, hyperresponsiveness	[11,16,17]
Epithelial barrier dysfunction	TSLP, IL-25, IL-33	Increased permeability, impaired mucociliary clearance	Enhanced allergen penetration, inflammation	[14,15]
Microbiome dysbiosis	Reduced diversity, dysbiosis	Chronic inflammation, immune dysregulation	Persistence and poor asthma control	[19,20]
Transcriptomic overlap	Shared gene expression (~91%)	Common epithelial and immune pathways	Unified inflammatory response	[13]

RS: rhinosinusitis, IL: interleukin, IgE: immunoglobulin E, TSLP: thymic stromal lymphopoietin.

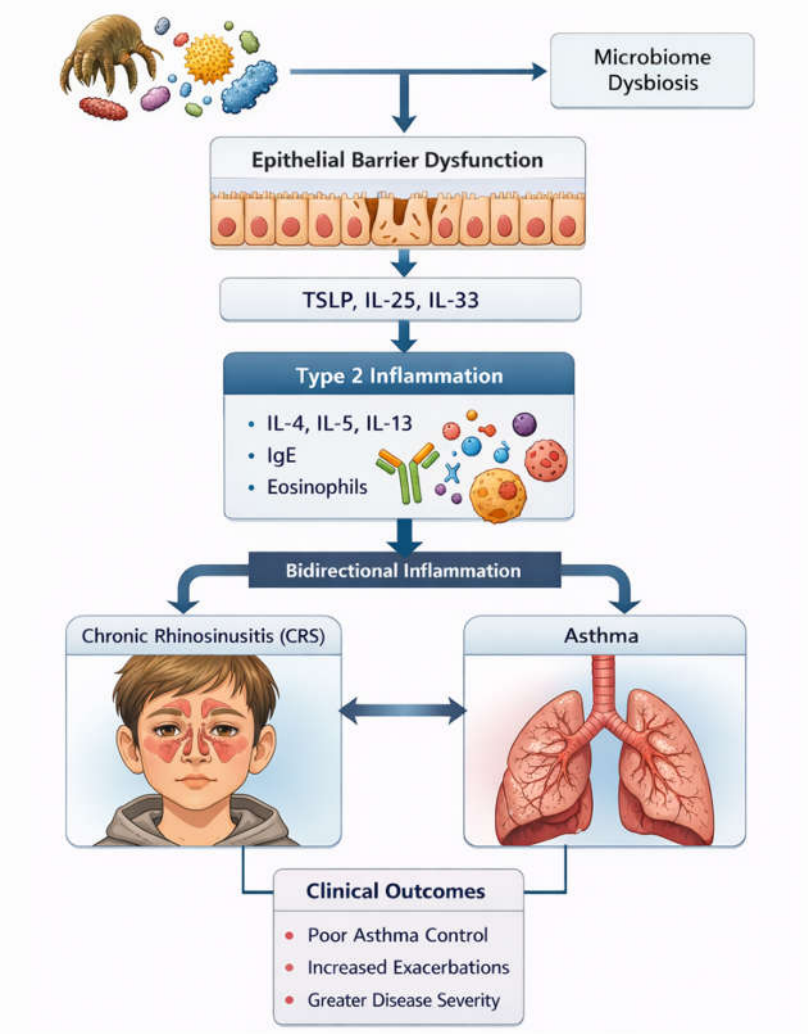

Figure 1. The unified airway model in children. Epithelial barrier dysfunction and type 2 inflammation (TSLP, IL-25, IL-33; IL-4, IL-5, IL-13, IgE, eosinophils) link chronic rhinosinusitis (CRS) and asthma through bidirectional airway interactions, contributing to poor disease control and increased severity; generated with assistance from ChatGPT (OpenAI) and reviewed by the authors.

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4. Epidemiology and Clinical Association of Rhinosinusitis and Asthma Control

RS is a clinically relevant comorbidity in paediatric asthma, associated with poorer disease control and increased airway obstruction, and its appropriate management has been shown to improve asthma outcomes [3,6,21–25]. Table 2 shows the epidemiological and clinical association between RS and asthma in children.

Epidemiological data indicate a substantial overlap between upper and lower airway disease in childhood. RS and asthma coexist in approximately 35–65% of children, and sinus abnormalities are present in up to 40% children with asthma, with higher prevalence in severe disease [22]. Previous data showed that in children with CRS, asthma was present in 18.1%, and was significantly more common in those with allergic rhinitis (AR) (40.7% vs 9.8%). Asthma was strongly associated with AR (OR: 6.24, 95% CI (confidence interval): 5.27–7.39, $p < 0.001$), indicating that the asthma-CRS relationship is largely mediated through AR rather than being direct [26]. In Choi's study, recurrent RS (defined as ≥3 episodes/year) and chronic RS were associated with higher asthma prevalence, with asthma present in 37.5% of chronic/recurrent RS vs 15.9% in acute/subacute RS, and 62.3% of children with asthma had chronic or recurrent RS [27].

Recent evidence confirms that RS is associated with worse asthma control, increased severity, and higher disease burden. A cross-sectional study reported a prevalence of allergic RS of 48.8% (60/123) among children with asthma and demonstrated a significant association with poor asthma control, observed in 66.7% of allergic RS - positive compared with 31.7% of allergic RS - negative children (OR: 2.4; 95% CI: 1.5–3.9; p=0.0002) [28]. A prospective observational study showed that allergic RS (59.2%) and adenoid hypertrophy (71%) are highly prevalent in children with asthma and are strongly associated with increased asthma severity. Importantly, both conditions, and especially their coexistence, were identified as independent predictors of severe asthma (allergic RS – OR: 7.35, $p = 0.004$; adenoid hypertrophy – OR: 12.65, $p = 0.001$; combined – OR: 98.65, $p < 0.001$), highlighting their substantial impact on disease burden [29]. In the study by Nyenhuis et al., RS in children was identified as an independent comorbidity associated with poorer asthma outcomes. Specifically, pediatric patients with RS were at higher risk of uncontrolled asthma (OR: 1.15; 95% CI: 1.01–1.31) and increased asthma severity (OR: 1.28; 95% CI: 1.07–1.53). Additionally, RS was more prevalent among children with more severe disease, particularly those managed by specialists (46.5% vs 19.3% in primary care) [30].

Prior data found that in patients aged 12–64 years with moderate-to-severe asthma, 34.2% experienced ≥1 exacerbation, and those patients had higher rates of comorbid upper airway diseases, including acute sinusitis (24.4% vs 16.4%), chronic sinusitis (14.0% vs 7.6%), and AR (38.9% vs 33.9%) compared to those without exacerbations. These patients also demonstrated poorer asthma control, reflected by more frequent systemic corticosteroid use (44.5% vs 20.5%) and a greater overall disease burden [31]. In Marseglia et al.'s study, among 294 children with asthma, 21 with uncontrolled asthma showed endoscopic evidence of sinusitis despite lacking clinical symptoms, indicating the presence of occult sinusitis. These findings suggest that subclinical sinonasal inflammation may contribute to poor asthma control and support the use of targeted endoscopic evaluation in selected patients [32].

In the Kang et al. adult study, 23.8% (15/63) of patients with CRS without nasal polyps (CRSsNP) and 26.9% (21/78) of patients with CRSwNP had concomitant asthma. In CRSsNP, asthma prevalence increased with disease extent (from 0% in single sinusitis to 58.3% in pansinusitis), and was significantly associated with sinus severity ($p = 0.049$), while pulmonary function impairment also correlated with inflammation severity ($p = 0.019$) [33].

Conversely, asthma may also influence the severity and clinical course of sinonasal disease. In Murtomäki et al.'s study of children with CRS, asthma was present in 47.1% (49/104) of cases, indicating a high burden of comorbidity between upper and lower airway disease. Moreover, asthma was identified as a significant predictor of disease severity, being associated with an increased risk of revision surgery after endoscopic sinus surgery (HR (hazard ratio): 2.3; 95% CI 1.5–3.4; $p < 0.0001$), highlighting its impact on disease persistence and control [34].

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Table 2. Epidemiological and clinical association between RS and asthma in children.

Study	Population	RS Type	Asthma Outcome	Key Results
Choi et al. [27]	Children with RS (n=296)	Chronic/Recurrent vs ARS/Subacute RS	Prevalence	Asthma prevalence by RS type: acute 12.5%, subacute 17.1%, recurrent 32.7%, and chronic 23.6%. 37.5% of children with asthma were in the chronic/recurrent group.
Nyenhuis et al. [30]	Children with asthma (n=13,479), adolescents and adults (n=15,029)	RS (unspecified) (as comorbidity)	Control and severity	Poor control in children (OR: 1.15); increased severity in children (OR: 1.28).
Manoj and Sanjay [28]	Children with asthma (n=123)	Allergic RS (as comorbidity)	Control	Poor control: 66.7% (allergic RS positive) versus 31.7% (allergic RS negative) (OR: 2.4; 95% CI 1.5–3.9; p = 0.0002).
Aroor et al. [29]	Children with asthma (n=76)	Allergic RS ± adenoid Hypertrophy (as comorbidity)	Severity	RS (OR: 7.35; 95% CI 1.904–28.405; p=0.004); adenoids (OR: 12.65); combined (OR: 98.65; p < 0.001).
Sedaghat et al. [26]	Children with CRS (n=4,044)	CRS	Prevalence	Asthma prevalence in CRS: 18.1%; 40.7% vs 9.8% (with vs without AR); OR: 6.24; 5% CI 5.27–7.39, p < 0.001.
Murtomäki et al. [34]	Children (n=104) and adults with CRS	CRS (CRSsNP, CRSwNP)	Prevalence	Asthma in 47.1%; increase of revision surgery risk (HR: 2.3).
Marseglia et al. [32]	Children with asthma (n=294)	Occult RS (as comorbidity)	Prevalence	RS detected in uncontrolled

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	asthma subgroup (n=21).

RS: rhinosinusitis, OR: odds ratio, ARS: acute rhinosinusitis, CRS: chronic rhinosinusitis, Cl: confidence interval, AR: allergic rhinitis, CRSsNP: chronic rhinosinusitis without nasal polyps, CRSwNP: chronic rhinosinusitis with nasal polyps, HR: hazard ratio.

5. Clinical Management of CRS and Its Impact on Asthma Control

RS represents a clinically relevant comorbidity in paediatric asthma and should be considered during routine assessment, particularly in children with persistent symptoms or frequent exacerbations. Current guidelines recommend systematic evaluation of comorbid conditions as part of asthma management, given their potential to influence disease control. Specifically, all children with difficult-to-treat or severe asthma require targeted evaluation for contributing comorbidities, particularly CRS, with optimization of their management as part of comprehensive asthma care [4,35]. Early recognition of upper airway involvement is especially important in patients at risk of more severe disease. Confirmation or exclusion of sinonasal pathology requires assessment by an ear, nose, and throat (ENT) specialist, including nasal endoscopy and, when indicated, imaging [23,32]. Once sinonasal involvement has been identified, appropriate management is required, as optimal treatment of CRS may improve asthma outcomes.

EPOS 2020 recommends that management of paediatric CRS is primarily medical, with saline nasal irrigation and intranasal corticosteroids as first-line therapy, while evidence for antibiotics and most adjunctive treatments remains limited. In children who fail appropriate medical treatment, adenoidectomy (± sinus irrigation) is considered the initial surgical approach, whilst endoscopic sinus surgery is reserved for selected or refractory cases following evaluation of comorbidities and underlying conditions [1].

Evidence on the impact of CRS treatment on asthma control in children is limited but suggests potential benefit. Treatment of concomitant CRS has been associated with significant improvement in asthma outcomes, including increased lung function (Forced Expiratory Volume in one second - FEV_1, $p < 0.001$) and symptom reduction ($p < 0.001$), with all participants shifting from moderate to mild or intermittent asthma within one month [36]. In adults, similar findings have been reported, with reductions in hospitalizations (from 7 to 2 after surgery and from 5 to 1 after medical therapy) and decreased systemic corticosteroid use (from 10 to 4 and from 7 to 1 course, respectively), alongside modest improvements in lung function (~3–6% FEV_1) and airway inflammation, although a small subset of patients (~8.7%) experienced worsening after surgery [37].

Earlier studies further support this association. Businco et al. demonstrated a significant reduction in asthma severity, with severe cases decreasing from 30 to 10 patients and 36% showing marked clinical improvement ($p < 0.001$) [38]. Friedman et al. reported clinical improvement in 87.5% of children following treatment for bacterial sinusitis, accompanied by improvements in lung function (FEV1: 93% to 116%; mid-expiratory flow rates - MMEFR: 57% to 80%) and an approximately twofold increase in bronchodilator responsiveness [39]. Another retrospective study also supported this relationship, as Parsons et al. showed that following functional endoscopic sinus surgery, asthma symptoms resolved in 58% and improved in 38% of children with asthma, with exacerbations decreasing from 6.7 to 2.5 episodes per month and a 79% reduction in emergency visits [39].

Objective improvements in airway responsiveness have also been demonstrated in smaller mechanistic studies. Oliveira et al. reported a significant increase in methacholine PC20 (0.76 to 2.57 mg/mL, $p < 0.05$), observed only in participants with radiological resolution of sinus disease [40]. At the same time, Tsao et al. confirmed these findings in a prospective study, showing improvements in bronchial hyperresponsiveness (PC20 from 3.68 to 7.31 mg/mL and from 3.11 to 7.00 mg/mL, $p \leq 0.001$) following CRS treatment [41].

However, the current evidence base remains limited, as most studies are small-scale, observational, and relatively outdated, with a marked absence of high-quality randomized controlled trials. Furthermore, there is a lack of contemporary data specifically evaluating the impact of CRS-

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targeted treatment on asthma control, highlighting an important gap in the current literature, which warrants further investigation and underscores the need for well-designed interventional studies in paediatric populations.

6. Knowledge Gaps and Future Directions

Despite increasing recognition of the association between paediatric RS and asthma [28,29], several important gaps remain in the current understanding of their interaction, particularly with respect to causality, disease heterogeneity, and clinical applicability. While observational studies consistently demonstrate an association between RS and impaired asthma control, the direction and magnitude of this relationship remain incompletely defined, and causal inference is limited by the predominance of cross-sectional and non-interventional study designs.

A further limitation relates to disease heterogeneity. Both asthma and RS comprise broad clinical spectra, with variable inflammatory endotypes, severity, chronicity, and treatment response [22,42]. In children, this complexity is compounded by age-related anatomical and immunological differences, the role of adenoids, and the evolving natural history of allergic disease [3,13]. Future studies should therefore aim to distinguish more clearly between ARS and CRS, infectious and allergic phenotypes, and eosinophilic versus non-eosinophilic inflammatory patterns, to determine which subgroups are most strongly linked to impaired asthma control.

Despite the availability of established guideline-based definitions [1,4], their implementation in paediatric research is inconsistent, and standardized, age-specific frameworks integrating upper and lower airway disease are lacking, thereby limiting cross-study comparability and evidence synthesis. Harmonization of these parameters would facilitate more reliable epidemiological estimates and a stronger synthesis of evidence. In parallel, biomarker-driven approaches, including transcriptomic, microbiome, and epithelial barrier studies, may help clarify shared mechanistic pathways and identify children in whom upper airway inflammation is most clinically relevant to lower airway disease.

Finally, interventional research should be prioritized. Well-designed longitudinal and randomized studies are needed to evaluate whether systematic identification and treatment of RS can improve asthma control, reduce exacerbations, decrease medication burden, or alter long-term respiratory outcomes in paediatric populations. Such work would help determine whether RS should be incorporated more explicitly into asthma management algorithms as a treatable trait.

7. Conclusions

Paediatric RS, particularly CRS, represents a clinically relevant and potentially modifiable determinant of asthma control within the unified airway framework. Accumulating epidemiological and mechanistic evidence supports a strong association between RS and increased asthma severity, poorer disease control, and higher exacerbation burden, while emerging data suggest that targeted treatment of CRS may lead to meaningful improvements in asthma outcomes. Despite these findings, the current evidence base remains limited by heterogeneity, small sample sizes, and a lack of high-quality interventional studies, precluding definitive conclusions regarding causality and optimal management strategies. Systematic identification and appropriate treatment of RS should therefore be considered in children with uncontrolled or severe asthma, while further well-designed prospective and randomized studies are needed to clarify its role as a treatable trait and to define its impact on long-term respiratory outcomes.

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