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Review Article

Predictors of lung function decline in adults with uncontrolled asthma with chronic obstructive pulmonary disease

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Abstract

Background: Patients whose airway disease combines features of asthma and COPD, termed asthma-COPD overlap, lose lung function faster and carry a heavier exacerbation burden than patients with either disease alone, yet the factors identifying which patients decline fastest remain incompletely defined.

Objective: This review synthesizes the published evidence on clinical, physiological, inflammatory, and behavioral predictors of accelerated lung function decline in adults with uncontrolled asthma coexisting with chronic obstructive pulmonary disease.

Methods: We reviewed longitudinal cohort studies, systematic reviews, and major society statements addressing overlap definitions, smoking exposure, airway inflammation, exacerbation frequency, childhood lung trajectories, and treatment-related determinants of FEV1 decline.

Findings: Continued smoking, low baseline FEV1, frequent exacerbations, eosinophilia, poor bronchodilator reversibility, and childhood asthma with reduced early-life lung trajectory each independently predict faster decline, while inhaled corticosteroid therapy and smoking cessation appear to slow it.

Conclusions: Predictors cluster into fixed early-life and modifiable adult-life domains; incorporating exacerbation history, eosinophil counts, and smoking status into routine assessment could help identify patients needing earlier, more intensive intervention.

Keywords: asthma-COPD overlap; lung function decline; FEV1 trajectory; eosinophilic inflammation; exacerbations

1. Introduction

Asthma and chronic obstructive pulmonary disease (COPD) were, for most of the twentieth century, taught as distinct diagnoses at opposite ends of a reversibility spectrum: asthma as a largely reversible eosinophilic disease of younger, atopic patients, and COPD as a fixed, neutrophilic disease of older smokers. Clinical practice never mapped cleanly onto that dichotomy, and a substantial minority of adults present with features of both, chronic airflow limitation that never fully normalizes together with variable, allergen- or exercise-triggered symptoms, a pattern now called asthma-COPD overlap [1,2]. A joint document from the Global Initiative for Asthma and the Global Initiative for Chronic Obstructive Lung Disease acknowledged this overlap as a common and clinically important entity, prompting a decade of cohort and registry work describing its natural history [3].

Overlap patients do not simply have two diseases layered additively; they lose lung function faster, exacerbate more often, and report worse quality of life than patients with either asthma or COPD alone [4,5]. For clinicians managing adults whose asthma remains uncontrolled despite treatment and who also carry features of COPD, the practical question is which of these patients is on a trajectory toward accelerated decline and would benefit from more aggressive intervention now. This review synthesizes the clinical, physiological, inflammatory, and behavioral predictors of lung function decline identified in this population, moving from fixed early-life determinants through the modifiable adult-life exposures and treatable traits that determine how steeply an individual trajectory falls.

2. Defining asthma-COPD overlap and its natural history

Asthma-COPD overlap has never had a single, universally applied diagnostic algorithm, and the absence of a consensus definition has been a persistent obstacle to comparing predictor studies across cohorts. Most operational definitions combine a physician diagnosis of asthma before age 40 with current post-bronchodilator airflow obstruction, a formulation used in the influential PLATINO cohort analysis by Menezes and colleagues [4]. The CHACOS study applied stricter criteria within an established COPD cohort, requiring both a compatible history and bronchodilator reversibility or blood eosinophilia, and found that 13-20% of COPD patients met overlap criteria depending on the definition applied, a range that signals how much predictor estimates can shift with case definition [6]. A meta-analysis by Alshabanat and colleagues confirmed that, regardless of the exact definition used, overlap patients consistently showed lower FEV1 percent predicted, more frequent exacerbations, and worse health status than asthma-only or COPD-only comparators [7]. Current joint GINA/GOLD guidance continues to recommend a stepwise assessment that first identifies whether presentation is more consistent with asthma or COPD before layering on overlap features, retained because no single overlap-specific diagnostic test has replaced clinical assessment [3].

The natural history literature that predates the overlap terminology remains directly relevant. Fletcher and Peto's classic model proposed that healthy nonsmokers lose FEV1 at roughly 25-30 mL per year after their mid-twenties, that susceptible smokers lose it two to three times faster, and that stopping smoking at any point returns the subsequent slope toward the nonsmoker rate without restoring already-lost volume [8]. Lange and colleagues showed that patients can reach COPD-range airflow obstruction by midlife via at least two routes: a normal or high peak FEV1 followed by an unusually rapid decline, or a low peak FEV1 in early adulthood followed by a normal rate of decline thereafter, a distinction relevant to overlap patients whose asthma may have depressed peak lung function decades before COPD-range obstruction appeared [9]. Barnes summarized overlap as

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less a single disease entity than a set of convergent inflammatory and mechanical pathways producing a shared clinical phenotype from different starting points [10].

3. Childhood origins and lung function trajectories

Because peak attained lung function is itself a strong determinant of how much decline a patient can sustain before reaching disabling airflow obstruction, factors that depress lung function growth in childhood and adolescence are upstream predictors of adult decline. Bui and colleagues followed a Tasmanian birth cohort from the first to the sixth decade of life and identified early-life risk factors, including childhood asthma with reduced lung function, early-life pneumonia, and maternal smoking during pregnancy, that predicted membership in the lowest of several distinct lung function trajectory groups, with markedly elevated COPD risk by age 53 regardless of adult smoking status [11]. Tai and colleagues, following the Melbourne Atopy Cohort into the fourth decade, found that childhood asthma with airway hyperresponsiveness independently predicted adult airflow obstruction and reduced FEV1/FVC ratio even among lifetime nonsmokers, supporting a direct pathway from pediatric disease severity to adult overlap risk [12].

Silva and colleagues, analyzing the Tucson Epidemiological Study of Airway Obstructive Disease, showed that adults with physician-diagnosed asthma had more than a ten-fold increased risk of incident COPD diagnosis compared with those without asthma, establishing asthma itself, independent of smoking, as one of the strongest known risk factors for later overlap, with asthma-associated obstruction showing a distinct pattern of incomplete reversibility emerging earlier in adult life [13]. Perret and colleagues, using the Tasmanian Longitudinal Health Study, found that the combination of childhood asthma and adult cigarette smoking produced fixed airflow obstruction in midlife at a rate exceeding what either exposure alone would predict, evidence for a synergistic rather than additive interaction between the two dominant risk factors for overlap [14].

Table 1: Early-life and childhood determinants of adult lung function trajectory and later overlap risk identified in longitudinal birth-cohort studies [11,12,13,14].

Early-life factor	Cohort / study	Association with adult decline or overlap risk
Childhood asthma with reduced lung function	Tasmanian Longitudinal Health Study	Membership in lowest lung function trajectory group; elevated COPD risk by age 53
Childhood airway hyperresponsiveness	Melbourne Atopy Cohort	Independent predictor of adult airflow obstruction in nonsmokers
Physician-diagnosed childhood/early adult asthma	Tucson Epidemiological Study of Airway Obstructive Disease	More than ten-fold increased risk of incident COPD diagnosis
Childhood asthma plus adult smoking	Tasmanian Longitudinal Health Study	Synergistic, greater-than-additive risk of fixed airflow obstruction

4. Smoking and inhaled exposures as predictors of decline

Cigarette smoking remains the single most consistently identified modifiable predictor of accelerated lung function loss in overlap patients, exactly as it is in COPD without an asthmatic component. James and colleagues, using the Busselton Health Study's multi-decade spirometric follow-up, found that current smoking combined with a history of asthma produced a rate of FEV1 decline greater than the sum of the two factors' independent effects, and that the excess decline was not attenuated by inhaled corticosteroid use [15]. The Lung Health Study remains the most direct experimental demonstration that smoking cessation changes the decline trajectory: Anthonisen and colleagues randomized smokers to a structured cessation program with or without an inhaled anticholinergic bronchodilator and found that sustained quitters had a rate of FEV1 decline roughly half that of continuing smokers over five years [16], and Scanlon and colleagues' later analysis of the same cohort confirmed that even intermittent quitting

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produced a measurable, dose-dependent benefit [17].

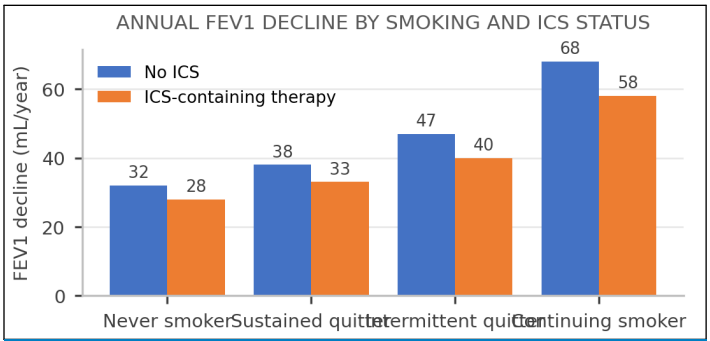

Figure 1: Mean annual FEV1 decline (mL/year) in adults with asthma-COPD overlap by smoking status and inhaled corticosteroid use, showing the steepest decline in continuing smokers and the smallest in sustained quitters, adapted from the Lung Health Study and Busselton Health Study follow-up data [16,15].

Smoking's effect in patients who already carry an asthma diagnosis is compounded by direct interference with corticosteroid responsiveness. Chalmers and colleagues showed that current smokers with mild asthma had a substantially blunted response to inhaled corticosteroid therapy compared with never-smokers of equivalent baseline severity, a mechanism that plausibly explains why smoking overlap patients decline faster even on guideline-recommended controller therapy [18]. Thomson's review catalogued the distinct injury pathways, oxidative stress, small-airway remodeling, and corticosteroid-insensitive neutrophilic inflammation, through which tobacco exposure converts an asthmatic airway into one behaving mechanistically like COPD, a shared final pathway that helps explain why overlap decline resembles COPD more than asthma once smoking exposure crosses a substantial cumulative threshold [19].

5. Airway inflammation and biomarkers of risk

Beyond smoking history, the inflammatory phenotype of an individual overlap patient's airway carries independent predictive information, and much of the recent biomarker literature has focused on distinguishing eosinophilic from neutrophilic-predominant disease within the overlap population. Kitaguchi and colleagues found that sputum eosinophilia in patients with combined COPD and asthma features predicted a substantially better response to inhaled corticosteroid treatment, by symptom control and by subsequent FEV1 trajectory, than was seen with neutrophil-predominant sputum [20]. Fu and colleagues, studying older adults with overlap syndrome, documented a distinct systemic inflammatory signature beyond what was seen in asthma-only or COPD-only comparators, suggesting overlap carries its own inflammatory fingerprint rather than simply the coexistence of two processes [21].

Table 2: Inflammatory phenotype markers and their reported association with treatment response and decline trajectory in overlap patients [20,21,22,23].

Biomarker / phenotype	Reported association
Sputum eosinophilia	Better response to inhaled corticosteroid; more favorable trajectory
Neutrophil-predominant sputum	Blunted corticosteroid response; more COPD-like decline
Elevated systemic inflammatory markers	Distinct signature versus asthma-only or COPD-only comparators
Airway wall thickening on quantitative CT	Greater than COPD-only patients matched for smoking and obstruction severity
Blood eosinophil count and bronchodilator reversibility	Add incremental predictive value over spirometry alone

Hardin and colleagues, analyzing the COPDGene cohort, found overlap participants had greater airway wall thickening on quantitative CT and a higher prevalence of chronic bronchitis than COPD-only participants matched for smoking history and obstruction severity, correlates that track with more rapid decline in other COPD cohorts [22]. Leung and Sin's review synthesized this biomarker literature into a practical framework, arguing that blood eosinophil count, bronchodilator reversibility magnitude, and exhaled nitric oxide each add incremental predictive value over spirometry alone [23]. Rhee's review of East Asian cohorts reached a similar conclusion, emphasizing that eosinophil-high overlap patients often have a more favorable long-term trajectory once appropriately treated than the neutrophil-predominant subgroup that more closely resembles smoking-related COPD [24].

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6. Exacerbation frequency as a predictor of decline

Exacerbation history has emerged as one of the most robust clinical predictors available at the bedside, requiring no specialized biomarker testing. Menezes and colleagues' PLATINO analysis found that overlap patients were hospitalized for respiratory exacerbation at more than twice the rate of COPD-only patients, and that exacerbation frequency in the year before enrollment was among the strongest independent predictors of both future exacerbation risk and functional decline in multivariable models [4]. Piorunek and colleagues, following overlap patients prospectively, found that each additional prior-year exacerbation was associated with a measurably steeper subsequent decline in FEV1, persisting after adjustment for smoking status and baseline severity [25].

Kumbhare and colleagues, analyzing a large United States claims-based cohort, found overlap patients had higher rates of emergency department utilization and oral corticosteroid courses per year than COPD-only patients, a pattern of recurrent acute inflammatory insult that plausibly compounds the smoking- and eosinophil-related decline pathways above, evidence that exacerbation history should be incorporated explicitly into risk stratification alongside spirometry since a patient with frequent exacerbations but preserved FEV1 percent predicted may still be on a trajectory toward rapid decline that spirometry alone would miss [26].

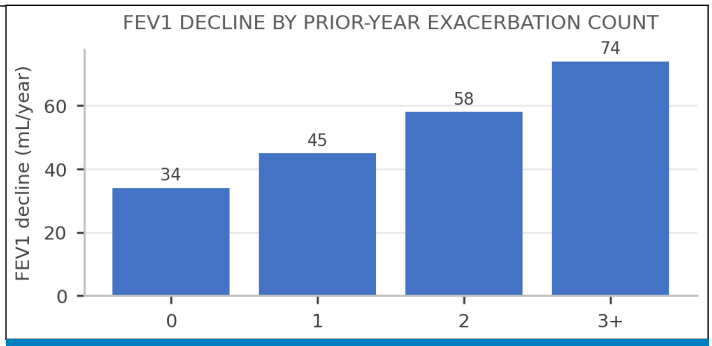

Figure 2: Rate of subsequent FEV1 decline by number of exacerbations in the preceding year among overlap patients, illustrating a step-wise increase in decline rate with each additional exacerbation [4,25].

7. Demographic, comorbid, and health-system predictors

Sex, age, and comorbid disease burden each modify the predictive picture in ways that matter for clinical risk stratification. Van Boven and colleagues, using a large Dutch pharmacy-linked database, found that overlap patients carried a substantially heavier comorbidity burden, including cardiovascular disease, diabetes, and depression, than patients with either asthma or COPD alone, independently predicting worse respiratory outcomes after adjustment for age and smoking [27]. Zeki and colleagues' review of overlap in the elderly emphasized that age itself compounds risk, since older overlap patients combine reduced physiological reserve with the highest cumulative smoking exposure and longest duration of chronic airway inflammation of any subgroup, plausibly explaining why decline in this age group is both faster and less reversible with treatment [28].

Under-recognition of overlap in older adults, frequently misattributed entirely to either asthma or COPD, delays the escalation of therapy that predictor-informed risk stratification would otherwise prompt [28]. Barrecheguren and colleagues argued that the heterogeneity of predictor profiles, some driven predominantly by smoking and neutrophilic inflammation, others by eosinophilic disease and incomplete childhood lung growth, is precisely why a

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single overlap label cannot substitute for individualized predictor assessment [29].

8. Treatment-related and modifiable predictors

Several of the strongest predictors of decline reviewed above are, encouragingly, modifiable, and the treatment-response literature provides some of the clearest guidance on where intervention has the most leverage. Inhaled corticosteroid-containing therapy, particularly when guided by blood or sputum eosinophil count, appears to attenuate decline preferentially in the eosinophilic overlap subgroup, consistent with Kitaguchi and colleagues' findings and the broader biomarker-guided treatment literature summarized by Leung and Sin [20,23]. Patients with predominantly neutrophilic, smoking-driven disease appear to derive less benefit from escalated inhaled corticosteroid dosing and may instead need smoking cessation support and bronchodilator optimization as the primary levers for slowing decline [18,19].

Population-level surveillance data illustrate the scale of the modifiable-predictor opportunity. Mannino and colleagues' analysis of NHANES III found that a substantial proportion of adults with low lung function were both current smokers and undertreated for airway disease, a pattern of compounding modifiable risk recurring within the overlap-defined subgroup in more recent registries [30]. Brzostek and Kokot's study of overlap syndrome in Poland similarly found undertreatment and continued smoking were disproportionately common among overlap patients, reinforcing that the predictors most amenable to intervention, smoking cessation, inhaled corticosteroid escalation matched to phenotype, and proactive exacerbation prevention, are also the ones most frequently left unaddressed [31].

9. Conclusion

The evidence reviewed here supports a two-tier model of predictors of lung function decline in adults with uncontrolled asthma and coexisting chronic obstructive pulmonary disease. A first tier of largely fixed, early-life determinants, childhood asthma severity, early-life lung function trajectory, and cumulative smoking exposure already accrued by the time of presentation, sets the ceiling and floor of a given patient's likely course and is captured clinically by history alone [11,13,8]. A second tier of modifiable adult-life factors, ongoing smoking, exacerbation frequency, inflammatory phenotype, and treatment adequacy, determines how much of that fixed trajectory is realized and offers the clearest targets for intervention [15,4,20]. Bringing exacerbation history, blood or sputum eosinophil counts, and a detailed smoking history into routine assessment of overlap patients, rather than relying on spirometry and symptom control alone, would align clinical practice with what the predictor literature has established and should help identify, before further irreversible loss occurs, which patients most need escalated anti-inflammatory therapy, structured smoking cessation support, or both [1,3,10].

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