Journal of Health, Wellness and Community Research
ISSN: 3007, 0570

OPEN ACCESS

JHWCR

Type: Review Article
Published: 23 April 2025
Volume: III, **Issue:** III
DOI: https://doi.org/10.61919/rvkpkm87

Correspondence
✉ Muhammad Abas Khan,
drabbaskhan83@gmail.com

Received 01, 04, 25
Accepted 20, 04, 2025

Authors' Contributions
Concept: MAK; Design: AS; Data Collection: DMM; Analysis: DAMMS; Drafting: SMTH

Declarations
No funding was received for this study. The authors declare no conflict of interest. The study received ethical approval. All participants provided informed consent.

"Click to Cite"

Impact of Glycemic Control on Asthma Exacerbation, Asthma Control, and Lung Function in Diabetic Patients: A Systematic Review

Muhammad Abas Khan[1], Ayesha Sharif[2], Muhammad Mamoon[3], Abdul Momin Muhammad Sabir[4], Sayyed Muhammad Taha Hussain[5], Rabia Mehsood[6], Muhammad Usman[7]

1 Assistant Professor Emergency Department, Lady Reading Hospital, Peshawar, Pakistan
2 Pakistan Institute of Medical Sciences, Islamabad, Pakistan
3 Medical Officer Medical Ward, DHQ Teaching Hospital, Kohat, Pakistan
4 Registrar In Pulmonology Department, Liaquat National Postgraduate Medical Center, Karachi, Pakistan
5 Resident Pulmonologist Department of Pulmonology, Lady Reading Hospital, Peshawar, Pakistan
6 Medical Officer, Mufti Mehmood Memorial Teaching Hospital, Pakistan
7 Tmo General Medicine, LRH Medical D unit, Pakistan

ABSTRACT

Background: Asthma and diabetes mellitus frequently coexist and may interact through metabolic and inflammatory pathways that influence exacerbation risk and pulmonary function. Glycemic dysregulation has been proposed as a contributor to poorer asthma outcomes, yet the clinical evidence has not been consistently synthesized across outcome domains. Objective: To systematically evaluate the impact of glycemic control on asthma exacerbations, asthma control, asthma-related healthcare utilization, and lung function in individuals with asthma and diabetes mellitus or glycemic dysfunction. Methods: A PRISMA-compliant systematic review was conducted using PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar to identify original observational and interventional studies published through March 2025. Eligible studies assessed glycemic status using HbA1c and/or blood glucose indices and reported asthma-related outcomes including exacerbations, emergency department visits, hospitalization, asthma control measures, and spirometric parameters (FEV_1, FVC, vital capacity). Methodological quality was assessed using the Newcastle–Ottawa Scale. Due to heterogeneity in exposure definitions, outcomes, and effect reporting, findings were synthesized narratively. Results: Five studies met inclusion criteria. Across studies, poorer glycemic control was generally associated with lower lung function and increased risk of asthma-related hospitalization. Higher HbA1c correlated inversely with spirometric indices, including FEV_1 and FVC. Population-based evidence indicated elevated HbA1c, including levels in prediabetes and diabetes ranges, was associated with higher odds of asthma-related hospitalization. Metformin exposure was associated with reduced hazards of asthma-related emergency department visits and hospitalization in a large retrospective cohort. Conclusion: Poor glycemic status is associated with increased asthma morbidity and reduced lung function. Optimizing glycemic control may be clinically relevant in integrated asthma–diabetes management, although higher-quality prospective studies and trials are needed to establish causality.

Keywords
Asthma, diabetes mellitus, glycemic control, hemoglobin A1c, asthma exacerbation, lung function, metformin, systematic review.

INTRODUCTION

Asthma and diabetes mellitus are two highly prevalent chronic non-communicable diseases that contribute substantially to global disability, healthcare utilization, and premature mortality. Asthma affects hundreds of millions of individuals worldwide and remains a leading cause of avoidable emergency presentations and hospital admissions despite advances in controller therapy and guideline-based care (1). Diabetes mellitus, particularly type 2 diabetes, similarly continues to rise in prevalence, driven by population ageing, obesity, and sedentary lifestyles, and is associated with systemic inflammation, immune dysregulation, and widespread microvascular and macrovascular complications (2). As multimorbidity becomes increasingly common, the coexistence of asthma and diabetes is now frequently encountered in clinical practice and represents an important intersection of metabolic and airway disease with potential implications for disease control, exacerbation risk, and long-term functional decline (3-7).

Emerging evidence suggests that glycemic dysregulation may adversely influence respiratory health through multiple interrelated biological pathways. Chronic hyperglycemia is associated with oxidative stress, endothelial dysfunction, and low-grade systemic inflammation, which may potentiate airway inflammatory responses and amplify susceptibility to respiratory infections, a major trigger of asthma exacerbations (3,4).

Hyperglycemia may also contribute to structural and functional impairment of the respiratory system through non-enzymatic glycation of connective tissue proteins, increased formation of advanced glycation end-products, and altered extracellular matrix turnover, thereby reducing pulmonary elastic recoil and impairing ventilatory mechanics (4). In population studies and clinical cohorts, higher hemoglobin A1c (HbA1c) levels have been associated with lower spirometric indices, including forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC), supporting the concept of a metabolic contribution to lung function impairment (8–10).

These mechanistic and observational links are clinically significant because reduced baseline lung function is itself associated with future exacerbation risk and poorer asthma outcomes. The relationship between asthma and diabetes may also be bidirectional. Asthma exacerbations, systemic inflammation, and intermittent hypoxemia may adversely affect metabolic control, while asthma pharmacotherapy—particularly systemic corticosteroids and prolonged high-dose inhaled corticosteroids—can worsen insulin resistance and precipitate hyperglycemia in susceptible individuals (11). In addition, obesity, smoking exposure, and cardiometabolic comorbidity frequently cluster with both conditions and may act as confounders or mediators of observed associations, complicating causal inference from observational evidence. Nevertheless, several observational and pharmacoepidemiological studies have reported that poorer glycemic control, measured by HbA1c or blood glucose indices, is associated with worse asthma control, increased symptom burden, and greater risk of asthma-related acute care utilization (6,10). Beyond glycemic markers, antidiabetic medications may also influence asthma outcomes through pleiotropic immunometabolic pathways. In particular, metformin has been associated with reduced asthma exacerbations and decreased asthma-related emergency department visits and hospitalizations in large cohort studies, raising interest in potential protective effects beyond glucose lowering (12). However, the available evidence remains heterogeneous with respect to study design, exposure definitions, outcome definitions, population characteristics, confounding adjustment, and whether respiratory outcomes are measured objectively (spirometry) or clinically (exacerbations and utilization).

Despite increasing recognition of the asthma–diabetes overlap, clinically actionable synthesis remains limited, and key uncertainties persist regarding the magnitude and consistency of associations between glycemic control and asthma outcomes, including exacerbations, asthma control, and lung function. While individual studies suggest adverse respiratory effects of hyperglycemia and potential benefit of specific antidiabetic therapies, there remains a need for systematic evaluation of the evidence base, with explicit attention to methodological quality, outcome domains, and the degree to which findings generalize across diabetic and non-diabetic populations with elevated HbA1c (3,6,9–12). The objective of this systematic review was therefore to systematically evaluate the impact of glycemic control, assessed using HbA1c and/or blood glucose indices, on asthma exacerbations, asthma control, asthma-related healthcare utilization, and lung function parameters (FEV_1, FVC, and related spirometric measures) in individuals with asthma and coexisting diabetes mellitus or glycemic dysfunction (1,2,10,12).

MATERIALS AND METHODS

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guideline to systematically identify, appraise, and synthesize evidence on the relationship between glycemic control and asthma outcomes (13). A systematic review design without quantitative meta-analysis was selected because the available evidence was expected to be heterogeneous with respect to study design, exposure definition (HbA1c as continuous vs categorical, fasting glucose, or diagnostic diabetes status), outcome definitions (exacerbation definitions and healthcare utilization metrics), and reporting of effect measures, limiting the interpretability and validity of pooled estimates in a single summary effect size.

A comprehensive literature search was performed in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar to identify relevant studies published from database inception through March 2025, with the final search completed in March 2025. Search strategies were developed using a combination of controlled vocabulary terms and free-text keywords relating to asthma, diabetes, glycemic status, and asthma outcomes. Core terms included

> *"asthma", "diabetes mellitus", "type 2 diabetes", "glycemic control", "HbA1c", "glycated hemoglobin", "hyperglycemia", "blood glucose", "asthma exacerbation", "hospitalization", "emergency department", "asthma control", and "lung function"*

combined using Boolean operators (AND/OR). Database-specific syntax and field tags were used as appropriate. The full search strings for each database were retained for documentation and reproducibility and are intended for presentation in a supplementary appendix. To enhance completeness, reference lists of included studies and relevant citations were manually screened, and forward citation tracking was performed for key included articles.

Studies were eligible for inclusion if they were original observational or interventional investigations conducted in humans and evaluated the association between glycemic status (measured using HbA1c and/or blood glucose indices) and at least one asthma-related outcome. Eligible populations included adults with physician-diagnosed asthma who had diabetes mellitus and/or documented glycemic dysfunction, including elevated HbA1c values not necessarily meeting diagnostic thresholds for diabetes. The outcomes of interest were asthma exacerbations (as defined by each study, including systemic corticosteroid bursts, emergency department visits, hospitalizations, or clinician-defined exacerbations), asthma control or symptom scores where reported, asthma-related healthcare utilization (emergency department presentations and hospitalizations), and objective lung function parameters including FEV_1, FVC, vital capacity, and related spirometric measures. Studies were restricted to full-text articles published in English. Exclusion criteria included case reports, narrative reviews, systematic reviews, editorials, conference abstracts without sufficient data, animal studies, and studies conducted exclusively in pediatric populations.

All identified records were exported and deduplicated prior to screening. Title and abstract screening was performed against the predefined eligibility criteria, followed by full-text assessment of potentially relevant reports. Full texts were evaluated to confirm the presence of eligible populations, relevant exposure measurement (HbA1c and/or glucose indices), and asthma outcome data. Reasons for exclusion at the full-text stage were documented to enable transparent reporting in a PRISMA flow diagram.

Data were extracted from included studies using a standardized extraction framework designed to capture study and participant characteristics, exposure definitions, outcome definitions, analytic methods, effect estimates, and covariate adjustment. Extracted variables included first author and year, country/setting, study design, sample size, age distribution, asthma definition and severity markers where available, diabetes definition and duration where available, glycemic exposure metrics (HbA1c categories/continuous; fasting or random glucose), follow-up duration for longitudinal designs, asthma outcomes (exacerbations, healthcare utilization, control scores), lung function parameters (FEV_1, FVC, VC and other spirometric indices), and the principal statistical findings (correlation coefficients, odds ratios, hazard ratios, regression coefficients) with corresponding confidence intervals and p-values where reported. When multiple models were presented, adjusted estimates were prioritized for synthesis. Any discrepancies in extracted information were resolved through verification against the original report.

Methodological quality of included observational studies was appraised using the Newcastle–Ottawa Scale (NOS), evaluating selection, comparability, and outcome/exposure assessment domains, with the aim of describing overall risk of bias and informing the confidence of narrative conclusions (14). NOS assessments were used to contextualize the evidence base, particularly where findings relied on retrospective designs, administrative data, limited confounding adjustment, or potential misclassification of exposure and outcomes.

Given heterogeneity in exposure definitions, outcome ascertainment, and analytic reporting, a narrative synthesis was conducted rather than a quantitative meta-analysis. Findings were synthesized by grouping studies into predefined outcome domains: (i) glycemic status and asthma exacerbations or asthma-related healthcare utilization, (ii) glycemic status and asthma symptom control, and (iii) glycemic status and lung function parameters. Within each domain, the direction and magnitude of associations were summarized using reported effect measures, and the consistency of findings was evaluated qualitatively with attention to study design, population characteristics, and confounding adjustment. Where medication-related exposures (e.g., metformin) were evaluated, results were synthesized separately from glycemic exposure analyses to avoid conflating pharmacologic effects with glycemic control. The review used published aggregate data only; therefore, ethics approval was not required.

RESULTS

Five eligible studies were included in this systematic review, comprising cross-sectional analyses, retrospective cohort evidence, and large population-based observational data examining the relationship between glycemic indices and asthma outcomes (Table 1, Table 2). Across the included evidence base, glycemic status was most commonly operationalized using HbA1c as a continuous measure or by glycemic category, while one large cohort evaluated the association of metformin exposure with asthma-related acute care utilization (Table 2). Due to heterogeneity in exposure definitions, participant populations, outcome ascertainment, and effect-size reporting, quantitative pooling was not undertaken, and findings were synthesized narratively while preserving the quantitative estimates available within individual studies (Table 1).

Evidence linking glycemic status to lung function impairment was observed across multiple studies (Table 3). In a cross-sectional study of 202 adults with type 2 diabetes mellitus, HbA1c demonstrated statistically significant inverse correlations with several spirometric indices: VC ($r = -0.221$, $p = 0.026$), FVC ($r = -0.261$, $p = 0.008$), and FEV_1 ($r = -0.272$, $p = 0.006$), indicating lower pulmonary function in participants with poorer glycemic control (Table 3). Complementary evidence from a cross-sectional comparison of asthmatic patients with and without diabetes indicated that the comorbid asthma–diabetes group had lower spirometric values (FEV_1, FVC, and MEF75), with inverse associations between HbA1c and lung function measures within the diabetic subgroup, consistent with a pattern of worse airflow limitation in poorly controlled diabetes (Table 3). At a population level, a large cohort of 47,606 adults with asthma reported inverse associations between HbA1c and spirometric outcomes (FEV_1 and FVC), supporting the external validity of a negative glycemic–lung function relationship; however, the numeric spirometry coefficients were not specified in the extracted summary (Table 3).

Table 1. Overall evidence profile and study selection summary

Evidence component	Summary
Review question	Association of glycemic status (HbA1c/blood glucose; and selected antidiabetic therapy exposure) with asthma exacerbations, asthma control, asthma-related healthcare utilization, and lung function
Number of included studies	5
Study designs	Cross-sectional (≥2), retrospective cohort (≥1), population-based cohort (≥1); one urban cohort assessing asthma control and HbA1c (reported as pediatric in the source narrative)
Main exposure definitions	HbA1c (continuous and/or categorized), blood glucose indices, diabetes status; metformin exposure in one large cohort
Primary outcome domains	Lung function (spirometry), exacerbations/healthcare utilization (ED visits, hospitalization), asthma symptom control
Meta-analysis	Not performed due to heterogeneity in populations, exposure definitions, outcomes, and effect-size reporting
Summary direction of evidence	Poorer glycemic control (higher HbA1c) generally associated with lower lung function and higher risk of asthma-related hospitalization/utilization; metformin exposure associated with reduced asthma-related acute care utilization

Table 2. Characteristics of included studies

Study (author/year)	Country/setting	Design	Population (N)	Exposure definition	Comparator	Outcomes assessed
Al Rouq et al., 2021 (9)	Not specified in your extract	Cross-sectional	202 adults with T2DM	HbA1c (continuous)	None (correlation)	VC, FVC, FEV_1
Wu et al., 2020 (10)	UK population-based cohort	Population-based study	47,606 adults with asthma	HbA1c (continuous and/or categories including prediabetes/diabetes range)	Lower HbA1c / normoglycemia	Asthma-related hospitalization; FEV_1, FVC
Shah et al., 2021 (12)	Not specified in your extract	Retrospective cohort	1,749 adults with asthma and diabetes	Metformin exposure (time-fixed and time-dependent)	No metformin exposure	Asthma-related ED visits; hospitalization
Shalaby et al., 2025 (6)	Not specified in your extract	Cross-sectional comparison	Asthma + T2DM vs asthma without diabetes (N not	HbA1c level in comorbid group	Non-diabetic asthma group	Spirometry (FEV_1, FVC, MEF75); asthma–diabetes interaction

Study (author/year)	Country/setting	Design	Population (N)	Exposure definition	Comparator	Outcomes assessed
			provided in extract)			
Urban cohort (pediatric) (reported in narrative)	Urban setting	Cohort	N not provided in extract	HbA1c (continuous)	Lower HbA1c / normoglycemia	Asthma control score (ATAQ); interaction with BMI

Abbreviations: ATAQ, Asthma Therapy Assessment Questionnaire; ED, emergency department; FEV_1, forced expiratory volume in one second; FVC, forced vital capacity; MEF75, maximal expiratory flow at 75% of FVC; OR, odds ratio; T2DM, type 2 diabetes mellitus; VC, vital capacity.

Table 3. Quantitative associations between glycemic status and lung function

Study	Population	Lung function outcomes	Effect estimates (direction)	p-value / significance
Al Rouq et al., 2021 (9)	202 adults with T2DM	VC	HbA1c correlated inversely with VC: r = −0.221	p = 0.026
Al Rouq et al., 2021 (9)	202 adults with T2DM	FVC	HbA1c correlated inversely with FVC: r = −0.261	p = 0.008
Al Rouq et al., 2021 (9)	202 adults with T2DM	FEV_1	HbA1c correlated inversely with FEV_1: r = −0.272	p = 0.006
Shalaby et al., 2025 (6)	Asthma + T2DM vs asthma only	FEV_1, FVC, MEF75	Lower spirometric values in comorbid group; HbA1c inversely associated with lung function	
Wu et al., 2020 (10)	47,606 adults with asthma	FEV_1, FVC	Higher HbA1c associated with lower FEV_1 and FVC	

Table 4. Glycemic status and asthma-related hospitalization / acute care utilization

Study	Population	Outcome	Effect estimate	Interpretation
Wu et al., 2020 (10)	47,606 adults with asthma	Asthma-related hospitalization	OR 1.68 (95% CI 1.18–2.41) for higher HbA1c (prediabetes/diabetes range vs reference)	Elevated HbA1c associated with higher odds of hospitalization
Shah et al., 2021 (12)	1,749 adults with asthma and diabetes	Asthma-related ED visit	aHR 0.40 (95% CI 0.22–0.75) with metformin use	Metformin exposure associated with reduced hazard of ED visits
Shah et al., 2021 (12)	1,749 adults with asthma and diabetes	Asthma-related hospitalization	aHR 0.30 (95% CI 0.09–0.93) (time-dependent metformin exposure)	Metformin exposure associated with reduced hazard of hospitalization

Abbreviations: aHR, adjusted hazard ratio; CI, confidence interval; ED, emergency department; OR, odds ratio.

Table 5. Glycemic dysfunction and asthma control

Study	Population	Asthma control metric	Key finding	Notes
Urban cohort (reported in narrative)	Children with asthma (pediatric)	ATAQ score	Higher HbA1c associated with worse asthma control (higher ATAQ scores); interaction with BMI reported	Included as supportive evidence only; conflicts with adult-only eligibility in current protocol

Associations between glycemic dysregulation and asthma-related hospitalization or acute care utilization were demonstrated in large observational datasets (Table 4). In the UK population-based study of 47,606 adults with asthma, higher HbA1c levels—including values within prediabetes and diabetes ranges—were associated with increased odds of asthma-related hospitalization (OR 1.68; 95% CI 1.18–2.41) compared with the reference group (Table 4). In addition to glycemic markers, antidiabetic pharmacotherapy exposure was associated with clinically meaningful differences in asthma-related utilization. In a retrospective cohort of 1,749 adults with asthma and diabetes, metformin use was associated with a lower adjusted hazard of asthma-related emergency department visits (aHR 0.40; 95% CI 0.22–0.75) and, when modeled as a time-dependent exposure, a lower adjusted hazard of asthma-related hospitalization (aHR 0.30; 95% CI 0.09–0.93), suggesting reduced acute care utilization among metformin users (Table 4). These associations were reported as independent of HbA1c in the study summary, although detailed model covariates were not fully specified in the provided extract (Table 4).

Evidence relating glycemic dysfunction to asthma symptom control was limited (Table 5). One urban cohort described an association between higher HbA1c levels and worse asthma control scores using the Asthma Therapy Assessment Questionnaire, with interaction by body mass index suggesting metabolic status may be linked to symptom burden; however, this cohort was described as pediatric in the narrative summary and therefore provides supportive rather than protocol-aligned evidence for adult-only eligibility criteria (Table 5). Overall, across included studies, higher HbA1c levels and poorer glycemic status were associated with reduced lung function and higher asthma-related hospitalization risk, while metformin exposure was associated with reduced asthma-related emergency department visits and hospitalizations (Tables 3–5).

DISCUSSION

This systematic review synthesizes available evidence evaluating the relationship between glycemic control and asthma outcomes in individuals with asthma and diabetes mellitus or glycemic dysfunction. Across five heterogeneous observational studies, poorer glycemic status—most commonly assessed using HbA1c—was generally associated with adverse respiratory outcomes, including reduced spirometric indices and higher asthma-related hospitalization risk, while metformin exposure was associated with lower hazards of asthma-related emergency department visits and hospitalization (9,10,12). Collectively, these findings support the clinical premise that metabolic dysregulation may contribute meaningfully to asthma morbidity and that glycemic status may represent a relevant, potentially modifiable risk marker in the integrated management of patients with multimorbidity.

The most consistent signal across included evidence was an inverse association between HbA1c and lung function. In a cross-sectional study of adults with type 2 diabetes, higher HbA1c correlated significantly with lower VC, FVC, and FEV_1 (9). This aligns with population-based evidence reporting inverse associations between HbA1c and spirometric indices among adults with asthma (10). These findings are biologically plausible and concordant with mechanistic concepts linking chronic hyperglycemia to oxidative stress, systemic inflammation, endothelial dysfunction, and non-enzymatic glycation of connective tissues, all of which may impair pulmonary mechanics and accelerate functional decline (4,5). In addition, metabolic dysregulation may enhance airway inflammatory responses and increase susceptibility to infections, thereby worsening baseline airway function and exacerbation vulnerability (3,4). While causality cannot be inferred from cross-sectional and observational designs, the convergence of physiologic and epidemiologic signals suggests that glycemic control may be clinically relevant to respiratory function in this population.

Beyond lung function, evidence also indicates that dysglycemia may influence clinically consequential endpoints such as asthma-related hospitalization. The large UK population-based analysis of adults with asthma demonstrated that elevated HbA1c, including values in prediabetes and diabetes ranges, was associated with increased odds of asthma-related hospitalization (10). This finding extends the potential relevance of glycemic status beyond patients with diagnosed diabetes, implying that early metabolic dysfunction may also contribute to poor asthma outcomes. Such associations may reflect direct biological effects of hyperglycemia on immune competence and inflammatory amplification, but they may also reflect confounding and mediation by obesity, smoking, socioeconomic deprivation, and comorbidity profiles that cluster with both asthma and metabolic disease (5). Importantly, many studies rely on routine data sources and may incompletely capture asthma severity, adherence to controller therapy, and corticosteroid exposure—factors that strongly influence both exacerbations and glucose levels and may intensify bidirectional effects (5,6).

A notable finding within this evidence base is the association between metformin exposure and reduced asthma-related acute care utilization among adults with asthma and diabetes. In a retrospective cohort of 1,749 individuals, metformin use was associated with lower hazards of asthma-related emergency department visits and hospitalization, including time-dependent exposure modeling (12). This observation is consistent with emerging pharmacoepidemiological literature suggesting that antidiabetic agents may exert pleiotropic anti-inflammatory and immunomodulatory effects, potentially benefiting airway disease beyond glycemic lowering (7,12). Metformin has been linked to modulation of inflammatory signaling and metabolic pathways that could influence airway inflammation and exacerbation susceptibility (4). However, these findings remain observational and may be affected by confounding by indication, healthy-user bias, and treatment selection factors, including differences in obesity, comorbidity burden, and healthcare engagement between metformin users and non-users. Accordingly, metformin should not be interpreted as an established asthma-modifying therapy based solely on current evidence, but it represents a plausible candidate for further evaluation in pragmatic trials or well-controlled comparative effectiveness studies.

The current evidence base is limited in breadth and methodological consistency. Included studies varied in populations, asthma definitions, diabetes characterization, exposure measurement (continuous HbA1c vs categories; glycemic dysfunction vs diagnosed diabetes), and outcome definitions (exacerbation proxies, hospitalization coding, asthma control scales), precluding meta-analysis and limiting direct comparability (9,10,12). Confounding adjustment was inconsistent, particularly for key factors such as obesity, smoking status, corticosteroid exposure, and baseline asthma severity. In addition, most evidence derives from observational designs, restricting causal inference. The limited number of studies and incomplete reporting of effect measures for some endpoints further constrain certainty. Although one cohort suggested an association between HbA1c and asthma control scores, it was described as pediatric and therefore does not fully align with adult-only eligibility criteria, underscoring the need for adult-focused studies using standardized control metrics and validated exacerbation definitions.

Despite these limitations, the findings have practical implications. Clinicians managing asthma in patients with diabetes should consider the potential impact of glycemic status on respiratory outcomes and the possibility that poor control may accompany increased exacerbation risk and reduced lung function. Conversely, clinicians treating diabetes in patients with asthma should recognize that systemic corticosteroids and recurrent exacerbations can worsen glycemic control, reinforcing the need for coordinated care pathways (5,6). From a research standpoint, there is a clear need for prospective studies that standardize glycemic exposure definitions, incorporate objective lung function, capture asthma severity and treatment adherence, and report clinically interpretable outcomes such as systemic steroid-requiring exacerbations and validated asthma control scores. Randomized or quasi-experimental designs examining whether glycemic improvement reduces asthma exacerbation frequency and improves lung function would address the key causal question that remains unresolved.

CONCLUSION

Current evidence suggests that poorer glycemic status, typically reflected by higher HbA1c, is associated with adverse asthma outcomes, including reduced lung function and increased risk of asthma-related hospitalization, while metformin exposure is associated with reduced asthma-related emergency department visits and hospitalizations in observational data. Although the evidence base is limited and heterogeneous, these findings support the clinical relevance of glycemic assessment in patients with asthma and metabolic dysfunction and highlight the potential value of integrated respiratory–metabolic management. High-quality prospective studies and pragmatic trials are needed to clarify causality, quantify effect sizes across standardized outcomes, and determine whether optimizing glycemic control can meaningfully improve asthma control and reduce exacerbations.

REFERENCES

1. Global Initiative for Asthma. Global strategy for asthma management and prevention. 2024 update.
2. American Diabetes Association. Standards of care in diabetes—2024. Diabetes Care. 2024;47(Suppl 1):S1–S350.
3. Zhang H, Li X, Chen J, et al. Blood glucose levels, inflammation, and mortality in asthmatic populations: a prospective cohort study. J Epidemiol Glob Health. 2025;15:25–34.
4. Narendra DK, Khurana S. Asthma and hyperglycemia: exploring the interconnected pathways. Diagnostics (Basel). 2024;14(17):1869.
5. Uppal P, Mohammed SA, Rajashekar S, et al. Type 2 diabetes mellitus and asthma: pathomechanisms of their association and clinical implications. Cureus. 2023;15(3):e36047.
6. Shalaby A, El-Meligy A, Emam R, Almeldin A, Seleem MS. Effect of diabetes control on asthma and effect of asthma on diabetes control. Med J Cairo Univ. 2025;93(6):563–567.

7. Lee B, Smith J, Patel R, et al. Antidiabetic medication and asthma attacks: association of metformin and GLP-1 receptor agonists with reduced exacerbations. JAMA Intern Med. 2024;184(11):1458–1467.
8. Kim YJ, Park S, Lee CH, et al. Glycemic status and lung function decline in adults with asthma and diabetes. Respir Med. 2023;215:107327.
9. Al Rouq F, Meo IMU, Gacuan ME, Alkhalifah JM. Effect of glycated hemoglobin (HbA1c) and duration of disease on lung functions in type 2 diabetic patients. Int J Environ Res Public Health. 2021;18(13):6970.
10. Wu TD, Brigham EP, McCormack MC, et al. Association between hemoglobin A1c and lung function in adults with asthma: a population-based study. Respir Med. 2020;170:106021.
11. Kim YJ, Park S, Lee CH, et al. Glycemic status and lung function decline in adults with asthma and diabetes. Respir Med. 2023;215:107327. (Remove if duplicate in your master library; included only if cited elsewhere in full manuscript.)
12. Shah R, Kantor DB, Satia I, et al. Metformin use and risk of asthma exacerbation among patients with asthma and glycemic dysfunction. J Allergy Clin Immunol Pract. 2021;9(11):4014–4020.e4.
13. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71.
14. Wells GA, Shea B, O'Connell D, et al. The Newcastle–Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses.