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THE ROLE OF OBESITY-RELATED CHRONIC INFLAMMATION IN THE PATHOGENESIS AND SEVERITY OF ASTHMA IN ADULTS: A SYSTEMATIC REVIEW

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ABSTRACT

Obesity and asthma are common chronic conditions that frequently coexist in adults and contribute to a substantial clinical and public health burden. Although excess body weight affects asthma through mechanical effects on lung volumes and airway closure, current evidence increasingly supports the role of obesity-related chronic low-grade inflammation as a central mechanism linking obesity with asthma development, severity, symptom burden, exacerbation risk, and therapeutic response. This systematic review summarizes contemporary evidence on the role of adipose-tissue inflammation, adipokines, cytokine signaling, metabolic dysfunction, oxidative stress, and immune dysregulation in the pathogenesis and clinical expression of obesity-related asthma in adults. A structured literature search was performed using major biomedical databases, with priority given to systematic reviews, meta-analyses, clinical reviews, cohort studies, mechanistic studies, and current asthma guidelines. The available literature indicates that obesity-related asthma is not a single uniform phenotype but a heterogeneous clinical entity involving overlapping inflammatory, metabolic, mechanical, and behavioral pathways. Increased systemic inflammatory activity, altered leptin and adiponectin signaling, elevated interleukin-6, neutrophilic airway inflammation, insulin resistance, and comorbidities such as obstructive sleep apnea and gastroesophageal reflux may all contribute to worse asthma control. Clinical management should therefore extend beyond inhaled pharmacotherapy and include assessment of obesity as a treatable trait, weight management, comorbidity screening, lifestyle intervention, and individualized evaluation of inflammatory phenotype. Future studies should clarify causal mechanisms and determine which interventions most effectively improve asthma outcomes in adults with obesity.

KEYWORDS

Asthma, Obesity, Chronic Inflammation, Adipokines, Severe Asthma, Adults

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Introduction

Asthma is a heterogeneous chronic inflammatory disease of the airways characterized by variable respiratory symptoms, airflow limitation, airway hyperresponsiveness, and fluctuating degrees of airway inflammation. In parallel, obesity has become one of the most important global health problems and is increasingly recognized not only as a metabolic disease but also as a chronic inflammatory condition. The coexistence of asthma and obesity is clinically important because adults with obesity frequently report greater symptom burden, poorer disease control, reduced quality of life, increased medication use, and a higher likelihood of difficult-to-treat or severe asthma. The relationship is not merely coincidental. Epidemiological studies and meta-analyses show that overweight and obesity increase the risk of incident asthma, and clinical studies demonstrate that excess adiposity can modify asthma expression and treatment response (Beuther & Sutherland, 2007; Peters et al., 2018).

For many years, the association between obesity and asthma was explained primarily by mechanical factors. Excess adipose tissue can reduce expiratory reserve volume and functional residual capacity, increase airway closure, alter chest wall mechanics, and contribute to dyspnea that may mimic or amplify asthma symptoms. These physiological effects remain clinically relevant, particularly in adults with severe obesity. However, mechanical restriction alone cannot fully explain the observed association. Obesity also modifies immune responses, systemic inflammatory tone, airway biology, and metabolic signaling. These mechanisms provide a more complete explanation for why asthma associated with obesity often has a different clinical course from asthma in lean adults.

Adipose tissue is now understood as an active endocrine and immune organ. In obesity, expansion of adipose tissue is accompanied by adipocyte hypertrophy, macrophage infiltration, altered adipokine production, oxidative stress, and increased release of pro-inflammatory mediators. This chronic low-grade systemic inflammation may influence the airways through circulating cytokines, adipokines, lipid mediators, and immune cell activation. Among the most frequently discussed mediators are interleukin-6 (IL-6), tumor

necrosis factor alpha (TNF-alpha), leptin, adiponectin, resistin, and markers of oxidative stress. Evidence from adult asthma cohorts suggests that systemic inflammatory pathways, particularly IL-6-associated metabolic inflammation, may be linked with poorer lung function, more frequent symptoms, and greater asthma severity (Peters et al., 2016).

The inflammatory profile of obesity-related asthma is complex. Traditional allergic asthma is often associated with type 2 inflammation, eosinophilia, and favorable response to inhaled corticosteroids. By contrast, asthma in adults with obesity may involve type 2-high, type 2-low, neutrophilic, or paucigranulocytic patterns. Some patients have pre-existing allergic asthma that becomes clinically more severe after weight gain. Others develop adult-onset asthma in the context of obesity, metabolic dysfunction, and low-grade systemic inflammation. These different pathways make obesity-related asthma difficult to define as a single phenotype. It is more accurately considered a group of overlapping phenotypes or treatable traits rather than one uniform disease entity (Peters et al., 2018; Scott et al., 2023).

The clinical implications of this relationship are substantial. Patients with obesity and asthma may experience diagnostic uncertainty because dyspnea can result from bronchoconstriction, deconditioning, obesity-related respiratory mechanics, obstructive sleep apnea, cardiac disease, or gastroesophageal reflux. Obesity may also reduce the apparent response to standard asthma therapy, especially when symptoms are driven by non-eosinophilic inflammation or mechanical limitation rather than classical steroid-responsive airway inflammation. Current clinical guidance emphasizes that comorbidities and modifiable traits, including obesity, should be assessed in patients with uncontrolled or difficult-to-treat asthma (Global Initiative for Asthma, 2024).

Understanding the inflammatory and metabolic links between obesity and asthma is therefore important for both pathogenesis and clinical management. A review focused on obesity-related chronic inflammation can help clarify why excess adiposity increases asthma risk, why some patients with obesity have more severe or poorly controlled asthma, and why management should include weight reduction and comorbidity assessment alongside conventional inhaled treatment. The objective of this review is to summarize contemporary evidence regarding the role of obesity-related chronic inflammation in the pathogenesis and severity of asthma in adults, with emphasis on adipose tissue biology, adipokines, cytokine signaling, airway inflammation, metabolic dysfunction, clinical phenotype, and implications for treatment.

Methodology

This article was prepared as a structured systematic literature review focused on adult patients and on the mechanistic and clinical relationship between obesity-related chronic inflammation and asthma. The review was developed in accordance with the general reporting principles of the PRISMA 2020 statement where applicable to qualitative evidence synthesis (Page et al., 2021). Because the aim was to integrate epidemiological, mechanistic, and clinical literature rather than calculate a single pooled effect estimate, findings were synthesized narratively.

The literature search was designed to identify publications addressing the association between obesity, chronic inflammation, and asthma in adults. The following databases and sources were considered: PubMed/MEDLINE, PubMed Central, Scopus, Web of Science, Embase, Cochrane Library, and current asthma guidance documents. Search terms were combined using Boolean operators and included: asthma, obesity, obese asthma, obesity-related asthma, severe asthma, chronic inflammation, systemic inflammation, airway inflammation, adipokines, leptin, adiponectin, interleukin-6, insulin resistance, metabolic dysfunction, weight loss, and adults. Reference lists of relevant systematic reviews and clinical reviews were also screened to identify additional key studies.

Eligible publications included systematic reviews, meta-analyses, narrative reviews, cohort studies, cross-sectional studies, randomized controlled trials, mechanistic human studies, and guideline documents that evaluated obesity, inflammatory markers, asthma risk, asthma severity, asthma control, lung function, exacerbations, airway inflammation, metabolic dysfunction, or treatment response in adults. Studies focused exclusively on children, animal models, isolated molecular pathways without clinical relevance, case reports, editorials without original synthesis, and publications not directly related to obesity-associated asthma were excluded from the main synthesis. Pediatric studies were not used as primary evidence because developmental mechanisms and body composition effects may differ from adult disease.

Data were extracted qualitatively with attention to the following domains: population characteristics, obesity definition, asthma definition or severity category, inflammatory markers, airway inflammation

phenotype, metabolic comorbidities, lung function outcomes, asthma control, exacerbation risk, treatment response, and conclusions relevant to clinical practice. Particular emphasis was placed on studies that helped explain mechanisms by which obesity-related chronic inflammation may contribute to asthma onset or worsening.

The review did not include a formal meta-analysis, risk-of-bias scoring, or certainty-of-evidence grading. Therefore, its conclusions should be interpreted as a structured qualitative synthesis rather than a quantitative evidence summary. However, priority was given to high-level evidence such as systematic reviews and meta-analyses when available.

Results

The literature consistently supports an association between obesity and asthma in adults. Meta-analytic evidence from prospective epidemiological studies indicates that overweight and obesity increase the likelihood of incident asthma, with a dose-response pattern suggesting greater risk at higher body mass index levels (Beuther & Sutherland, 2007). More recent syntheses have extended this observation by evaluating different measures of adiposity, including abdominal fatness and weight change, showing that asthma risk is not determined solely by body mass index but may also be related to fat distribution and changes in adiposity over time. This distinction is important because visceral adipose tissue is metabolically active and more strongly associated with systemic inflammation than subcutaneous fat.

The epidemiological association is supported by clinical observations. Adults with asthma and obesity often have worse symptom control than lean adults with asthma. They may report more dyspnea, greater rescue medication use, reduced exercise tolerance, and more frequent health-care utilization. Obesity has also been associated with progression toward difficult-to-treat asthma and severe asthma in some cohorts. However, the association is heterogeneous. Not all patients with obesity have severe asthma, and not all severe asthma in adults with obesity is driven by obesity-related inflammation. This heterogeneity is central to interpretation of the evidence.

One of the major findings from contemporary literature is that obesity-related asthma is not a uniform entity. Cluster analyses and phenotyping studies have described subgroups of adults in whom obesity interacts differently with asthma onset, inflammatory pattern, lung function, and treatment response (Haldar et al., 2008; Moore et al., 2010; Sutherland et al., 2012). In some adults, obesity complicates pre-existing allergic asthma. In others, asthma begins later in life in association with obesity, female sex, lower levels of classical allergic inflammation, and high symptom burden. This suggests that obesity can both increase asthma risk and modify the clinical expression of established asthma.

The inflammatory effects of obesity are mediated in part by adipose tissue. In lean individuals, adipose tissue participates in energy storage, endocrine regulation, and immune signaling. In obesity, adipocyte enlargement and local tissue stress promote immune cell infiltration, especially macrophage accumulation, and increased production of pro-inflammatory mediators. This state is often described as chronic low-grade systemic inflammation. Unlike acute infection-related inflammation, it is persistent, moderate in intensity, and closely linked with metabolic dysfunction. Circulating inflammatory mediators can influence the lung and airway environment, potentially increasing airway reactivity, altering immune responses, and amplifying symptoms.

Adipokines represent a key mechanistic link. Leptin, which is generally increased in obesity, has pro-inflammatory properties and may promote immune cell activation, oxidative stress, and airway inflammation. Adiponectin, which tends to decrease with increasing adiposity, has anti-inflammatory and insulin-sensitizing effects. A relative increase in leptin signaling together with reduced adiponectin may therefore favor a pro-inflammatory environment. Systematic evidence has suggested that leptin may be one biological link between obesity and asthma, although causality and clinical utility as a biomarker remain uncertain (Zheng et al., 2023).

Cytokine signaling is also important. IL-6 has received particular attention because it links metabolic dysfunction, obesity, and inflammatory disease. In adult asthma cohorts, higher plasma IL-6 concentrations have been associated with metabolic dysfunction and greater asthma severity (Peters et al., 2016). IL-6-related inflammation may identify a subgroup of patients in whom systemic metabolic inflammation contributes to asthma morbidity. This pathway is clinically relevant because it may partly explain why some adults with obesity have severe symptoms despite limited evidence of eosinophilic airway inflammation.

The relationship between obesity and airway inflammation remains debated. A recent systematic review and meta-analysis specifically examining adults with asthma found evidence that obesity is associated with

altered airway and systemic inflammation. In that analysis, sputum neutrophils were higher in adults with obesity than in non-obese adults with asthma, supporting a potential relationship between obesity and neutrophilic airway inflammation (Scott et al., 2023). However, the literature is not entirely consistent across biomarkers, and not all studies show stronger eosinophilic inflammation in patients with obesity. This supports the view that obesity may shift airway inflammation toward mixed or non-type 2 patterns in some individuals while amplifying type 2 disease in others.

Metabolic dysfunction may further contribute to asthma severity. Insulin resistance, dyslipidemia, hypertension, and metabolic syndrome are more common in adults with obesity and may influence airway smooth muscle, immune responses, and lung function. Hyperinsulinemia and insulin resistance have been hypothesized to increase airway contractility and alter inflammatory signaling. Although the causal direction remains difficult to establish, the coexistence of asthma with metabolic syndrome appears to identify patients with greater clinical complexity and potentially worse outcomes (Baffi et al., 2016; Peters et al., 2018).

Oxidative stress provides another mechanism connecting obesity-related inflammation and asthma. Excess adiposity can increase systemic oxidative burden through mitochondrial dysfunction, inflammatory cell activation, lipid peroxidation, and reduced antioxidant defenses. In the airways, oxidative stress may damage epithelial cells, increase bronchial hyperresponsiveness, and reduce responsiveness to corticosteroids. This pathway overlaps with both neutrophilic inflammation and metabolic dysfunction, making it difficult to separate from other inflammatory mechanisms. Nevertheless, it contributes to a broader picture in which obesity creates a systemic environment that may worsen airway disease.

Obesity also affects respiratory mechanics in ways that interact with inflammation. Reduced lung volumes can promote airway narrowing and closure, particularly in peripheral airways. Low lung volumes may increase airway hyperresponsiveness and enhance symptom perception. Mechanical effects may then amplify inflammatory pathways by increasing airway stress and altering ventilation distribution. Thus, the impact of obesity on asthma cannot be interpreted as purely inflammatory or purely mechanical. In many adults, both domains operate simultaneously.

Several comorbidities may mediate or amplify the obesity-asthma relationship. Obstructive sleep apnea, gastroesophageal reflux disease, chronic rhinosinusitis, depression, anxiety, physical inactivity, and deconditioning are common among adults with obesity and asthma. These conditions may worsen symptoms, mimic asthma, increase nighttime respiratory complaints, and complicate interpretation of asthma control. Current asthma guidance emphasizes assessment of comorbidities and modifiable factors in patients with uncontrolled symptoms or difficult-to-treat asthma (Global Initiative for Asthma, 2024).

Treatment response appears to be modified by obesity in some adults. Inhaled corticosteroids remain central to asthma management, especially for type 2 inflammatory asthma. However, obesity-related asthma may be associated with reduced corticosteroid responsiveness, particularly when symptoms are driven by non-eosinophilic inflammation, mechanical limitation, or metabolic dysfunction. This does not mean that inhaled corticosteroids are ineffective or should be withdrawn; rather, it means that persistent symptoms in adults with obesity should prompt reassessment of phenotype, adherence, inhaler technique, comorbidities, and treatable traits.

Weight loss interventions provide indirect evidence for the clinical importance of obesity in asthma. Lifestyle interventions, dietary programs, exercise, pharmacological weight reduction, and bariatric surgery may improve asthma symptoms, quality of life, lung function, and systemic inflammation in selected patients, although the magnitude of benefit varies between studies (Dias-Júnior et al., 2014; Foer et al., 2024; Freitas et al., 2015). Importantly, weight reduction may act through multiple pathways: reduced mechanical load, improved lung volumes, decreased systemic inflammation, improved metabolic function, increased physical activity, and reduced comorbidity burden.

Overall, the results indicate that obesity-related chronic inflammation contributes to asthma pathogenesis and severity through multiple interconnected mechanisms. The strongest evidence supports obesity as a risk factor and disease modifier for asthma, with systemic inflammation, altered adipokines, IL-6-associated metabolic dysfunction, neutrophilic airway inflammation, and comorbid conditions representing important links. However, heterogeneity remains substantial, and no single biomarker currently defines obesity-related asthma in adults.

Discussion

The evidence reviewed suggests that chronic inflammation associated with obesity plays an important role in the pathogenesis and severity of asthma in adults. The association is clinically meaningful because it affects not only disease risk but also symptom interpretation, diagnostic confidence, treatment response, and long-term management. However, the relationship is not simple. Obesity-related asthma should not be understood as a single inflammatory phenotype. Rather, it is best conceptualized as a clinical state in which excess adiposity interacts with airway disease through overlapping inflammatory, metabolic, mechanical, and behavioral mechanisms.

One practical implication is that body mass index alone is an insufficient explanation of asthma severity. BMI is useful as a screening measure and is widely available, but it does not capture visceral adiposity, fat distribution, muscle mass, metabolic health, inflammatory state, or respiratory mechanics. Two patients with identical BMI may differ substantially in airway inflammation, insulin resistance, physical fitness, sleep apnea risk, and asthma control. This explains why the relationship between obesity and asthma severity is inconsistent across studies and why individualized assessment is required.

Chronic low-grade inflammation provides a biologically plausible explanation for many clinical observations. Adipose tissue inflammation increases circulating cytokines and adipokines that can interact with airway immune responses. The IL-6 pathway is particularly relevant because it links obesity, metabolic dysfunction, and asthma severity. Findings from adult asthma cohorts suggest that IL-6-associated systemic inflammation may identify patients with severe disease and metabolic comorbidity (Peters et al., 2016). This supports the concept that inflammatory signals generated outside the lung may influence asthma expression within the lung.

At the same time, the relationship between systemic inflammation and airway inflammation is not always direct. Some adults with obesity and asthma have elevated systemic inflammatory markers without clear evidence of eosinophilic airway inflammation. Others have type 2-high asthma that is worsened by obesity-related physiology and comorbidities. Still others may have neutrophilic or paucigranulocytic disease. The systematic review by Scott and colleagues supports increased neutrophilic airway inflammation in adults with obesity and asthma, but heterogeneity across studies remains important (Scott et al., 2023). This means that inflammatory biomarkers should be interpreted in clinical context rather than used as isolated diagnostic labels.

The distinction between type 2-high and type 2-low inflammation is important for treatment. Biologic therapies targeting immunoglobulin E, interleukin-5, interleukin-4 receptor alpha, thymic stromal lymphopoietin, or other pathways have transformed the management of severe asthma. However, the response of patients with obesity may vary depending on the dominant inflammatory pathway and the degree to which symptoms are caused by asthma itself. Adults with obesity may still benefit from biologic therapy when a relevant type 2 inflammatory endotype is present, but persistent dyspnea may remain if obesity-related mechanics, deconditioning, sleep apnea, or reflux are not addressed. This reinforces the need for multidimensional assessment.

Another major clinical issue is overestimation or underestimation of asthma severity. In adults with obesity, dyspnea may be caused by airway obstruction, reduced lung volumes, poor conditioning, cardiac disease, anxiety, or sleep-disordered breathing. If all respiratory symptoms are attributed to asthma, clinicians may escalate inhaled or systemic corticosteroids unnecessarily. This can be harmful because systemic corticosteroids may promote weight gain, worsen glucose metabolism, and amplify the very comorbidity that contributes to poor asthma control. Conversely, if asthma symptoms are attributed only to obesity, airway inflammation may be undertreated. Careful objective evaluation is therefore essential.

Spirometry, bronchodilator testing, assessment of exacerbation history, inflammatory biomarkers, and evaluation of comorbidities are important in this population. Current asthma guidance emphasizes confirming the diagnosis and evaluating modifiable factors in difficult-to-treat asthma (Global Initiative for Asthma, 2024). This is especially relevant for adults with obesity, where symptom burden may be high even when airflow limitation is variable or absent. Objective evidence of variable expiratory airflow limitation, together with inflammatory assessment where available, can help avoid inappropriate treatment escalation.

Weight management should be considered a component of asthma care rather than a separate issue. Evidence from interventional studies suggests that weight loss can improve asthma control and quality of life in adults with obesity-related asthma, although the optimal strategy and expected magnitude of benefit vary (Foer et al., 2024). Lifestyle interventions may improve physical conditioning, diet quality, systemic inflammation, and respiratory mechanics. Bariatric surgery may produce larger weight loss and has been

associated with improvements in asthma outcomes in selected patients, but it is not appropriate for all individuals and requires long-term multidisciplinary care.

Exercise may be particularly valuable because it targets several mechanisms at once. Aerobic training can improve fitness, reduce symptoms related to deconditioning, and may reduce systemic inflammation and bronchial hyperresponsiveness in some adults with asthma (Freitas et al., 2015). However, exercise prescriptions must be realistic and individualized, especially for patients with severe obesity, uncontrolled symptoms, osteoarthritis, cardiovascular risk, or anxiety related to breathlessness. The goal should not be simply weight reduction but improvement in respiratory function, symptom confidence, metabolic health, and quality of life.

Dietary quality may also influence obesity-related asthma through inflammatory and metabolic pathways. Diets rich in saturated fats may promote systemic inflammation, whereas diets with higher intake of fruits, vegetables, fiber, and unsaturated fats may support anti-inflammatory pathways and improve metabolic health. However, dietary interventions are difficult to study because they interact with weight change, microbiome composition, socioeconomic factors, physical activity, and medication use. Therefore, current evidence supports general healthy dietary patterns and weight management rather than a single asthma-specific diet for all adults with obesity.

Pharmacological weight-loss therapies, including glucagon-like peptide-1 receptor agonists and dual incretin-based therapies, are emerging as potentially important interventions for adults with obesity and asthma. Their benefits may extend beyond weight reduction through effects on systemic inflammation, insulin resistance, and cardiometabolic risk. However, asthma-specific randomized trials remain limited. These therapies should therefore be viewed as promising but not yet fully established as asthma-directed treatments. Future research should clarify whether improvements in asthma outcomes are proportional to weight loss alone or whether additional anti-inflammatory or metabolic effects are relevant.

The role of adipokines as biomarkers remains uncertain. Leptin, adiponectin, resistin, and related mediators are biologically plausible and may contribute to asthma pathogenesis. However, routine measurement is not currently part of standard clinical asthma assessment. Their concentrations vary with sex, body composition, metabolic state, medications, and inflammatory comorbidities. Before adipokines can guide routine clinical management, studies must demonstrate reproducibility, predictive value, and impact on treatment decisions.

The reviewed evidence also has implications for public health. Asthma and obesity are both common conditions, and their overlap affects health-care utilization, medication burden, work productivity, and quality of life. Interventions that reduce obesity prevalence or improve metabolic health may therefore have respiratory benefits at population level. At the same time, asthma can limit physical activity and contribute to weight gain, creating a cycle in which respiratory symptoms and obesity reinforce each other. Breaking this cycle requires coordinated clinical and lifestyle support rather than isolated medication escalation.

Several limitations of the available literature should be acknowledged. Many studies are observational, making causal inference difficult. Obesity may precede asthma in some patients, but asthma symptoms and corticosteroid exposure may contribute to weight gain in others. Confounding by smoking, physical activity, diet, socioeconomic factors, sex hormones, comorbidities, and medication use is difficult to eliminate. In addition, asthma definitions, obesity measures, inflammatory markers, and outcome measures vary across studies. This heterogeneity limits direct comparison and makes it difficult to identify a single dominant mechanism.

Another limitation is that many clinical trials of asthma therapy do not specifically stratify outcomes by obesity phenotype or metabolic status. As a result, evidence for treatment response in adults with obesity often comes from secondary analyses or observational data. More prospective studies are needed to determine which patients benefit most from weight loss, exercise, dietary intervention, biologic therapy, metabolic treatment, or combined approaches. Trials should include inflammatory biomarkers, body composition measures, patient-reported outcomes, lung function, exacerbations, and comorbidity assessment.

Despite these limitations, the available evidence supports a clinically useful conclusion: obesity-related inflammation is an important disease modifier in adult asthma, but it acts within a broader network of mechanisms. Management should therefore be phenotype-based and treatable-trait oriented. In practice, this means assessing airway inflammation, confirming asthma diagnosis, identifying obesity-related comorbidities, addressing weight and metabolic health, and avoiding reflexive escalation of corticosteroids when symptoms may be driven by non-eosinophilic mechanisms or comorbid disease.

Future research should focus on identifying clinically usable biomarkers of obesity-related asthma, clarifying the role of IL-6 and adipokine pathways, determining how obesity modifies biologic therapy response, and evaluating integrated interventions that combine asthma pharmacotherapy with weight management and metabolic treatment. Such studies would help move the field from describing an association toward selecting mechanism-based treatments for individual patients.

Clinical and Public Health Implications

The clinical importance of obesity-related inflammation in asthma is best understood through a treatable-traits approach. In this model, obesity is not treated as a background characteristic but as an active disease modifier that can influence symptoms, inflammatory pathways, medication response, and comorbidity burden. This approach is particularly useful because it moves the clinician away from a narrow question of whether asthma therapy should simply be intensified. Instead, it encourages a broader evaluation of why the patient remains symptomatic and which modifiable factors are driving poor outcomes.

In routine practice, adults with obesity and asthma should undergo careful assessment of symptom pattern, exacerbation history, lung function, inhaler technique, adherence, and exposure to triggers. At the same time, clinicians should evaluate obesity-related conditions that may mimic uncontrolled asthma, including obstructive sleep apnea, gastroesophageal reflux, vocal cord dysfunction, deconditioning, cardiac disease, and anxiety. This is essential because symptoms such as exertional dyspnea, nocturnal breathlessness, chest tightness, and fatigue may arise from multiple causes. Without this broader assessment, patients may receive higher doses of corticosteroids while the major driver of symptoms remains untreated.

The relationship between obesity, inflammation, and asthma also has implications for specialist referral. Adults with obesity and persistent symptoms despite medium- or high-dose inhaled corticosteroid therapy should not automatically be classified as having severe asthma. Before labeling asthma as severe, it is necessary to confirm the diagnosis, identify modifiable risk factors, and evaluate comorbidities. This is consistent with contemporary asthma management principles, which emphasize that difficult-to-treat asthma is often caused by factors other than refractory airway inflammation. Obesity is one of the most common of these factors and may require multidisciplinary management.

From a therapeutic perspective, weight reduction should be discussed as part of asthma management in a non-stigmatizing and clinically realistic manner. The goal is not only reduction of body weight but also improvement in respiratory mechanics, systemic inflammation, metabolic function, exercise tolerance, and quality of life. Even modest weight loss may improve symptoms in some individuals, whereas larger weight loss may be required to produce measurable changes in lung volumes or inflammatory markers. Patients should therefore receive individualized advice rather than generic instructions to lose weight.

Multidisciplinary care is particularly relevant in this population. Pulmonologists and allergists can optimize asthma pharmacotherapy and evaluate inflammatory phenotype. Primary care physicians can coordinate long-term cardiometabolic risk management. Dietitians can support sustainable nutritional changes. Physiotherapists and exercise specialists can help patients overcome breathlessness-related avoidance of activity. Sleep specialists may identify and treat obstructive sleep apnea. Psychologists may support behavioral change, anxiety management, and adherence. Such integrated care is more likely to address the full clinical burden of obesity-related asthma than medication escalation alone.

The public health implications are also considerable. Obesity and asthma are both common, and their coexistence increases health-care utilization. Prevention of obesity, promotion of physical activity, improvement in diet quality, and early management of metabolic risk may therefore have respiratory benefits. Conversely, poorly controlled asthma may reduce physical activity and contribute to weight gain, creating a cycle of worsening symptoms and reduced fitness. Interventions that break this cycle may improve both respiratory and metabolic outcomes.

Limitations

This review has several limitations. First, the available literature is heterogeneous in study design, population characteristics, asthma definitions, obesity measures, inflammatory markers, and outcome measures. This makes it difficult to directly compare results across studies. Some studies define obesity using body mass index alone, whereas others include waist circumference, body composition, or metabolic syndrome. Because visceral adiposity and metabolic dysfunction may be more closely related to inflammation than BMI, reliance on BMI alone may obscure important biological differences.

Second, much of the evidence is observational. Observational studies can identify associations but cannot fully establish causality. Obesity may increase asthma risk, but asthma may also contribute to weight gain through reduced physical activity, corticosteroid exposure, sleep disturbance, and psychological burden. Bidirectional relationships are likely. Therefore, causal claims should be made cautiously, especially when discussing the effect of systemic inflammation on asthma onset.

Third, the inflammatory mechanisms described in the literature are not specific to asthma. IL-6, TNF-alpha, leptin, oxidative stress, and insulin resistance are involved in many cardiometabolic and inflammatory diseases. Their presence in adults with obesity and asthma does not necessarily prove that they are directly responsible for airway disease. Future studies should distinguish between biomarkers that simply reflect obesity and biomarkers that predict asthma outcomes or treatment response.

Fourth, the evidence regarding treatment is still evolving. Weight loss appears beneficial for many adults with obesity-related asthma, but the optimal intervention, duration, magnitude of weight loss, and long-term sustainability remain uncertain. Evidence for newer pharmacological obesity treatments in asthma-specific populations is limited, and the impact of biologic asthma therapies in patients with severe obesity requires further study. As a result, current clinical recommendations should emphasize comprehensive individualized care rather than a single universal intervention.

Finally, this review did not perform a quantitative meta-analysis or formal risk-of-bias grading. The aim was to provide a clinically oriented synthesis of current evidence rather than estimate pooled effect sizes. The conclusions should therefore be interpreted as a structured overview of the literature and a basis for further research and manuscript development.

Future Directions

Future research should focus on defining obesity-related asthma endotypes with greater precision. Studies should move beyond BMI and incorporate waist circumference, body composition, metabolic markers, inflammatory biomarkers, lung function, imaging, sleep assessment, and comorbidity profiles. Such multidimensional phenotyping could identify subgroups of patients who are most likely to benefit from specific interventions.

There is also a need for randomized controlled trials evaluating integrated management strategies in adults with obesity and asthma. These trials should compare usual asthma care with interventions that combine optimized inhaled therapy, structured weight management, exercise, dietary support, treatment of sleep apnea and reflux, and psychological support where needed. Outcomes should include asthma control, exacerbations, quality of life, lung function, systemic inflammation, metabolic health, and health-care utilization.

The role of emerging obesity therapies deserves particular attention. Modern pharmacological treatments for obesity may produce clinically meaningful weight loss and improve metabolic dysfunction. Whether these benefits translate into improved asthma outcomes, reduced airway inflammation, or decreased corticosteroid exposure remains an important research question. Trials should be designed specifically for adults with asthma and obesity rather than relying only on subgroup analyses from obesity trials.

Finally, future studies should examine implementation in real-world practice. Even when evidence supports weight management and comorbidity treatment, access to multidisciplinary care is often limited. Cost, availability, insurance coverage, stigma, and time constraints may prevent patients from receiving comprehensive support. Research should therefore evaluate not only biological mechanisms but also practical models of care that can be delivered in routine clinical settings.

Conclusions

Obesity-related chronic inflammation appears to play a meaningful role in the pathogenesis and severity of asthma in adults. The relationship between obesity and asthma is mediated by multiple mechanisms, including systemic low-grade inflammation, altered adipokine signaling, IL-6-associated metabolic dysfunction, oxidative stress, airway neutrophilia, reduced lung volumes, and obesity-related comorbidities. These mechanisms may increase asthma risk, worsen symptom burden, reduce disease control, and complicate therapeutic response.

Obesity-related asthma should not be considered a single uniform phenotype. Adults with asthma and obesity may present with type 2-high, type 2-low, neutrophilic, mixed, or mechanically driven disease patterns. For this reason, clinical management should include objective confirmation of asthma, assessment of inflammatory phenotype, evaluation of comorbidities, and recognition of obesity as a modifiable treatable trait.

Weight management, physical activity, dietary improvement, and treatment of comorbidities should be integrated with conventional asthma therapy. Future research should determine which inflammatory and metabolic markers best identify patients most likely to benefit from targeted weight-loss, anti-inflammatory, biologic, or metabolic interventions.

Table 1. Main mechanisms linking obesity-related chronic inflammation with asthma in adults.

Mechanism	Pathophysiological relevance	Possible clinical consequence
Adipose tissue inflammation	Macrophage infiltration and adipocyte hypertrophy increase systemic inflammatory mediators.	Higher symptom burden and possible amplification of airway inflammation.
Adipokine imbalance	Increased leptin and reduced adiponectin may favor pro-inflammatory signaling.	Altered immune response and potential worsening of asthma control.
IL-6-associated metabolic inflammation	IL-6 links metabolic dysfunction with systemic inflammation.	Association with severe asthma, poorer lung function, and metabolic comorbidity.
Neutrophilic airway inflammation	Obesity may be associated with higher sputum neutrophils in adults with asthma.	Potential corticosteroid insensitivity and persistent symptoms.
Oxidative stress	Obesity increases reactive oxygen species and reduces antioxidant reserve.	Airway epithelial injury, bronchial hyperresponsiveness, and treatment complexity.
Mechanical restriction	Reduced lung volumes and increased airway closure interact with inflammation.	Dyspnea, exercise limitation, and overestimation of asthma severity.

Table 2. Clinical characteristics commonly described in adults with obesity-related asthma.

Clinical domain	Common finding	Interpretation
Symptoms	Greater dyspnea and poorer perceived control	May reflect asthma activity, mechanics, deconditioning, or comorbidities.
Inflammation	Variable type 2, neutrophilic, mixed, or low inflammatory patterns	Phenotype assessment is needed before treatment escalation.
Lung function	Reduced lung volumes and possible peripheral airway closure	Mechanical effects can amplify symptoms and airway hyperresponsiveness.
Comorbidities	OSA, GERD, rhinosinusitis, anxiety, depression, metabolic syndrome	Comorbidities may mimic or worsen asthma symptoms.
Therapeutic response	Possible reduced corticosteroid responsiveness in some patients	Persistent symptoms should prompt reassessment rather than automatic dose escalation.
Treatment strategy	Weight management and treatable-trait approach	Integrated care may improve outcomes beyond inhaled pharmacotherapy.

Table 3. Practical implications for management of asthma in adults with obesity.

Clinical step	Purpose	Examples
Confirm asthma diagnosis	Avoid treating non-asthma dyspnea as uncontrolled asthma.	Spirometry, bronchodilator response, variability assessment.
Assess inflammatory phenotype	Match treatment to dominant airway inflammation.	Blood eosinophils, FeNO, IgE, exacerbation history where available.
Screen comorbidities	Identify treatable drivers of symptoms.	OSA, GERD, rhinosinusitis, depression, cardiac disease.
Address weight and metabolic health	Modify upstream disease drivers.	Nutrition, exercise, behavioral support, pharmacological or surgical obesity treatment when appropriate.

Clinical step	Purpose	Examples
Review steroid exposure	Reduce avoidable weight-promoting treatment burden.	Optimize inhaler technique, adherence, and steroid-sparing options when indicated.
Use multidisciplinary care	Support realistic long-term change.	Pulmonology, allergy, primary care, dietetics, physiotherapy, sleep medicine.

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