



Novel understanding in diagnosis and management of exercise-induced bronchoconstriction

John W. Dickinson

To cite this article: John W. Dickinson (14 Oct 2026): Novel understanding in diagnosis and management of exercise-induced bronchoconstriction, Expert Review of Clinical Immunology, DOI: [10.1080/1744666X.2026.2747863](https://doi.org/10.1080/1744666X.2026.2747863)

To link to this article: <https://doi.org/10.1080/1744666X.2026.2747863>



© 2026 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group.



Published online: 14 Oct 2026.



Submit your article to this journal [↗](#)



Article views: 7



View related articles [↗](#)



View Crossmark data [↗](#)

REVIEW



Novel understanding in diagnosis and management of exercise-induced bronchoconstriction

John W. Dickinson

Sport, Exercise, and Rehabilitation Sciences, School of Natural Sciences, University of Kent, Canterbury, UK

ABSTRACT

Introduction: Exercise-induced bronchoconstriction (EIB) is a common cause of exercise-related respiratory symptoms in athletes and is traditionally diagnosed by demonstrating airway narrowing following exercise or surrogate bronchoprovocation testing. Whilst this provides objective evidence of bronchoconstriction, it offers limited insight into the underlying pathophysiology of airway dysfunction.

Areas covered: This review discusses emerging developments in EIB, focusing on biomarkers of airway inflammation and injury, respiratory oscillometry, breathing pattern assessment and wearable respiratory monitoring. The concept that EIB may represent the clinical manifestation of a broader process of airway injury and remodeling is also considered. Relevant literature was identified through searches of PubMed and Google Scholar with no lower date restriction to May 2026, supplemented by reference-list searching.

Expert opinion: The future challenge in EIB is no longer simply confirming bronchoconstriction but identifying athletes at risk of airway dysfunction before clinically significant disease develops. Emerging biomarkers, oscillometry and continuous respiratory monitoring may enable earlier phenotyping and more personalized management than conventional bronchoprovocation testing alone. Collectively, these approaches have the potential to shift EIB care from diagnosing established disease toward preserving airway health within a holistic model of athlete respiratory care.

ARTICLE HISTORY

Received 2 July 2026
Accepted 8 October 2026

KEYWORDS

Airway injury; respiratory oscillometry; breathing pattern; wearable technology; respiratory monitoring; airway health

1. Introduction

Exercise-induced bronchoconstriction (EIB) is characterized by transient narrowing of the airways that occurs during or following exercise [1]. The high ventilatory demands of exercise increase respiratory heat and water loss, resulting in airway cooling and dehydration. The consequent increase in airway surface osmolarity promotes cell shrinkage and the release of inflammatory mediators that stimulate airway smooth muscle contraction, while post-exercise rewarming may further contribute to airway narrowing [2]. EIB may occur in individuals with or without a preexisting diagnosis of asthma and represents one of the most common causes of exercise-related respiratory symptoms in athletic populations [3–5].

Recent systematic reviews estimate that approximately 22% of athletes demonstrate objective evidence of EIB [6], although the prevalence varies considerably (8–68%) according to sport, competitive level and environmental exposure [3,4,7]. The prevalence is substantially higher than that reported in the general population and is particularly evident in sports characterized by high ventilatory demands or exposure to adverse environmental conditions. For example, prevalence estimates exceeding 40% have been reported in elite cross-country skiers exposed to cold, dry air [8–11] and in competitive swimmers exposed to chlorine-rich environments [7,11,12]. These observations suggest that participation in certain sporting environments may contribute to the development of airway dysfunction and highlight the importance of effective diagnostic and management strategies.

One of the most consistent observations across studies is the poor relationship between respiratory symptoms and objective evidence of airway dysfunction [13,14]. Athletes frequently report symptoms such as breathlessness, chest tightness, wheeze or excessive mucus production during exercise, yet many demonstrate negative bronchoprovocation tests [15]. These symptoms are not exclusive to EIB and could be due to a number of conditions (Table 1). Conversely, athletes with no previous EIB history and/or do not report symptoms may exhibit significant airway hyperresponsiveness when formally assessed [4,16]. As a result, symptom-based diagnosis continues to result in both over-diagnosis and under-diagnosis of EIB, reinforcing the importance of objective assessment.

A limitation of current diagnostic pathways is that they focus primarily on detecting the physiological consequence of airway dysfunction rather than the mechanisms responsible for disease activity. Regardless of the challenge employed, current assessments ultimately rely on demonstrating a reduction in forced expiratory volume in one second (FEV₁) following airway provocation (see Table 2). Whilst this provides objective evidence of bronchoconstriction, it offers limited insight into the pathophysiological processes contributing to airway narrowing, including airway inflammation, epithelial injury, airway remodeling and environmental influences. Furthermore, no single diagnostic test can reliably confirm or exclude EIB in all athletes. Indirect bronchoprovocation challenges provide objective evidence of

Article highlights

- Current diagnostic pathways for exercise-induced bronchoconstriction (EIB) accurately identify bronchoconstriction but provide limited insight into the underlying pathophysiology of airway dysfunction.
- Airway dysfunction in athletes is heterogeneous, with similar bronchoprovocation responses potentially arising through differing pathophysiological processes involving type 2 inflammation and airway injury.
- Biomarkers of airway inflammation and epithelial injury, including FeNO and CC16, may complement bronchoprovocation testing by improving disease phenotyping and identifying athletes at risk of airway dysfunction.
- Respiratory oscillometry offers a promising adjunct to spirometry by providing complementary assessment of airway mechanics and may detect physiological abnormalities not identified by conventional lung function testing.
- Advances in breathing pattern assessment and wearable respiratory monitoring technologies have the potential to enable continuous, real-world evaluation of respiratory function beyond the laboratory.
- Future approaches to EIB should move beyond confirming bronchoconstriction toward preserving airway health through earlier identification of airway injury and physiological dysfunction, with emerging biomarkers, physiological assessments and digital monitoring technologies potentially facilitating more personalized and holistic respiratory care.

Table 1. Common causes of exercise respiratory symptoms associated with EIB.

Exercise Induced Bronchoconstriction
Cardiovascular disease
Lack of fitness/returning to play
Rhinitis
Exercise Induced Laryngeal Obstruction
Breathing Pattern Disorder
Large Airway Collapse
Diaphragm Paralysis
Musculoskeletal injury or tightness around the torso

Table 2. Different types of bronchoprovocation challenges and the fall in FEV₁ required for a test deemed to be positive and indicative of EIB.

Assessment	Type	Positive test
Methacholine	Direct	≥20% fall in FEV ₁
Histamine	Direct	≥20% fall in FEV ₁
Exercise Challenge	Indirect	≥10% fall in FEV ₁
Eucapnic Voluntary Hyperpnoea (EVH)	Indirect	≥10% fall in FEV ₁
Mannitol	Indirect	≥10–15% fall in FEV ₁
Hypertonic Saline	Indirect	≥15% fall in FEV ₁

EIB when positive, but a negative result does not necessarily exclude clinically relevant airway dysfunction. Conversely, direct challenges may be useful for excluding significant airway hyper-responsiveness but lack specificity for EIB. These limitations, together with the considerable biological variability in test responses and the poor relationship between symptoms and objective evidence of airway dysfunction, mean that current assessments are effective at identifying bronchoconstriction but provide limited information regarding disease phenotype, future risk of airway dysfunction or likely treatment responsiveness.

Considerable progress has been made in the diagnosis and management of EIB over the past two decades, resulting in the development of standardized bronchoprovocation protocols and evidence-based treatment recommendations [17–19].

These advances have significantly improved diagnostic accuracy and reduced reliance on symptom-based diagnosis alone [20].

The future challenge is no longer simply identifying whether bronchoconstriction occurs following exercise or a surrogate challenge such as eucapnic voluntary hyperpnea (EVH). Rather, it is determining how best to characterize airway health in athletic populations and identify those athletes at risk of developing clinically relevant airway dysfunction. Addressing this challenge will require diagnostic approaches that move beyond traditional measures of airflow limitation and incorporate novel assessments of airway inflammation, airway mechanics and real-world respiratory function. The concepts discussed throughout this review are summarized in Figure 1.

Accordingly, the purpose of this review is not to revisit established diagnostic algorithms or pharmacological treatment pathways, which have been comprehensively addressed in international consensus statements. Rather, this article examines the limitations of current approaches and discusses emerging developments that may shape the future diagnosis and management of EIB. Particular focus is placed on novel methods of assessing airway inflammation, the potential role of oscillometry in identifying exercise-related airway dysfunction, advances in digital and field-based monitoring technologies, and the emerging importance of breathing pattern assessment within athlete respiratory care.

1.1. Literature search

This article represents an expert narrative review rather than a systematic review. Relevant literature was identified through searches of PubMed and Google Scholar with no lower date restriction, with the final search conducted in May 2026. Searches included combinations of terms relating to EIB, airway inflammation and injury, airway remodeling, respiratory oscillometry, breathing pattern and wearable respiratory monitoring. Additional publications were identified from the reference lists of relevant articles and from literature known to the author through research in the field.

2. Can assessments of airway inflammation and airway injury improve the diagnosis and management of EIB?

Current bronchoprovocation tests provide objective evidence of exercise-induced airway narrowing but offer limited insight into the mechanisms responsible for the response. Consequently, athletes demonstrating similar reductions in FEV₁ following exercise challenge may have markedly different underlying airway pathology [21,22]. This observation has led to increasing interest in whether assessments of airway inflammation and airway injury can provide additional information that may improve the diagnosis and management of EIB.

There is increasing recognition that airway dysfunction in athletes is heterogeneous. The proposed biological heterogeneity of EIB is summarized in Figure 2. Some athletes

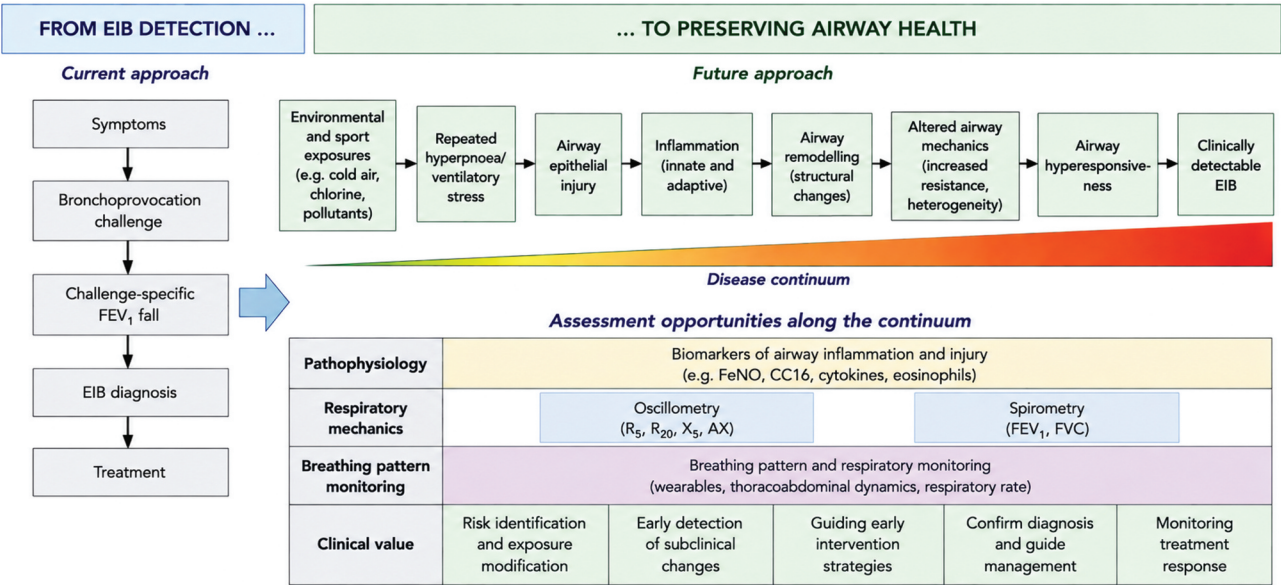


Figure 1. From EIB Detection to Preserving Airway Health. Current diagnostic pathways for exercise-induced bronchoconstriction (EIB) focus on confirming bronchoconstriction through demonstration of a post-challenge reduction in FEV₁ following exercise or surrogate bronchoprovocation testing. In contrast, future approaches may increasingly focus on identifying biological and physiological changes that occur throughout the development of airway dysfunction. Repeated exposure to high ventilatory demands and adverse environmental conditions may contribute to airway epithelial injury, inflammation, airway remodeling and altered airway mechanics before clinically detectable EIB develops. This disease continuum creates opportunities for earlier assessment using biomarkers of airway inflammation and injury, oscillometry-derived measures of airway mechanics and breathing pattern monitoring technologies. Collectively, these approaches may facilitate a transition from diagnosing established EIB toward preserving airway health throughout an athlete's career. This figure was generated using ChatGPT's image-generation capability and was iteratively reviewed and revised by the author to ensure scientific accuracy and clarity of presentation.

Abbreviations: AX, area of reactance; CC16, club cell secretory protein 16; EIB, exercise-induced bronchoconstriction; EVH, eucapnic voluntary hyperpnea; FeNO, fractional exhaled nitric oxide; FEV₁, forced expiratory volume in one second; FVC, forced vital capacity; R₅, resistance at 5 Hz; R₂₀, resistance at 20 Hz; X₅, reactance at 5 Hz.

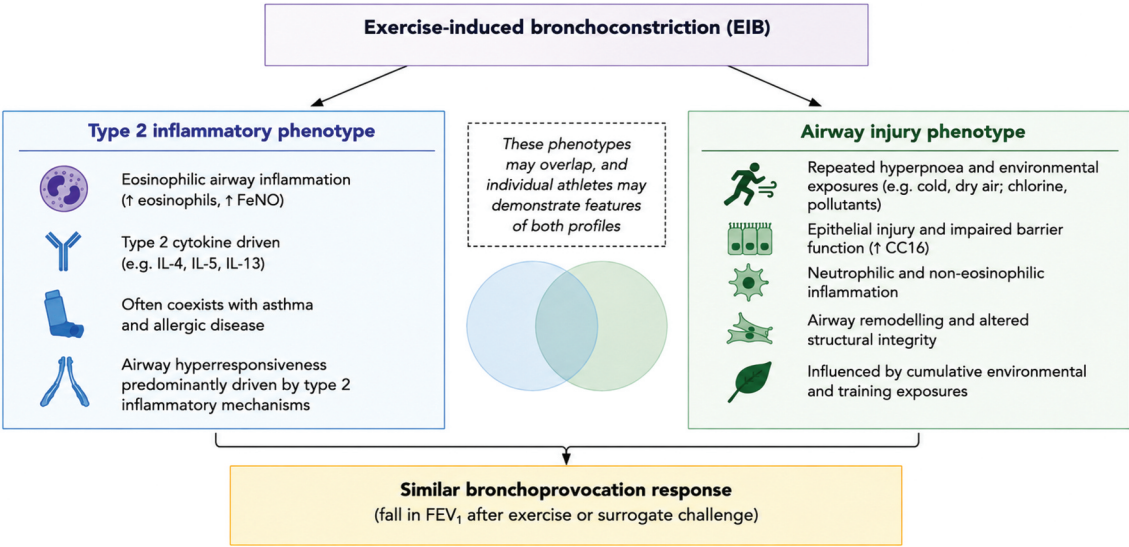


Figure 2. Heterogeneity of airway dysfunction in athletes. Athletes with objective evidence of exercise-induced bronchoconstriction (EIB) may demonstrate differing pathophysiological profiles. A type 2 inflammatory phenotype is characterized by eosinophilic airway inflammation, elevated FeNO and type 2 cytokine activity. In contrast, an airway injury phenotype is associated with repeated ventilatory stress and environmental exposures that promote epithelial injury, impaired barrier function, non-eosinophilic inflammation and airway remodeling. These profiles may overlap, and both may produce similar reductions in FEV₁ following bronchoprovocation testing. Recognition of this heterogeneity may facilitate more personalized approaches to the diagnosis and management of EIB. This figure was generated using ChatGPT's image-generation capability and was iteratively reviewed and revised by the author to ensure scientific accuracy and clarity of presentation.

Abbreviations: CC16, club cell secretory protein 16; EIB, exercise-induced bronchoconstriction; FeNO, fractional exhaled nitric oxide; FEV₁, forced expiratory volume in one second.

demonstrate features consistent with type-2 airway inflammation [23], typically characterized by eosinophilic inflammation and elevated FeNO, whereas others appear to develop airway dysfunction through repeated exposure to high ventilatory demands and adverse environmental conditions such as cold dry air, chlorination by-products and airborne pollutants [24,25]. In these athletes, repeated airway dehydration and epithelial injury may initiate complex inflammatory and repair

responses involving multiple cellular and molecular pathways that are not adequately reflected by conventional markers of type-2 airway inflammation [26,27]. These mechanisms should not necessarily be considered mutually exclusive, and individual athletes may demonstrate overlapping features [2]. Collectively, this heterogeneity suggests that similar bronchoprovocation responses may arise through differing pathophysiological processes [28].

Fractional exhaled nitric oxide (FeNO) is currently the most widely available noninvasive marker of airway inflammation and has received considerable attention in EIB research. Elevated FeNO levels are associated with eosinophilic airway inflammation [29]. Studies in pediatric populations have investigated whether FeNO may also help predict EIB, with higher FeNO associated with an increased likelihood and severity of bronchoconstriction following exercise [30,31]. However, the predictive performance of FeNO has varied between studies, and FeNO alone has not consistently provided sufficient diagnostic accuracy to determine the need for exercise testing. Similarly, whilst higher FeNO values provide reasonable specificity for identifying athletes with objective evidence of EIB, sensitivity remains poor and many athletes with positive bronchoprovocation challenges exhibit normal FeNO values [23]. These findings suggest that FeNO identifies only one inflammatory phenotype of EIB. Consequently, a normal FeNO value should not be interpreted as evidence of normal airway health, but rather as an indication that alternative inflammatory pathways or mechanisms of airway dysfunction may be contributing to bronchoconstriction. Nevertheless, in athletes demonstrating both elevated FeNO and objective evidence of EIB, serial FeNO measurements may provide a useful means of monitoring airway inflammation and assessing response to anti-inflammatory therapy [32,33].

The observation that many athletes with objective EIB demonstrate normal FeNO values has stimulated interest in mechanisms beyond eosinophilic inflammation. Exercise-induced bronchoconstriction is thought to arise through a complex interaction between airway dehydration, thermal stress, epithelial injury and inflammatory mediator release. Whilst eosinophilic inflammation may be important in some athletes, other pathways involving mast-cell derived mediators such as histamine, leukotrienes and prostaglandins are also recognized contributors to exercise-induced bronchoconstriction. In addition, studies in athletic populations have identified evidence of non-type-2 inflammatory responses and airway injury, particularly in athletes exposed to adverse environmental conditions such as cold dry air and chlorinated swimming pools. Consequently, FeNO provides valuable insight into one aspect of airway inflammation but is unlikely to capture the full spectrum of biological processes contributing to EIB.

2.1. Airway epithelium damage and airway remodeling

Over the past two decades, increasing evidence has emerged suggesting that repeated exposure to exercise hyperpnea may result in airway epithelial injury [34,35]. The airway epithelium represents the primary interface between the external environment and the respiratory system and is increasingly recognized as a central component in the pathogenesis of EIB. Repeated

cycles of airway dehydration and cooling during exercise may disrupt epithelial integrity, facilitating inflammatory responses and initiating repair processes that, over time, contribute to airway remodeling and airway hyperresponsiveness [35].

Evidence supporting this concept has been demonstrated across multiple athletic populations. The cumulative ventilatory stress imposed on the airway epithelium is unlikely to be equivalent across sporting disciplines and may reflect not only the magnitude, duration and frequency of high ventilation, but also the environmental conditions in which this occurs. Consequently, athletes repeatedly exposed to high ventilation in cold dry air, polluted environments or air containing respiratory irritants may experience a greater airway epithelial burden than athletes undertaking similar ventilatory demands in more favorable conditions. Studies in elite cross-country skiers, who repeatedly undertake high ventilation in cold dry air, have identified inflammatory changes and structural airway remodeling in athletes without a clinical diagnosis of asthma [24]. Similarly, investigations in competitive swimmers have demonstrated airway epithelial damage, increased permeability, innate cytokine activation and neutrophilic airway inflammation associated with chronic exposure to chlorination by-products [36,37]. Collectively, these findings suggest that the potential for airway injury may reflect the interaction between ventilatory demand, cumulative exposure and the specific environmental conditions in which exercise is performed, and may occur independently of clinically apparent EIB.

Several biomarkers of airway injury have been investigated in an attempt to better characterize these processes. Club cell secretory protein 16 (CC16) is among the most extensively studied. CC16 is produced by epithelial cells within the distal airways and increases in circulation and urine following disruption of epithelial barrier integrity. Studies employing exercise and EVH challenges have consistently demonstrated increases in CC16 following hyperpnea [38]. Importantly, these increases occur not only in athletes with EIB but also in athletes without demonstrable bronchoconstriction [39]. Such findings suggest that airway epithelial perturbation may be a common consequence of high ventilatory exercise and that epithelial injury can occur independently of measurable reductions in FEV₁.

However, the clinical significance of exercise-induced changes in CC16 remains uncertain. Previous work has demonstrated acute changes in CC16 following exercise or hyperpnea [38], including in athletes without demonstrable bronchoconstriction, but it has not been established whether these changes are associated with respiratory symptoms or predict the subsequent development of EIB or asthma. Similarly, the relationship between CC16 and oscillometry-derived measures of airway mechanics has not been adequately characterized. Prospective longitudinal studies combining biomarkers of airway injury with symptoms, bronchoprovocation and measures of airway mechanics are therefore required to determine whether CC16 can identify athletes at increased risk of developing clinically significant airway dysfunction.

The observation that airway injury may occur in athletes without objective evidence of EIB raises an intriguing possibility. Current diagnostic pathways identify athletes only once

airway dysfunction becomes sufficiently severe to produce bronchoconstriction during challenge testing. However, airway injury, inflammation and remodeling may precede the development of clinically detectable EIB by many years. From this perspective, EIB may represent the clinical manifestation of an underlying process that begins with repeated airway injury and culminates in airway hyperresponsiveness and bronchoconstriction (Figure 3).

This concept aligns closely with previous proposals that airway dysfunction in athletes may represent an occupational disease [40]. Airway dysfunction is most prevalent in sports characterized by sustained high ventilatory demands and chronic exposure to potentially harmful environmental conditions, including cold dry air, chlorination by-products and airborne pollutants and particulate matter [3,4,6]. The repeated exposure of athletes to these environments throughout training and competition may therefore be analogous to occupational exposure observed in other work settings. Whether airway dysfunction in athletes should be formally classified as an occupational disease remains open to debate. Nevertheless, the concept highlights an important shift in perspective. Rather than viewing EIB solely as a condition requiring diagnosis and treatment once bronchoconstriction has developed, there is increasing recognition that preserving airway health throughout an athlete's career may be equally important.

A major barrier to identifying airway injury before clinically significant dysfunction develops is the limited availability of accessible assessment tools. This contrasts with FeNO, which can be measured rapidly and non-invasively at the point of care and provides an established marker of type 2 airway inflammation, although it is not specific to EIB. Current measures of airway remodeling, epithelial injury and inflammatory profiling remain largely confined to research environments.

Bronchial biopsy remains the most direct method of assessing structural airway remodeling but is invasive and unsuitable for routine use in athletic populations [24]. Induced sputum, exhaled breath condensate and circulating biomarkers such as CC16 have contributed substantially to understanding airway inflammation and injury in athletes [27]. Studies using exhaled breath condensate in competitive athletes provide further evidence of its research utility [41], but their translation into routine athlete respiratory assessment remains challenging. Induced sputum requires carefully standardized induction and processing procedures and appropriate clinical supervision [42], while exhaled breath condensate requires standardization of collection, storage and analytical procedures, with methodological variability limiting interpretation and clinical application [43]. Blood-based inflammatory profiling presents similar translational challenges, requiring invasive sample collection, appropriately trained personnel and access to analytical facilities. Similarly, CC16 currently requires biological sample collection and analysis, limiting its application in routine clinical practice despite its potential value as a marker of airway epithelial barrier disruption. Consequently, although these approaches provide valuable insight into airway pathology, their current practical requirements limit the feasibility of repeated surveillance within most sporting environments.

The development of accessible methods for monitoring airway injury may create a new clinical challenge, as there is currently little consensus regarding how athletes demonstrating airway injury in the absence of EIB should be managed. Nevertheless, the concept of monitoring airway health rather than simply detecting bronchoconstriction represents an important shift in perspective. The ultimate goal should not be to identify airway dysfunction only after clinically significant EIB has developed, but rather to identify athletes at increased risk of progression and intervene early enough to

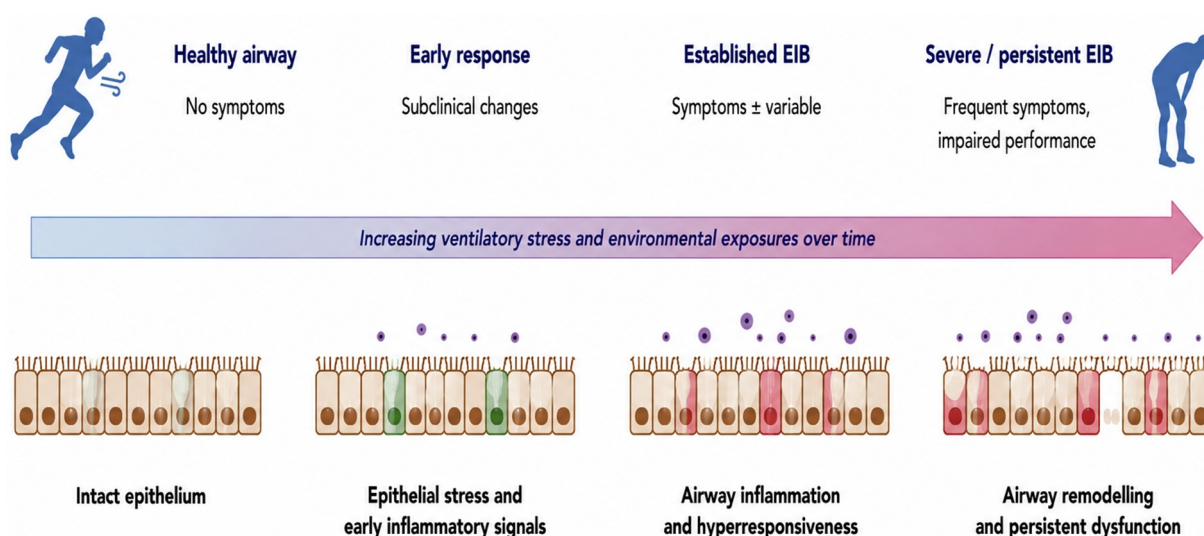


Figure 3. A continuum of airway responses in athletes: opportunities to preserve airway health.

Repeated exposure to high ventilatory demands and adverse environmental conditions may drive a continuum of airway responses in athletes. This process may begin with a structurally healthy airway and progress through epithelial stress and early inflammatory signaling to airway inflammation, hyperresponsiveness and, ultimately, clinically detectable exercise-induced bronchoconstriction (EIB). In some athletes, continued exposure may contribute to airway remodeling and persistent airway dysfunction. This continuum highlights the opportunity to identify biological and physiological changes before clinically significant EIB develops and supports a shift toward earlier intervention aimed at preserving airway health. This figure was generated using ChatGPT's image-generation capability and was iteratively reviewed and revised by the author to ensure scientific accuracy and clarity of presentation.

Abbreviations: EIB, exercise-induced bronchoconstriction; FEV₁, forced expiratory volume in one second.

preserve long-term respiratory health. Future research should therefore focus on developing accessible biomarkers of airway injury and determining whether early intervention can alter the trajectory from airway injury to clinically significant EIB.

3. Can oscillometry improve the diagnosis and management of EIB?

The diagnosis of EIB remains heavily dependent on spirometry and the demonstration of a reduction in forced expiratory volume in one second (FEV_1) following exercise or surrogate bronchoprovocation testing (Table 2; [17–19]). Spirometry is widely available, relatively inexpensive and supported by well-established diagnostic thresholds [17–19]. Consequently, a post-challenge reduction in FEV_1 remains the cornerstone of EIB diagnosis. However, spirometry is not without limitations. The technique requires maximal respiratory effort and is dependent on patient cooperation and test quality [44,45]. Furthermore, spirometry primarily reflects airflow and volume characteristics of the lower airway, and provides limited information regarding airway resistance and the mechanical properties of the respiratory system.

These limitations have stimulated interest in alternative physiological assessments that may provide additional insight into airway dysfunction. Respiratory oscillometry, also referred to as the forced oscillation technique (FOT) or impulse oscillometry (IOS), measures respiratory system resistance and reactance during normal tidal breathing through the application of small external pressure oscillations at the mouth [46]. In simple terms, oscillometry assesses how easily these pressure waves travel through the respiratory system, providing information on airway caliber and the mechanical properties of the lungs, particularly within the peripheral airways. Unlike spirometry, oscillometry requires minimal patient effort and can be performed repeatedly without the need for forced expiratory maneuvers. These characteristics have supported its increasing use as an adjunct to conventional lung function assessment in respiratory clinical practice, particularly where spirometry is difficult to perform or additional information regarding respiratory mechanics is required. However, oscillometry is not currently established as part of routine diagnostic assessment for EIB in athletes. As a result, oscillometry offers a fundamentally different assessment of respiratory physiology and may identify subtle alterations in airway mechanics before clinically significant changes in spirometric indices become apparent.

Interest in oscillometry has increased considerably over the last decade because of its potential sensitivity to changes occurring within the peripheral airways, which may not be detected by conventional spirometric assessment [46]. This is particularly relevant in EIB, where airway dysfunction may develop before substantial reductions in spirometric indices become apparent [24,47]. Several studies have demonstrated significant changes in oscillometric measures following exercise challenge and eucapnic voluntary hyperpnea (EVH), with some reports suggesting that oscillometry may identify airway abnormalities in athletes who do not meet conventional spirometric criteria for EIB [48,49]. To date, most EIB studies have

focused on oscillometric variables such as resistance at 5 Hz (R_5), area of reactance (AX) and resonant frequency (F_{res}), which demonstrate sensitivity to bronchoconstrictive responses following EVH and exercise challenge testing [48,49]. However, advances in oscillometry and increasing interest in small airway physiology raise the possibility that other indices may provide greater sensitivity to early airway dysfunction than the parameters explored in the current EIB literature.

The potential value of oscillometry in EIB lies not in replacing spirometry, but in providing complementary information regarding airway mechanics. When considered alongside the emerging evidence of airway injury and remodeling, oscillometry offers the possibility of detecting subtle physiological abnormalities in the lower airways before clinically significant reductions in FEV_1 occur. This may be particularly relevant in athletes with exercise-related respiratory symptoms despite normal spirometry or those with evidence of airway injury where conventional lung function remains preserved. If airway injury represents an early stage in the development of airway dysfunction, oscillometry may help identify athletes at increased risk of progression before overt airway hyperresponsiveness and clinically detectable EIB develop.

Despite encouraging findings, the role of oscillometry in EIB assessment remains incompletely defined. Although oscillometry-derived measures of airway mechanics appear sensitive to bronchoconstrictive responses and may identify abnormalities not apparent on conventional spirometry, the current evidence base is relatively small and there is insufficient evidence to establish standardized diagnostic thresholds for athletic populations [46,49]. Until clinically meaningful cutoff values are defined and validated, oscillometry is best viewed as an adjunctive assessment that complements spirometry. Future research should focus on establishing standardized protocols, defining clinically meaningful thresholds and determining whether oscillometric abnormalities predict the subsequent development of airway hyperresponsiveness and EIB. The greatest potential value of oscillometry may ultimately not be the diagnosis of established EIB, but its ability to identify subtle abnormalities in airway mechanics before clinically significant reductions in FEV_1 become apparent. Such an approach would align closely with the emerging concept of preserving airway health throughout an athlete's career rather than simply detecting disease once it has become established.

4. Can continuous monitoring of breathing patterns transform the management of EIB?

Current assessment of EIB remains episodic. Athletes typically undergo respiratory evaluation only when objective testing is required to confirm an EIB diagnosis or assess management of inhaler therapy. Consequently, clinical decision-making is often based on isolated measurements obtained within laboratory environments that may not reflect respiratory health and EIB status during training, competition or daily life. This contrasts with many other areas of sports science and medicine where continuous physiological monitoring has become commonplace [50–52].

Recent advances in breathing pattern assessment raise the possibility that respiratory monitoring could become an important component of future EIB management. Traditionally, breathing pattern assessment has been used to identify breathing pattern disorders and other causes of exercise-related respiratory symptoms. However, emerging evidence suggests that breathing patterns may also provide insight into airway dysfunction itself [53].

Using optoelectronic plethysmography (OEP), recent work has demonstrated that individuals with EIB exhibit distinct alterations in breathing pattern during bronchoconstriction compared with healthy controls [53]. Specifically, bronchoconstriction was associated with changes in thoracoabdominal synchronization, including altered phase relationships between the upper and lower rib cage and shoulder compartments, together with changes in the sequence of chest wall compartment recruitment during breathing. Importantly, many of these abnormalities reversed following bronchodilator administration, suggesting that they were directly related to the bronchoconstriction rather than representing fixed breathing characteristics. Similar alterations in breathing pattern and thoracoabdominal synchronization have been reported in other respiratory conditions, including asthma and chronic obstructive pulmonary disease [54,55], indicating that the changes currently identified are unlikely to be specific to EIB. Rather, the changes observed during experimentally induced bronchoconstriction appear to represent a dynamic physiological response to airway narrowing. At present, breathing pattern should therefore not be considered diagnostic of EIB; however, identification of changes from an individual's normal breathing pattern could provide an indication that airway dysfunction is occurring and prompt further assessment or appropriate treatment. Future research should determine whether specific characteristics of breathing pattern can distinguish EIB from other causes of airway dysfunction and whether such changes can reliably inform clinical intervention.

At present, OEP represents the most comprehensive method of assessing breathing pattern and chest wall mechanics. The technique provides detailed information regarding compartmental contributions to breathing, thoracoabdominal synchronization and regional chest wall motion. However, OEP requires specialist equipment, multiple cameras, reflective markers and substantial post-processing, limiting its use to research and specialist clinical environments [56]. Consequently, despite its ability to characterize breathing patterns in considerable detail, OEP is unlikely to become a practical tool for routine monitoring of athletes.

The rapid expansion of wearable technologies may change this landscape. Advances in motion sensors, fiber optic sensing systems, smart textiles and wearable respiratory monitoring devices increasingly allow respiratory measurements to be obtained outside laboratory environments [57–59]. Studies have demonstrated the feasibility of wearable systems for monitoring variables such as respiratory rate and thoracoabdominal movement, including sensor-embedded garments and fiber-optic systems [57–60]. However, these studies have predominantly focused on technological feasibility and validation rather than the longitudinal detection or monitoring of EIB. Consequently, whether wearable-derived breathing

pattern metrics can reliably identify changes associated with bronchoconstriction or deteriorating airway health in athletes remains to be established.

The implications of such technology extend beyond diagnosis. Continuous monitoring of breathing patterns could provide a novel method of assessing disease control, detecting exacerbations and evaluating responses to treatment. More importantly, longitudinal monitoring may reveal subtle alterations in respiratory behavior before clinically significant airway dysfunction develops. Such an approach would complement emerging assessments of airway inflammation, airway injury and airway mechanics by providing a continuous physiological measure of respiratory health, potentially enabling earlier identification of athletes at risk of progressing toward clinically detectable EIB.

Significant challenges remain before these approaches can be implemented clinically. The breathing pattern changes most relevant to EIB have not yet been fully defined, normative data for athletic populations are lacking and the relationship between breathing pattern alterations and clinically meaningful outcomes remains uncertain. Furthermore, methods are required to translate complex laboratory-derived metrics into robust wearable measures that can be collected reliably in real-world environments.

Nevertheless, the convergence of respiratory physiology, wearable sensing technologies and digital health represents an exciting opportunity for the future management of EIB. The greatest potential value may not lie in diagnosing EIB itself, but in enabling continuous assessment of respiratory function and airway health throughout an athlete's career. Such an approach would fundamentally shift respiratory care from periodic testing toward personalized, real-world monitoring, allowing interventions to be guided by changes in respiratory physiology rather than isolated laboratory assessments.

5. From EIB detection to preserving airway health

Considerable progress has been made in the diagnosis and management of EIB over the past two decades. Objective bronchoprovocation testing has significantly improved diagnostic accuracy and remains essential for confirming the presence of exercise-induced airway narrowing. However, current diagnostic pathways remain largely focused on identifying the physiological consequence of airway dysfunction rather than the biological and physiological processes that contribute to its development.

The emerging evidence reviewed in this article suggests that future approaches to EIB assessment may increasingly focus on characterizing airway health rather than simply detecting bronchoconstriction. Assessments of airway inflammation and airway injury may help identify distinct disease phenotypes and provide insight into mechanisms that are not captured by conventional bronchoprovocation testing. Similarly, oscillometry offers the potential to detect subtle alterations in airway mechanics that may precede clinically significant reductions in FEV₁, whilst advances in breathing pattern assessment and wearable monitoring technologies raise the possibility of continuous real-world evaluation of respiratory function. A proposed framework for integrating

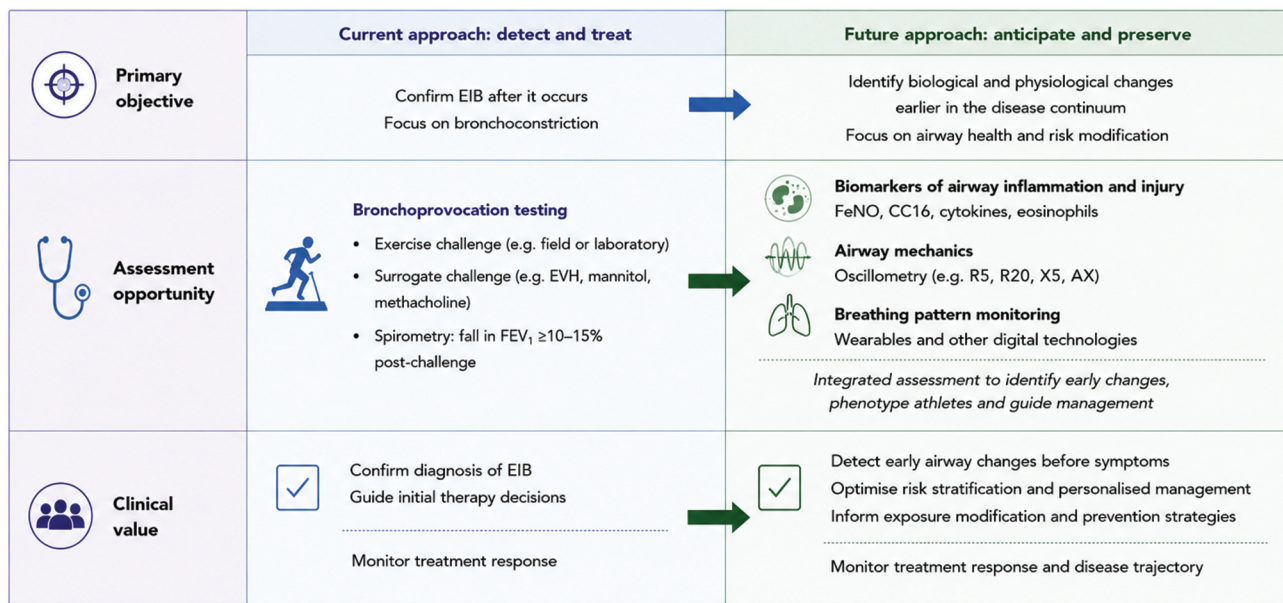


Figure 4. Integrating emerging technologies into future EIB diagnosis and management.

Current management of exercise-induced bronchoconstriction (EIB) focuses on confirming disease once bronchoconstriction has occurred using bronchoprovocation testing and spirometry. Emerging approaches seek to identify earlier biological and physiological changes through biomarkers of airway inflammation and injury, respiratory oscillometry and breathing pattern monitoring. Integrating these complementary assessments may facilitate earlier identification of athletes at risk of airway dysfunction, improve phenotyping, support personalized management strategies and enable interventions aimed at preserving airway health rather than simply diagnosing established disease. Monitoring treatment response remains an important component of both current and future clinical pathways. This figure was generated using ChatGPT's image-generation capability and was iteratively reviewed and revised by the author to ensure scientific accuracy and clarity of presentation.

Abbreviations: AX, area of reactance; CC16, club cell secretory protein 16; EIB, exercise-induced bronchoconstriction; EVH, eucapnic voluntary hyperpnea; FeNO, fractional exhaled nitric oxide; FEV₁, forced expiratory volume in one second; R5, resistance at 5 Hz; R20, resistance at 20 Hz; X5, reactance at 5 Hz.

these emerging technologies into future diagnostic and management pathways is presented in [Figure 4](#).

In practice, a strategy focused on preserving airway health could involve longitudinal assessment of athletes considered at increased risk of airway dysfunction, incorporating periodic evaluation of symptoms, spirometry and bronchoprovocation alongside complementary measures of airway inflammation and respiratory mechanics where clinically appropriate. As accessible biomarkers of epithelial injury and wearable respiratory monitoring technologies become available, these could provide additional opportunities to identify changes in airway health between formal clinical assessments. Such surveillance could also be combined with monitoring of environmental exposures, enabling modification of training or competition practices where potentially harmful conditions are identified.

Importantly, the value of these approaches may extend beyond improving EIB diagnosis alone. Respiratory symptoms in athletes are frequently multifactorial and often reflect the coexistence of several conditions affecting the upper and lower airways. Recent work has demonstrated that athletes rarely present with a single isolated respiratory abnormality, with asthma and EIB commonly occurring alongside rhinitis, allergy, breathing pattern dysfunction, laryngeal dysfunction and other airway disorders [20]. Future diagnostic pathways should therefore adopt a more holistic approach to athlete respiratory health, recognizing that optimal management requires identification and treatment of all relevant contributors to exercise-related respiratory symptoms.

Emerging evidence suggests that biomarkers, physiological assessments and wearable technologies could increasingly be embedded within the longitudinal management of airway

health in athletes. The challenge for future research will be to determine how these complementary measures can be integrated into practical clinical pathways and used to inform meaningful decisions regarding monitoring, treatment and environmental exposure. Prospective longitudinal studies will remain important to establish which athletes are most likely to benefit from closer surveillance, how changes in these measures should be interpreted and whether earlier intervention can alter the progression toward clinically significant airway dysfunction. Such an approach has the potential to move athlete respiratory care beyond episodic assessment toward more personalized monitoring and preservation of airway health throughout an athlete's career.

6. Expert opinion

The diagnosis of exercise-induced bronchoconstriction (EIB) has advanced considerably over the past two decades. The introduction of objective bronchoprovocation testing has improved diagnostic accuracy and reduced reliance on symptoms alone, providing clinicians with robust methods for confirming exercise-induced airway narrowing. However, in my opinion, the next phase of research should focus less on developing additional methods of confirming established bronchoconstriction and more on identifying athletes at risk of airway dysfunction before clinically significant disease develops. Rather than viewing EIB as an isolated event identified by a reduction in FEV₁, future approaches should increasingly consider airway health as a continuum that can be monitored throughout an athlete's career.

One area likely to become increasingly important is the integration of complementary assessments rather than the search for a single replacement for spirometry or bronchoprovocation testing. Bronchoprovocation tests will undoubtedly remain central to confirming EIB, but future diagnostic pathways may increasingly incorporate biomarkers of airway inflammation and injury, assessments of airway mechanics and continuous physiological monitoring to provide a broader understanding of airway health. Such an approach has the potential to improve disease phenotyping, guide management decisions and identify athletes who may benefit from closer respiratory surveillance before clinically significant airway dysfunction develops. Importantly, the challenge is unlikely to be determining which individual assessment performs best, but rather understanding how complementary technologies can be combined to provide clinically meaningful information that is not available from any single investigation.

A major challenge will be translating promising biomarkers from research laboratories into accessible clinical tools. Fractional exhaled nitric oxide (FeNO) provides an excellent example of how technological advances transformed a research biomarker into a practical point-of-care assessment that is now widely used in respiratory medicine. Similar progress may be possible for biomarkers of airway injury and non-type 2 inflammation. Club cell secretory protein (CC16), circulating eosinophils and inflammatory cytokines have all improved understanding of athlete airway pathology, yet their application remains largely confined to specialist centers and research studies. Developing rapid, reproducible and affordable methods of measuring these biomarkers within sporting environments could substantially improve our ability to characterize airway health. Equally important will be determining whether combinations of biomarkers provide greater clinical value than individual measurements alone and whether changes in these biomarkers can identify athletes at risk of developing airway dysfunction before objective bronchoconstriction becomes apparent.

In my opinion, advances in wearable technologies represent another exciting area for future research. Elite sport has already embraced continuous monitoring of training load, cardiovascular physiology and recovery. Extending this approach to respiratory health appears a logical progression. Wearable systems capable of monitoring breathing pattern, thoracoabdominal motion or other respiratory variables during training and competition may provide objective indicators of deteriorating airway health or impending EIB exacerbations before athletes become symptomatic. Integration with smartphone applications and cloud-based analytics could enable longitudinal respiratory surveillance, allowing clinicians to identify meaningful deviations from an athlete's normal respiratory profile and intervene earlier than is currently possible. Although considerable technological and validation challenges remain, developments in this area have the potential to make respiratory assessment more continuous, personalized and representative of an athlete's real-world environment than current laboratory-based evaluations.

An equally important consideration is whether greater emphasis should be placed on preventing airway injury rather

than simply detecting its consequences. The evidence reviewed in this article suggests that repeated exposure to cold dry air, chlorinated environments and airborne pollutants may contribute to progressive airway injury in susceptible athletes. Consequently, preserving respiratory health may require greater attention to reducing cumulative environmental exposure throughout an athlete's career. This could include routine consideration of ambient air quality during training and competition, particularly exposure to particulate matter (PM_{2.5}), ozone and nitrogen oxides (NO_x), with training schedules or locations modified where potentially harmful conditions are identified. Personal exposure monitoring combined with behavioral interventions has shown potential to reduce exposure to PM_{2.5} in people with asthma [61], providing a model that could be evaluated in athletic populations. This responsibility should extend beyond individual athletes and clinicians to sporting organizations, event organizers and governing bodies. Future initiatives could include consideration of air quality when selecting competition locations and scheduling events, improving air quality within sporting venues, reducing avoidable airborne irritants such as pyrotechnics and flares at sporting events, optimizing training schedules during periods of poor air quality and continuing to develop lightweight heat- and moisture-exchange devices that athletes can comfortably wear during training in challenging environments. Such preventative strategies have the potential to complement advances in diagnosis by reducing repeated airway injury before clinically significant EIB develops.

The successful implementation of these advances will depend upon evidence that earlier identification of airway dysfunction results in meaningful improvements in athlete health. Demonstrating that biomarkers, oscillometry or wearable monitoring can detect physiological abnormalities is only the first step. Future research must establish how these measurements should be interpreted, determine appropriate thresholds for clinical intervention and demonstrate that earlier identification of airway dysfunction leads to improved respiratory outcomes. Addressing these questions will require prospective longitudinal studies that follow athletes throughout their sporting careers rather than cross-sectional studies of established disease. Such studies will also be essential for understanding the natural history of athlete airway dysfunction and identifying those individuals most likely to benefit from preventative strategies.

Looking ahead, I anticipate that respiratory assessment in athletes will become increasingly holistic, combining objective bronchoprovocation testing with complementary assessments of airway inflammation, airway injury, respiratory mechanics and breathing pattern. Rather than replacing current diagnostic approaches, these technologies are likely to enhance them, providing a more comprehensive understanding of airway health and supporting more personalized clinical decision-making. If this can be achieved, the future of EIB management may shift from diagnosing established disease toward preserving respiratory health through earlier identification of airway dysfunction, targeted preventative strategies and interventions designed to minimize the cumulative effects of high ventilatory exercise throughout an athlete's career.

Funding

This paper was not funded.

Declaration of interests

J. Dickinson has received part-funding from POWERbreathe for a PhD scholarship investigating inspiratory muscle training in breathing pattern disorders. J. Dickinson was an unpaid scientific advisor to Bedfont Scientific Ltd, a manufacturer of fractional exhaled nitric oxide measurement devices, between 2017 and 2021, and received payment for specific device-related review work during this period. J. Dickinson provides consultancy services for athletes with respiratory problems through the University of Kent and Marylebone Health Group. J. Dickinson is an unpaid scientific advisor to Alveos Oy, developer of wearable respiratory monitoring technology. J. Dickinson is a co-investigator on the EPSRC-funded project UKRI3606: AIRTIME: AI-Enabled Mobile First Screening for Early Respiratory Disease Detection. The authors have no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

Reviewer disclosures

Peer reviewers on this manuscript have no relevant financial or other relationships to disclose.

Acknowledgments

During the preparation of this manuscript, the author used ChatGPT (OpenAI, GPT-5.6 Sol) to assist with idea generation and exploration, language refinement, and the development of the illustrative figures. The figures were generated using ChatGPT's image-generation capability and were iteratively reviewed and revised by the author to ensure scientific accuracy and clarity of presentation. All scientific content, interpretation and final editorial decisions were made by the author, who accepts full responsibility for the content and accuracy of the manuscript and figures.

Author contributions

CRedit: **John W. Dickinson:** Conceptualization, Project administration, Visualization, Writing – original draft, Writing – review & editing.

References

- Parsons JP, Teal, Hallstrand S, et al. Online supplement an official American Thoracic Society clinical practice guideline: exercise-induced bronchoconstriction. *Am J Respir Crit Care Med*. 2013;187(9):1016–27. doi: [10.1164/rccm.201303-0437ST](#)
- Couto M, Kurowski M, Moreira A, et al. Mechanisms of exercise-induced bronchoconstriction in athletes: current perspectives and future challenges. *Allergy: Eur J Allergy Clin Immunol*. 2018;73(1):8–16. doi: [10.1111/all.13224](#)
- Dickinson JW, Whyte GP, McConnell AK, et al. Impact of changes in the IOC-MC asthma criteria: a British perspective. *Thorax*. 2005;60(8):629–632.
- Dickinson J, McConnell A, Whyte G. Diagnosis of exercise-induced bronchoconstriction: eucapnic voluntary hyperpnoea challenges identify previously undiagnosed elite athletes with exercise-induced bronchoconstriction. *Br J Sports Med*. 2011;45(14):1126–1131. doi: [10.1136/bjsm.2010.072520](#)
- Jackson A, Allen H, Hull JH, et al. Diagnosing exercise-induced bronchoconstriction: over- or under-detection? *Allergy: Eur J Allergy Clin Immunol*. 2020;75(2):460–463. doi: [10.1111/all.14005](#)
- Price OJ, Sewry N, Schwellnus M, et al. Prevalence of lower airway dysfunction in athletes: a systematic review and meta-analysis by a subgroup of the IOC consensus group on acute respiratory illness in the athlete. *Br J Sports Med*. 2022;56(4):213–222. doi: [10.1136/bjsports-2021-104601](#)
- Levai IK, Hull JH, Loosemore M, et al. Environmental influence on the prevalence and pattern of airway dysfunction in elite athletes. *Respirology*. 2016;21(8):1391–1396. doi: [10.1111/resp.12859](#)
- Pohjantähti H, Laitinen J, Parkkari J. Exercise-induced bronchospasm among healthy elite cross country skiers and non-athletic students. *Scand J Med Sci Sports*. 2005;15(5):324–328. doi: [10.1111/j.1600-0838.2004.00423.x](#)
- Mäki-Heikkilä R, Karjalainen J, Parkkari J, et al. Asthma in competitive cross-country skiers: a systematic review and meta-analysis. *Sports Med*. 2020;50(11):1963–1981. doi: [10.1007/s40279-020-01334-4](#)
- Sue-Chu M, Larsson L, Björner L. Prevalence of asthma in young cross-country skiers in central Scandinavia: differences between Norway and Sweden. *Respir Med*. 1996;90(2):99–105. doi: [10.1016/S0954-6111\(96\)90206-1](#)
- Bougault V, Boulet LP. Airways disorders and the swimming pool. *Immunol Allergy Clin N Am*. 2013;33(3):395–408. doi: [10.1016/j.jac.2013.02.008](#)
- Nemery B, Hoet PHM, Nowak D. Indoor swimming pools, water chlorination and respiratory health. *Eur Respir J*. 2002;19(5):790–793. doi: [10.1183/09031936.02.00308602](#)
- Weiss P, Rundell KW. Imitators of exercise-induced bronchoconstriction. *All Asth Clin Immun*. 2009;5(1). doi: [10.1186/1710-1492-5-7](#)
- Rundell KW, Im J, Mayers LB, et al. Self-reported symptoms and exercise-induced asthma in the elite athlete [Internet]. *Med Sci Sports Exerc*. 2001;33(2):208–213.
- Ansley L, Kippelen P, Dickinson J, et al. Misdiagnosis of exercise-induced bronchoconstriction in professional soccer players. *Allergy: Eur J Allergy Clin Immunol*. 2012;67(3):390–395. doi: [10.1111/j.1398-9995.2011.02762.x](#)
- Dickinson JW, Whyte GP, McConnell AK, et al. Screening elite winter athletes for exercise induced asthma: a comparison of three challenge methods. *Br J Sports Med*. 2006;40(2):179–182. doi: [10.1136/bjsm.2005.022764](#)
- Reier-Nilsen T, Sewry N, Chenuel B, et al. Diagnostic approach to lower airway dysfunction in athletes: a systematic review and meta-analysis by a subgroup of the IOC consensus on 'acute respiratory illness in the athlete. *Br J Sports Med*. 2023;57(8):481–489. doi: [10.1136/bjsports-2022-106059](#)
- Hull JH, Burns P, Carre J, et al. BTS clinical statement for the assessment and management of respiratory problems in athletic individuals. *Thorax*. 2022;77(6):540–551. doi: [10.1136/thoraxjnl-2021-217904](#)
- Weiler JM, Brannan JD, Randolph CC, et al. Exercise-induced bronchoconstriction update—2016. *J Allergy Clin Immunol*. 2016;138(5):1292–1295.e36. doi: [10.1016/j.jaci.2016.05.029](#)
- Hull JH, Jackson AR, Ranson C, et al. The benefits of a systematic assessment of respiratory health in illness-susceptible athletes. *Eur Respir J*. 2021;57(6):2003722. doi: [10.1183/13993003.03722-2020](#)
- Couto M, Stang J, Horta L, et al. Two distinct phenotypes of asthma in elite athletes identified by latent class analysis. *J Asthma*. 2015;52(9):897–904. doi: [10.3109/02770903.2015.1067321](#)
- Rasmussen SM, Hansen ESH, Backer V. Asthma in elite athletes – do they have type 2 or non-type 2 disease? A new insight on the endotypes among elite athletes. *Front Allergy*. 2022;3. doi: [10.3389/falgy.2022.973004](#)
- Dickinson J, Gowers W, Sturridge S, et al. Fractional exhaled nitric oxide in the assessment of exercise-induced bronchoconstriction: a multicenter retrospective analysis of UK-based athletes. *Scand J Med Sci Sports*. 2023;33(7):1221–1230. doi: [10.1111/sms.14367](#)
- Karjalainen E-M, Laitinen A, Sue-Chu M, et al. Evidence of airway inflammation and remodeling in ski athletes with and without bronchial hyperresponsiveness to methacholine. *Am J Respir Crit Care Med [Internet]*. 2000;161(6):2086–2091. doi: [10.1164/ajrccm.161.6.9907025](#)

25. Bougault V, Boulet LP. Airway dysfunction in swimmers. *Br J Sports Med.* 2012;46(6):402–406. doi: [10.1136/bjsports-2011-090821](https://doi.org/10.1136/bjsports-2011-090821)
26. Kippelen P, Anderson SD. Pathogenesis of exercise-induced bronchoconstriction. *Immunol Allergy Clin N Am.* 2013;33(3):299–312. doi: [10.1016/j.iac.2013.02.002](https://doi.org/10.1016/j.iac.2013.02.002)
27. Kippelen P, Fitch KD, Anderson SD, et al. Respiratory health of elite athletes – preventing airway injury: a critical review. *Br J Sports Med.* 2012;46(7):471–476. doi: [10.1136/bjsports-2012-091056](https://doi.org/10.1136/bjsports-2012-091056)
28. Rasmussen SM, Halvard Hansen ES, Stensrud T, et al. Asthma endotypes in elite athletes: a cross-sectional study of European athletes participating in the Olympic games. *Allergy.* 2022;77(7):2250–2253. doi: [10.1111/all.15313](https://doi.org/10.1111/all.15313)
29. Kharitonov SA, Barnes PJ. Exhaled Biomarkers. *Chest.* 2006;130(5):1541–1546. doi: [10.1378/chest.130.5.1541](https://doi.org/10.1378/chest.130.5.1541)
30. Grzelewski T, Grzelewska A, Majak P, et al. Fractional exhaled nitric oxide (FeNO) may predict exercise-induced bronchoconstriction (EIB) in schoolchildren with atopic asthma. *Nitric Oxide.* 2012;27(2):82–87. doi: [10.1016/j.niox.2012.05.002](https://doi.org/10.1016/j.niox.2012.05.002)
31. Schoos A-M, Christiansen CF, Stokholm J, et al. FeNO and exercise testing in children at risk of asthma. *J Allergy Clin Immunol Pract.* 2018;6(3):855–862.e2.
32. Jackson AR, Hull JH, Hopker JG, et al. Impact of detecting and treating exercise-induced bronchoconstriction in elite footballers. *ERJ Open Res.* 2018;4(2):00122–2017. doi: [10.1183/23120541.00122-2017](https://doi.org/10.1183/23120541.00122-2017)
33. Rouhos A, Ekroos H, Karjalainen S, et al. Exhaled nitric oxide and exercise-induced bronchoconstriction in young male conscripts: association only in atopics. *Allergy: Eur J Allergy Clin Immunol.* 2005;60(12):1493–1498. doi: [10.1111/j.1398-9995.2005.00901.x](https://doi.org/10.1111/j.1398-9995.2005.00901.x)
34. Atchley TJ, Smith DM. Exercise-induced bronchoconstriction in elite or endurance athletes: pathogenesis and diagnostic considerations. *Ann Allergy Allergy Asthma Immunol.* 2020;125(1):47–54. doi: [10.1016/j.anai.2020.01.023](https://doi.org/10.1016/j.anai.2020.01.023)
35. Kippelen P, Anderson SD. Airway injury during high-level exercise. *Br J Sports Med.* 2012;46(6):385–390. doi: [10.1136/bjsports-2011-090819](https://doi.org/10.1136/bjsports-2011-090819)
36. Bougault V, Turmel J, Boulet LP. Airway hyperresponsiveness in elite swimmers: is it a transient phenomenon? *J Allergy Clin Immunol.* 2011;127(4):892–898. doi: [10.1016/j.jaci.2010.11.003](https://doi.org/10.1016/j.jaci.2010.11.003)
37. Couto M, Bernard A, Delgado L, et al. Health effects of exposure to chlorination by-products in swimming pools. *Allergy.* 2021;76(11):3257–3275. doi: [10.1111/all.15014](https://doi.org/10.1111/all.15014)
38. Bolger C, Tufvesson E, Sue-Chu M, et al. Hyperpnea-induced bronchoconstriction and urinary CC16 levels in athletes. *Med Sci Sports Exerc.* 2011;43(7):1207–1213. doi: [10.1249/MSS.0b013e31820750d8](https://doi.org/10.1249/MSS.0b013e31820750d8)
39. Chimenti L, Morici G, Paternò A, et al. Bronchial epithelial damage after a half-marathon in nonasthmatic amateur runners. *Am J Physiol Lung Cellular Mol Physiol.* 2010;298(6):L857–L862. doi: [10.1152/ajplung.00053.2010](https://doi.org/10.1152/ajplung.00053.2010)
40. Price OJ, Ansley L, Menzies-Gow A, et al. Airway dysfunction in elite athletes – an occupational lung disease? *Allergy.* 2013;68(11):1343–1352. doi: [10.1111/all.12265](https://doi.org/10.1111/all.12265)
41. Morissette MC, Murray N, Turmel J, et al. Increased exhaled breath condensate 8-isoprostane after a swimming session in competitive swimmers. *Eur J Sport Sci.* 2016;16(5):569–576. doi: [10.1080/17461391.2015.1063702](https://doi.org/10.1080/17461391.2015.1063702)
42. Djukanović R, Sterk PJ, Fahy JV, et al. Standardised methodology of sputum induction and processing. *Eur Respir J.* 2002;20(37 suppl):1s–2s. doi: [10.1183/09031936.02.00000102](https://doi.org/10.1183/09031936.02.00000102)
43. Horváth I, Hunt J, Barnes PJ. Exhaled breath condensate: methodological recommendations and unresolved questions. *Eur Respir J.* 2005;26(3):523–548. doi: [10.1183/09031936.05.00029705](https://doi.org/10.1183/09031936.05.00029705)
44. Johnson B, Steenbruggen I, Graham BL, et al. Improving spirometry testing by understanding patient preferences. *ERJ Open Res.* 2021;7(1):00712–02020. doi: [10.1183/23120541.00712-2020](https://doi.org/10.1183/23120541.00712-2020)
45. Cooper BG. Limitations to spirometry being performed in ‘the office’. *Chron Respir Dis.* 2005;2(2):113–115. doi: [10.1191/1479972305cd072xx](https://doi.org/10.1191/1479972305cd072xx)
46. King GG, Bates J, Berger KI, et al. Technical standards for respiratory oscillometry. *Eur Respir J.* 2020;55(2):1900753. doi: [10.1183/13993003.00753-2019](https://doi.org/10.1183/13993003.00753-2019)
47. Anderson SD, Kippelen P. Airway injury as a mechanism for exercise-induced bronchoconstriction in elite athletes. *J Allergy Clin Immunol.* 2008;122(2):225–235. doi: [10.1016/j.jaci.2008.05.001](https://doi.org/10.1016/j.jaci.2008.05.001)
48. Rundell KW, Evans TM, Baumann JM, et al. Lung function measured by impulse oscillometry and spirometry following eucapnic voluntary hyperventilation. *Can Respir J.* 2005;12(5):257–263. doi: [10.1155/2005/297472](https://doi.org/10.1155/2005/297472)
49. Price OJ, Ansley L, Bikov A, et al. The role of impulse oscillometry in detecting airway dysfunction in athletes. *J Asthma.* 2016;53(1):62–68. doi: [10.3109/02770903.2015.1063647](https://doi.org/10.3109/02770903.2015.1063647)
50. Sperlich B, Aminian K, Düking P, et al. Editorial: wearable sensor technology for monitoring training load and health in the athletic population. *Front Physiol.* 2020;10:10. doi: [10.3389/fphys.2019.01520](https://doi.org/10.3389/fphys.2019.01520)
51. Sperlich B, Holmberg H-C. The responses of elite athletes to exercise: an all-day, 24-h integrative view is required! *Front Physiol.* 2017;8:564. doi: [10.3389/fphys.2017.00564](https://doi.org/10.3389/fphys.2017.00564)
52. Pi-Rusñol R, Calvo M, Grazioli G, et al. Incremental diagnostic value of 24-hour holter monitoring during training in elite athlete pre-participation screening. *BMJ Open Sport Exerc Med.* 2026;12(2):e003258. doi: [10.1136/bmjsem-2026-003258](https://doi.org/10.1136/bmjsem-2026-003258)
53. Dickinson JW, Smyth CME, Winter SL. Breathing pattern changes in response to bronchoconstriction in physically active adults. *J Asthma [Internet].* 2025;62(1):14–23. doi: [10.1080/02770903.2024.2383632](https://doi.org/10.1080/02770903.2024.2383632)
54. Aliverti A, Stevenson N, Dellacà RL, et al. Regional chest wall volumes during exercise in chronic obstructive pulmonary disease. *Thorax.* 2004;59(3):210–216.
55. Aliverti A, Rodger K, Dellacà RL, et al. Effect of salbutamol on lung function and chest wall volumes at rest and during exercise in COPD. *Thorax.* 2005;60(11):916–924.
56. Aliverti A, Pedotti A. Opto-electronic plethysmography. *Monaldi Arch Chest Dis.* 2003;59(1):12–6.
57. Adhikari NG, Hunsicker E, Pain MTG, et al. Agreement between a pre-marked T-shirt and manual marker placement for opto-electronic plethysmography (OEP) measures. *Sensors [Internet].* 2025;25(14):4464. doi: [10.3390/s25144464](https://doi.org/10.3390/s25144464)
58. Massaroni C, Nicolò A, Lo Presti D, et al. Contact-based methods for measuring respiratory rate. *Sensors.* 2019;19(4):908. doi: [10.3390/s19040908](https://doi.org/10.3390/s19040908)
59. Massaroni C, Saccomandi P, Schena E. Medical smart textiles based on fiber optic technology: an overview. *J Funct Biomater.* 2015;6(2):204–221. doi: [10.3390/jfb6020204](https://doi.org/10.3390/jfb6020204)
60. Tavares C, Leitão C, Lo Presti D, et al. Respiratory and heart rate monitoring using an FBG 3D-printed wearable system. *Biomed Opt Express [Internet].* 2022;13(4):2299. doi: [10.1364/BOE.452115](https://doi.org/10.1364/BOE.452115)
61. McCarron A, Semple S, Swanson V, et al. Piloting co-developed behaviour change interventions to reduce exposure to air pollution and improve self-reported asthma-related health. *J Expo Sci Environ Epidemiol.* 2025;35(2):242–253. doi: [10.1038/s41370-024-00661-2](https://doi.org/10.1038/s41370-024-00661-2)