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Exercise-Induced Bronchoconstriction (EIB) in Athletes — Diagnosis, Pathophysiology, Prevention, and Implications for Training

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Abstract

Introduction. Competitive sports training places unprecedented demands on the respiratory system. Targeted pharmacotherapy and modifications to environmental conditions have proven to be highly effective tools for supporting respiratory reserve, leading to the protection of lung structure and an improvement in maximal oxygen uptake (VO2max). Nevertheless, serious concerns remain regarding common diagnostic errors and the risk of violating strict anti-doping regulations (WADA).

Research objective. This literature review synthesizes current knowledge on the pathophysiology, objective diagnosis, differential diagnosis, screening, treatment, and training implications of exercise-induced bronchoconstriction (EIB) in elite athletes, taking into account current anti-doping regulations.

Methodology. The review is based on a structured search of the PubMed database through May 2026. Keywords included: “exercise-induced bronchoconstriction”, “elite athletes”, “asthma in sports”, “exercise-induced laryngeal obstruction”, “airway remodeling”, and “WADA regulations”. Priority was given to clinical trials, systematic reviews, large observational studies, and international consensus documents regarding the management of respiratory system dysfunctions in sports. Articles were analyzed for their relevance to describing pathophysiological mechanisms, objective diagnostic algorithms, differential diagnosis, and pharmacotherapy compliant with anti-doping regulations.

Conclusions. Objective diagnostics and targeted anti-inflammatory treatment represent significant progress in the care of elite athletes with EIB, ensuring the preservation of aerobic capacity and lung health; however, their implementation requires careful monitoring of drug dosage limits and avoidance of tachyphylaxis. The use of provocation tests is essential to optimize therapy, rule out other upper respiratory tract pathologies, and ensure safe, level competition.

Keywords:exercise-induced bronchoconstriction, elite athletes, anti-doping regulations, differential diagnosis, sports medicine

Content

1. Introduction

Competitive sports training, particularly in endurance disciplines, places extreme physiological demands on an athlete's respiratory system. During maximal exertion, minute ventilation in professional athletes can increase up to twentyfold compared to resting values. This permanent hyperventilation - often developing under demanding environmental and microclimatic conditions - predisposes athletes to specific respiratory tract pathologies, among which the most common clinical entity is exercise-induced bronchoconstriction (EIB) [1, 2, 3, 4].

In the past, EIB was viewed solely as a secondary symptom of classic bronchial asthma. Modern sports medicine recognizes it as a distinct, independent clinical phenotype. It is important that, EIB can develop in elite athletes who have no history of atopy or chronic asthma [4, 5]. In certain high-risk disciplines, such as winter sports, competitive swimming, or long-

distance running, the prevalence of EIB reaches striking levels, affecting from 30% to 50% of athletes [5, 6]. Despite the scale of the problem, EIB remains a commonly undiagnosed condition in daily sports medicine practice. On the other hand, empirical diagnoses based solely on nonspecific subjective symptoms are highly prone to error, leading to unjustified polypharmacy and the use of chronic anti-asthmatic medications in completely healthy individuals [7, 8].

Untreated or incorrectly controlled EIB constitutes a direct, mechanical barrier to exercise capacity, significantly lowering the oxygen threshold (VO2max or VO2peak) and disrupting the continuity of the training process [9, 10, 11]. Over the course of a long-term athletic career, repeated microtraumas to the bronchial mucosa initiate chronic inflammation and irreversible airway remodeling [5, 12]. This review aims to provide a comprehensive synthesis of current knowledge on the pathophysiology of EIB, evaluate modern diagnostic algorithms based on objective provocation tests, establish criteria for differentiating EIB from upper airway pathologies (such as exercise-induced laryngeal obstruction—EILO), and a detailed discussion of therapeutic strategies that are fully compliant with the daily dose limits specified by the World Anti-Doping Agency (WADA) [5, 13, 14].

2. Pathophysiology of Exercise-Induced Bronchoconstriction (EIB)

EIB in athletes is defined as a transient, reversible narrowing of the airway lumen, resulting directly from intense pulmonary ventilation during physical exertion. Unlike classic allergic asthma, the pathophysiological mechanism of EIB in competitive athletes is closely linked to extreme parameters of the airway microclimate, and the key elements of the pathophysiological cascade are: mechanical and thermal damage to epithelial cells, a secondary inflammatory response, and disturbances in vascular autoregulation in the bronchial wall [1, 2, 3, 4].

The pathological process is initiated by a state of hyperventilation, characterized by a high minute ventilation volume. Under physiological conditions, air is warmed and humidified in the upper airways. During extreme exertion (often in a cold, dry, or polluted environment), these mechanisms become inadequate, leading to direct contact between unprepared air and the lower airways. The consequence of this is rapid dehydration and cooling of the respiratory epithelial surface [1, 15, 16, 17].

Repeated osmotic and thermal stimuli generate microdamage to the epithelial integrity, resulting in the release of damage-associated molecular patterns (DAMPs) and epithelial

cytokines (so-called alarmines). This leads to rapid degranulation of mast cells and the activation of eosinophils and neutrophils [3, 4]. Studies in young and elite athletes with EIB have confirmed significantly elevated levels of inflammatory markers in induced sputum or serum, including interleukins 5 (IL-5) and 8 (IL-8) - responsible for the chemotaxis and activation of granulocytes - as well as tumor necrosis factor (TNF-α), Clara cell protein (CC16, a direct marker of epithelial barrier damage), and immunoglobulin E (IgE) [2, 6, 18].

Inflammatory mediators released from mast cells and eosinophils with potent bronchoconstrictive effects - primarily histamine, cysteinyl leukotrienes (CysLTs), and prostaglandin D2 (PGD2) - directly act on bronchial smooth muscle receptors, altering the tone of their walls and inducing a sudden contraction [1, 4, 17, 19]. In the long term, permanent exposure to these mediators and repeated microtraumas initiate the process of airway remodeling. This manifests as chronic bronchial hyperreactivity, thickening and fibrosis of the extracellular matrix of the bronchial wall, and hypertrophy of the muscular component [1, 5, 20], which may permanently limit an athlete's ventilatory reserve.

Modern sports medicine explains the mechanism of EIB by integrating two classical concepts: the osmotic theory and the thermovascular theory. In elite athletes, these hypotheses are not mutually exclusive but demonstrate profound pathophysiological synergy.

According to the osmotic theory, the primary turning point is massive water loss from the airway surface liquid (ASL) due to evaporation during intense breathing. This leads to a rapid increase in ASL osmolarity. The resulting osmotic gradient forces water out of the interior of epithelial cells and mast cells, causing their mechanical contraction. This process activates mechanosensitive ion channels, which initiates a biochemical cascade and results in the aforementioned massive release of histamine and leukotrienes, causing smooth muscle contraction and the overproduction of thick mucus [1, 3, 15, 17].

In contrast, the thermovascular theory focuses on the thermodynamic aspects of breathing. Inhaling cool air causes a drastic drop in the temperature of the bronchial walls, which induces a defensive vasoconstriction of the mucosal blood vessels to limit heat loss. The key pathological moment, however, occurs immediately after the end of physical exertion, when ventilation drops sharply. A process of rapid, reactive "rewarming" of the airways then takes place, which triggers secondary, sudden hyperemia and vasodilation. The consequence of this phenomenon is massive exudation and swelling of the bronchial wall, which mechanically narrows their lumen from the inside, independent of the activity of the smooth muscles themselves [1, 15, 16].

In the population of endurance athletes, extreme hyperventilation generates such profound disturbances in homeostasis that both of these mechanisms interfere with each other simultaneously: dehydration induces the release of mediators, and sudden temperature changes exacerbate vascular edema [1, 15, 16, 17].

Additionally, so-called priming stimuli - that is, specific environmental factors (such as chlorine and chloramine compounds in swimming pools, high concentrations of PM2.5 and PM10 particulate matter in municipal stadiums, or ice aerosols on ski slopes) - act as adjuvants. They exacerbate the initial damage to the epithelial barrier, significantly lowering the bronchial reactivity threshold to osmotic and thermal stimuli during each subsequent training cycle [2, 3, 6, 18].

3. Diagnosis of Exercise-Induced Bronchoconstriction (EIB) in Elite Athletes

A diagnosis of EIB cannot be established solely on the basis of a clinical history or subjective symptoms reported by the athlete. Due to the highly nonspecific nature of exercise-induced dyspnea, clinical symptoms correlate poorly with objective functional measurements. Therefore, confirmation of EIB requires objective documentation of a post-exercise or post-provocation decrease in forced expiratory volume in one second (FEV1) [7, 13, 21, 22].

The diagnostic process begins with a detailed medical history and physical examination. Although these are essential for ruling out alarm symptoms (e.g., heart disease) and alternative causes of dyspnea, the clinical history alone has low predictive value in diagnosing EIB. A baseline resting spirometry test should be performed in every patient with suspected EIB. A normal resting spirometry result does not rule out EIB in elite athletes, as their baseline lung function parameters are often significantly higher than those typical for the general population. However, if the baseline examination reveals airway obstruction that shows significant reversibility after administration of a short-acting beta-2 agonist (SABA), this suggests a diagnosis of coexisting asthma with concomitant EIB [7, 13, 21].

A standardized exercise test is the primary physiological method for diagnosing EIB, particularly in children and adolescents. The protocol requires the use of a treadmill or cycle ergometer and is designed to rapidly induce high minute ventilation (reaching the target heart rate within the first 2–3 minutes), followed by maintaining intense exercise for the next 6 minutes. Spirometry is performed before the test and at regular intervals after exercise (typically at 5, 10, 15, and 30 minutes). A decrease in FEV1 of ≥10% from baseline in any of the post-exercise measurements confirms EIB [13, 21].

For elite athletes, the laboratory ECT test often does not reflect the climatic realities of their sport. To maximize the test's sensitivity, tests should, whenever possible, mimic the specific environmental conditions of the given discipline (e.g., low ambient temperature, low air humidity, extreme ventilatory demand) [23, 24].

In situations where a standardized exercise test is unavailable, inconclusive, or logistically difficult to perform, indirect provocation tests that mimic osmotic stress on the airways are used, these include:

- Eucapnic hyperventilation (EVH): Widely used in sports medicine and officially recommended by the International Olympic Committee (IOC). EVH requires the athlete to hyperventilate a dry gas mixture containing approximately 5% CO2 (to prevent hypocapnia) at 85% of maximum voluntary ventilation (MVV) for 6 minutes. Although EVH is characterized by a high negative predictive value, more recent data indicate that it is not an absolute gold standard; it exhibits significant intra-individual variability and may yield false-negative results in athletes with mild or seasonal forms of EIB [25, 26, 27, 28].
- Inhalation provocation test with mannitol: An indirect osmotic test involving the inhalation of dry mannitol powder in increasing doses. It serves as an easily accessible laboratory alternative to EVH or ECT, faithfully replicating the hyperosmolarity mechanism leading to mast cell degranulation [13, 21, 23].
- Field tests combined with portable spirometry: Field tests involve performing discipline-specific training combined with spirometric measurements before and after exercise. The use of validated portable spirometers integrated with smartphones enables the conduct of serial tests under real-world training conditions, which significantly increases diagnostic sensitivity [24, 29].

A key goal in the care of elite athletes is to distinguish true EIB from other conditions that present with exercise-induced dyspnea, thereby preventing the unnecessary and inappropriate initiation of anti-asthmatic medication.

Exercise-induced laryngeal obstruction (EILO), historically (and somewhat imprecisely) referred to as vocal cord dysfunction (VCD), is the primary cause of upper airway-related dyspnea in young and elite athletes. It very often coexists with EIB [22, 30, 31, 32]. EILO is characterized by inspiratory stridor and a distinct sensation of tightness or blockage in the throat, which increases and peaks during maximal exertion, then resolves very quickly (within 1–5 minutes) after cessation of exertion. In contrast, EIB typically develops only after the end of exercise and manifests as expiratory wheezing [31, 33, 34]. The gold standard is

continuous laryngoscopy during exercise (CLE test), which allows direct visualization of the paradoxical inspiratory collapse of the glottic or supraglottic structures during a maximal exercise protocol [22, 31, 32, 33]. Alternatively, the EVH test combined with simultaneous nasofibroscopy allows visualization of pathological adduction of the vocal folds under high ventilatory load [35]. Short, validated questionnaires (e.g., the Asthma Control Test—ACT or the Dyspnea Index—DI) can serve as clinical triage tools to help identify candidates for CLE testing in centers with limited access to exercise laryngoscopy [36].

It is essential to rigorously distinguish isolated EIB (bronchospasm occurring exclusively after exercise, without daytime or nighttime symptoms) from classic asthma with EIB (where exercise is only one of many triggers, and the patient reports diurnal PEF variability, atopy, or allergic rhinitis) [7]. Misdiagnosis of asthma based solely on subjective symptoms is extremely common in primary care; up to 50% of patients diagnosed with EIB or asthma in primary care show completely negative results in objective EVH or ECT provocation tests [8].

Cardiac anomalies must be aggressively ruled out, particularly when exertional dyspnea is accompanied by chest pain, palpitations, syncope, or a positive family history of sudden cardiac death. Such presentations require immediate ECG, echocardiography, and urgent cardiology consultation [13, 21].

Normal physiological limitation, manifests as severe exertional dyspnea, but without an objective post-exercise decrease in FEV1. In such cases, a cardiopulmonary exercise test (CPET) shows premature reaching of the anaerobic threshold without signs of mechanical limitation of ventilatory flow. The management strategy here is progressive physical training, not asthma pharmacotherapy [13].

4. Prevention and Treatment of Exercise-Induced Bronchoconstriction (EIB) in Elite Athletes

Effective treatment of EIB in professional athletes requires an integrated approach that combines rigorous modification of the training environment, non-pharmacological protective strategies, and carefully selected drug therapy. The primary therapeutic goal is full symptom control and preservation of the athlete's ventilatory reserve, while strictly adhering to current anti-doping regulations of the World Anti-Doping Agency (WADA) [7, 13, 14].

The basis of non-pharmacological prevention is to minimize exposure to environmental triggers of bronchospasm. In clinical and training practice, it is recommended to avoid exposure

to cold and dry air, air pollution (including PM10 and PM2.5 particulate matter), and high concentrations of inhaled allergens. For athletes training in indoor facilities, it is crucial to avoid swimming pools with inadequate ventilation, where toxic chloramines accumulate, as well as sports halls with high dust levels [13, 14, 20, 37]. Practical recommendations for athletes include:

- Scheduling outdoor training sessions outside of peak traffic hours and during periods of lower smog levels.
- Strictly avoiding exposure to tobacco smoke.
- Using special masks with a heat and moisture exchanger (HME) in cold conditions, which effectively warm and humidify inhaled air, though they may reduce breathing comfort during maximum exertion.
- Implementation of dietary interventions with potential supportive effects, such as supplementation with omega-3 fatty acids, vitamin D, and vitamin C, as well as limiting sodium intake in the daily diet [5, 13, 38].

From the perspective of exercise physiology, a key non-pharmacological element is the protective warm-up. A properly planned protocol, based on exercises of varying intensity or short intervals, intentionally induces a so-called refractory period in the athlete. This is a phenomenon in which, following an initial, controlled micro-constriction of the bronchi, there is a temporary depletion of inflammatory mediators in mast cells. As a result, subsequent, actual physical exertion triggers a significantly weaker bronchoconstrictive response. This protective effect persists in the athlete's body for approximately two hours [5, 13, 20].

Importantly, long-term, well-structured aerobic training under medical supervision improves overall cardiovascular and respiratory fitness (by increasing VO2max) without negatively affecting the severity of EIB [13, 37].

Pharmacological management of EIB is divided into acute prevention of attacks immediately before exercise and chronic treatment to control airway inflammation. The use of SABA medications (e.g., salbutamol) 10–15 minutes before planned physical activity constitutes the first line of pharmacological prophylaxis in athletes with isolated EIB and in patients with comorbid asthma. These medications provide effective protection against bronchoconstriction for several hours after inhalation.

However, athletes must be categorically warned against the serious clinical error of regular, daily SABA monotherapy. Frequent and habitual use of these medications leads to the rapid development of tachyphylaxis (receptor tolerance). This results in a drastic decrease in the drug's protective efficacy, a shortening of its duration of action, and the risk of a "rebound"

phenomenon—that is, a paradoxical exacerbation of bronchial hyperreactivity after the SABA's effect wears off [7, 13, 22].

In athletes in whom EIB recurs regularly, has a severe course, or coexists with classic asthma, the cornerstone of therapy is the chronic use of inhaled glucocorticosteroids (ICS). ICS medications effectively suppress the underlying inflammatory process, regenerate the epithelial barrier, and consequently reduce the frequency and severity of EIB attacks [1, 7, 13]. Studies in long-distance runners have shown that 30 days of regular combination therapy with ICS and a long-acting beta-2 agonist (LABA) not only significantly reduced the post-exercise decline in FEV1 but also substantially improved the athletes' oxygen uptake (VO2peak) and aerobic capacity [9].

Leukotriene receptor antagonists (LTRA, e.g., montelukast) are successfully used as an alternative or as an add-on therapy to ICS; they demonstrate high efficacy in forms of EIB with a strong leukotriene component [7, 38]. Mast cell stabilizers (cromones) are a pre-exercise option, but they are used less frequently in modern sports medicine [7, 38].

Current guidelines increasingly highlight the efficacy of modern regimens, such as the use of a combined ICS and formoterol formulation on an "as-needed" basis before exercise or as part of MART (modulated and maintenance) therapy. This allows for simultaneous bronchodilation and administration of an anti-inflammatory dose, minimizing the risk of tachyphylaxis, although it requires rigorous control of daily doses in competitive sports [13, 38].

Managing pharmacotherapy in professional athletes requires the physician to have a thorough understanding of the current WADA Prohibited List. Beta-2 agonists are prohibited by default unless administered via inhalation at strictly defined, maximum therapeutic doses that do not exhibit systemic (anabolic) effects. According to WADA regulations, the permitted (not requiring a Therapeutic Use Exemption – TUE) dose limits for the most common inhaled medications are:

- Salbutamol: A maximum of 1,600 micrograms over a 24-hour period, provided that this dose does not exceed 600 micrograms within any 8-hour period, calculated from the administration of any dose.
- Formoterol: The maximum dose administered must not exceed 54 micrograms over a 24-hour period.
- Salmeterol: A maximum of 200 micrograms within 24 hours.

Exceeding the above daily doses or using other beta-2 agonists via inhalation or systemically (e.g., orally) is considered a violation of anti-doping regulations, unless the athlete

has an approved TUE application based on complete medical documentation. Good clinical practices in the care of elite athletes include strictly basing the diagnosis of EIB on objective functional tests (ECT, EVH), meticulous documentation of the medical history, precise planning of dosing schedules around training and competition cycles, and avoiding chronic monotherapy with SABA medications [5, 7, 14]. Properly and legally administered treatment allows athletes to complete their full training regimens and achieve success on the international stage without compromising their health.

5. Exercise Consequences and Training Implications of Exercise-Induced Bronchoconstriction (EIB)

The presence of EIB in elite athletes has serious consequences that go beyond a mere temporary episode of shortness of breath. Untreated or improperly managed EIB directly limits the body's adaptive potential, impairs the quality of training sessions, and affects the athlete's long-term respiratory health [9, 10, 39].

The main physiological consequence of EIB is a significant reduction in aerobic capacity parameters, including the oxygen threshold (VO2max or VO2peak), and a deterioration in the efficiency of gas exchange in the alveoli [5, 9, 10, 39]. This limitation becomes particularly evident during maximal exertion and in the final stages of competition. Studies evaluating athletes during a demanding stair run have shown that athletes who experienced a post-exercise decrease in FEV1 of 10% or more achieved significantly worse times and ran slower, especially in the final, decisive phase of the test [40, 41].

Evidence of how severely untreated EIB limits an athlete is provided by the promising results of targeted pharmacotherapy. In elite marathon runners with EIB, the use of long-term combination therapy (ICS + LABA) led to a significant reduction in bronchoconstriction and a real increase in VO2peak within just 30 days, with no changes to their existing training loads [9]. A similar relationship was observed in soccer players with newly diagnosed EIB, in whom the initiation of treatment resulted in a dramatic improvement in VO2peak test results, while in the group of healthy players (without EIB), these parameters remained stable [10]. It has also been demonstrated in a population of amateur soccer players that controlling bronchospasm through pharmacotherapy results in an improvement in VO2max, which unequivocally confirms that subclinical EIB constitutes a mechanical barrier to achieving full performance capacity at the highest intensities [11].

However, the impact of EIB on the respiratory system of elite athletes is more complex and does not always manifest as a classic decrease in FEV1 on spirometry. New data point to the immense importance of so-called small airway dysfunction (SAD). In studies conducted on professional cyclists with isolated SAD (in whom no standard, diagnostic 10% or greater decline in FEV1 was observed), it was shown that their overall exercise capacity was significantly lower, and their lung ventilation reserve was markedly worse compared to healthy peers. These parameters were nearly identical in them to those of cyclists with full-blown, classic EIB [12]. This demonstrates that subclinical changes in the distal parts of the bronchial tree can silently limit a professional athlete's respiratory reserves.

In the context of daily athletic practice, EIB drastically reduces the quality of individual training sessions. Symptoms such as shortness of breath, wheezing, or a persistent cough very often force the athlete to prematurely interrupt intense interval sessions or subconsciously avoid so-called high-intensity training intervals. In younger athletes, this is also a common reason for missing scheduled training sessions and physical education classes [7, 10, 42].

From the perspective of a long-term athletic career, the consequences are even more serious. Competitive training in endurance sports involves many hours of hyperventilation. Cycles of micro-injuries to the epithelium and its continuous repair (injury-repair cycles), repeated over the years, lead to the development of chronic inflammation, persistent hyperreactivity, and peribronchial fibrosis. This phenomenon, which is the essence of airway remodeling, is currently regarded in sports medicine as an occupational disease of endurance athletes [5, 6, 12].

Additionally, untreated EIB devastates an athlete's well-being and quality of life, generating fear of maximal physical exertion, which in the long term results in a reduction in total "training volume" over months and years [7, 42].

On the other hand, a key and optimistic conclusion drawn from the scientific literature is that the implementation of proper treatment and wise environmental modifications allow athletes with EIB to perform full training loads and compete at the absolute highest, world-class level, with their performance fully matching that of their healthy peers [5, 6, 10].

6. Conclusions

Diagnosing exercise-induced bronchoconstriction in elite athletes based solely on reported subjective symptoms is insufficient and carries a very high risk of diagnostic error [7, 8]. The gold standard in sports medicine remains the objective assessment of the decline in

FEV1 using standardized provocation tests (ECT, EVH). Furthermore, due to the frequent coexistence of comorbidities, it is necessary to rigorously differentiate EIB from exercise-induced laryngeal obstruction (EILO) using continuous laryngoscopy during exercise (CLE), which helps avoid unjustified polypharmacy [13, 33].

In the pathomechanism of EIB in elite athletes, permanent hyperventilation and the resulting osmotic-thermal stress on the epithelium play a key role. These phenomena initiate an inflammatory cascade and irreversible airway remodeling, often through a mechanism completely independent of IgE-mediated classical atopy [1, 2]. Subclinical dysfunction of the bronchial tree—including the small airways (SAD)—constitutes a mechanical ventilatory barrier that has been proven to lower the maximal oxygen uptake (VO2max) and determines a decline in the body's performance during maximal exercise loads [10, 12].

Optimizing treatment for athletes with EIB requires an immediate shift away from chronic monotherapy with short-acting beta-2 agonists (SABA) due to the proven risk of rapid receptor tachyphylaxis [5, 14]. The gold standard is chronic control of inflammation using inhaled glucocorticosteroids (ICS), which effectively restores normal respiratory parameters [9]. Strict adherence to the therapeutic dosing limits specified in World Anti-Doping Agency (WADA) regulations allows elite athletes to fully restore respiratory capacity and compete in a fully legal and safe manner [5, 14].

Disclosure

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