



QUALITY IN SPORT

eISSN 2450-3118 · Open Access · Peer-reviewed

apcz.umk.pl/QS Nicolaus Copernicus University in Toruń



PACEK, Aleksandra, BRYL, Jakub, PLUTA, Wioleta, PLEWA-KIJAK, Aleksandra, MILANIUK, Roksana, DRZEWIECKA, Aleksandra, SURÓWKA, Beata, DANISZEWSKI, Szymon, CZERNY, Izabela, TALAROWSKI, Krzysztof, TYLEC, Piotr, ŚCIUBAK, Arkadiusz and OSTOJSKA, Kinga. Pool Chlorination By-Products and Airway Hyperresponsiveness in Swimmers: Pathomechanism, Pulmonary Diagnostics and Preventive Management — A Narrative Synthesis of Evidence Published Since 2020. *Quality in Sport*. 2026;75:75256. <https://doi.org/10.12775/QS.2026.75.75256>

ARTICLE TIMELINE

Received: 12.08.2026. Accepted: 22.09.2026. Published: 26.09.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych). © The Authors 2026.

OPEN ACCESS · CC BY-NC-SA 4.0 This article is published with open access under the License Open Journal Systems of Nicolaus Copernicus University in Toruń, Poland, and is distributed under the terms of the Creative Commons Attribution Non-commercial Share Alike License (<http://creativecommons.org/licenses/by-nc-sa/4.0/>), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the work is properly cited. The authors declare no conflict of interest regarding the publication of this paper.

Pool Chlorination By-Products and Airway Hyperresponsiveness in Swimmers: Pathomechanism, Pulmonary Diagnostics and Preventive Management — A Narrative Synthesis of Evidence Published Since 2020

Aleksandra Pacek, ORCID <https://orcid.org/0009-0009-4950-3577>

E-mail: pacek.ola@wp.pl

Medical University in Lublin, Aleje Raławickie 1, 20-059 Lublin, Poland

Jakub Bryl, ORCID: <https://orcid.org/0009-0000-8772-2839>

E-mail: Jakub.bryl0202@gmail.com

Medical University in Lublin, Aleje Raławickie 1, 20-059 Lublin, Poland

Wioleta Pluta, ORCID <https://orcid.org/0009-0004-8990-7238>

E-mail: violeta.pluta@gmail.com

Independent Public Health Care Facility in Puławy, Józefa Bema 1, 24-100 Puławy, Poland

Aleksandra Plewa-Kijak ORCID: <https://orcid.org/0009-0003-7258-2863>

E-mail: plewaola@gmail.com

Stefan Wyszyński Regional Specialist Hospital in Lublin, Breast Surgery Department

Roksana Milaniuk, ORCID <https://orcid.org/0009-0004-8199-4672>

E-mail: roksana.demianiuk@gmail.com

University Clinical Hospital No. 4 in Lublin, Doktora Kazimierza Jaczewskiego 8, 20-954
Lublin, Poland

Aleksandra Drzewiecka, ORCID <https://orcid.org/0009-0004-7030-3266>

E-mail: ola.drzewiecka25@gmail.com

University Clinical Hospital No. 4 in Lublin, Doktora Kazimierza Jaczewskiego 8, 20-954
Lublin, Poland

Beata Surówka, ORCID <https://orcid.org/0009-0008-0284-0077>

E-mail: beatasurowka@wp.pl

University Clinical Hospital No. 1 in Lublin, Stanisława Staszica 16, 20-081 Lublin, Poland

Szymon Daniszewski, ORCID <https://orcid.org/0009-0009-9481-1149>

E-mail: szymon.daniszewski.1999@wp.pl

Independent Public Healthcare Institution of the Ministry of the Interior and Administration
in Lublin, ul. Grenadierów 3, 20-331, Lublin, Poland

Izabela Czerny, ORCID <https://orcid.org/0009-0005-6770-4314>

E-mail czernyizab@gmail.com

Independent Public Healthcare Institution of the Ministry of the Interior and Administration
in Lublin, ul. Grenadierów 3, 20-331, Lublin, Poland

Krzysztof Talarowski, ORCID: <https://orcid.org/0009-0007-5651-6208>

E-mail: Krzysztoftalarowski704@gmail.com

Independent Public Health Care Centre in Puławy, Józefa Bema 1, 24-100 Puławy, Poland

Piotr Tylec, ORCID <https://orcid.org/0009-0000-0490-2974>

E-mail: piotrtylec@wp.pl

Independent Public Health Care Facility in Puławy, Józefa Bema 1, 24-100 Puławy, Poland

Arkadiusz Ściubak, ORCID <https://orcid.org/0009-0002-3292-1085>

E-mail arkadiusz.sciubak@opoczta.pl

University Clinical Hospital No. 4 in Lublin, Doktora Kazimierza Jaczewskiego 8, 20-954
Lublin, Poland

Kinga Ostojka, ORCID: <https://orcid.org/0009-0005-9266-9215>

E-mail kinga.ostojka@gmail.com

The Copernicus Memorial Hospital in Łódź, Pabianicka 62, 93-513 Łódź

Corresponding Author

Aleksandra Pacek, ORCID <https://orcid.org/0009-0009-4950-3577>

E-mail: pacek.ola@wp.pl

Medical University in Lublin, Aleje Racławickie 1, 20-059 Lublin, Poland

Abstract

Background: Competitive swimmers train in indoor facilities where chlorine-based disinfection generates volatile by-products like trichloramine. Aquatic disciplines report the highest prevalence of lower airway dysfunction of any sporting group. The clinical picture, often labelled ‘swimmer’s asthma’, sits between classical exercise-induced bronchoconstriction and chemically mediated airway disorder.

Aim: To synthesise evidence (2020 onwards) on mechanisms by which chlorination by-products and sustained hyperpnoea injure airway epithelium in swimmers, appraise diagnostic strategies, and set out preventive and pharmacological options within anti-doping constraints.

Material and methods: This narrative synthesis retrieved PubMed/MEDLINE records (Jan 2020–Jul 2026), combining exposure, respiratory, and anti-doping terms. Priority was given to consensus statements, meta-analyses, and clinical trials.

Results: Meta-analyses place lower airway dysfunction prevalence at 21.8% across athletes and 39.9% in aquatic disciplines. The dominant mechanism combines osmotic/mechanical stress from hyperpnoea with chemical irritation, disrupting the epithelial barrier. Club cell protein 16 marks this disruption, though interpretation is complicated by acute vs. chronic changes. Symptom-based diagnosis performs poorly; objective bronchoprovocation is required (eucapnic voluntary hyperpnoea shows 46% sensitivity, 74% specificity). Inhaled beta-2 agonists are permitted below daily thresholds, but the therapeutic-to-reportable margin is narrow.

Conclusions: Swimmers' airway hyperresponsiveness is an occupational disorder of mixed osmotic and chemical origin, not classical asthma. Objective testing must precede prescription, treatment must align with anti-doping limits, and environmental control of by-products requires greater clinical emphasis.

Keywords: trichloramine, disinfection by-products, swimmers, airway hyperresponsiveness, exercise-induced bronchoconstriction, club cell protein 16, anti-doping, narrative review

1. Introduction

Swimming is among the most widely recommended forms of physical activity, and its cardiorespiratory and metabolic benefits are well documented [1]. That endorsement sits alongside a paradox that has occupied respiratory medicine for three decades: the athletes who spend the most time in indoor pools display the highest prevalence of lower airway dysfunction of any competitive group [13].

Chlorine remains the disinfectant of choice in most indoor facilities because of its efficacy and cost. It reacts with nitrogenous organic matter introduced by bathers — urea, sweat, skin cells — to generate a family of disinfection by-products including mono-, di- and trichloramine and the trihalomethanes [1]. Trichloramine is the species of greatest respiratory interest: it is volatile, denser than air, and accumulates in the layer immediately above the water surface and deck, precisely the layer from which a swimmer inhales [2].

The exposure is compounded by the mode of breathing. Exercise shifts respiration from nasal to oronasal, bypassing the conditioning and filtering capacity of the upper airway, and elite training volumes sustain minute ventilation at levels far above resting values for hours each day [5]. The airway epithelium is therefore subjected simultaneously to osmotic and mechanical stress and to a chemical irritant [1].

The clinical consequence has acquired the informal label ‘swimmer’s asthma’. That label is convenient but imprecise, and the imprecision has practical costs. Classical exercise-induced bronchoconstriction, chemically induced airway hyperresponsiveness and atopic asthma are not the same entity, may respond differently to inhaled therapy, and carry different implications for career planning and for the interpretation of anti-doping regulation [11]. Distinguishing them is a task for objective pulmonary diagnostics rather than for symptom history [14].

A further consideration gives the question public-health as well as sporting significance. Competitive swimming careers typically begin in childhood, so that the cumulative exposure of an elite adult swimmer may extend over fifteen years or more of near-daily attendance in indoor facilities. Whatever the magnitude of the per-session effect, the relevant exposure metric is cumulative, and the population at issue is not confined to the small number of athletes who reach international level. Recreational swimmers, swimming teachers and lifeguards share the same environment, and the last two groups do so as an occupational exposure in the formal sense [1]. It should be recorded at the outset that the childhood

question is not settled: a systematic review of cohort and cross-sectional studies of children who swam in chlorinated pools in early childhood found no association with subsequent asthma development [3]. The evidence assembled below concerns competitive athletes subject to far larger cumulative exposures, and should not be read as confirming a risk to ordinary childhood swimming.

Research Objective. Three aims guided the present work. The first was to set out the mechanistic pathway linking chlorination by-products and sustained hyperpnoea to airway epithelial injury, with particular attention to the biomarker evidence. The second was to appraise the diagnostic instruments available to the pulmonologist assessing a symptomatic swimmer. The third was to examine preventive and pharmacological management within the constraints that anti-doping regulation imposes on the treating physician.

Research Problems. Three questions organise the synthesis that follows:

1. By what mechanism do chlorination by-products and training-related hyperpnoea interact to produce airway hyperresponsiveness in swimmers, and how far does biomarker evidence support that account?
2. Which diagnostic strategies reliably distinguish exercise-induced bronchoconstriction from chemically induced airway hyperresponsiveness and from atopic asthma in this population?
3. What preventive measures and pharmacological options are available, and how do current anti-doping thresholds constrain the treating physician?

Research Hypotheses. It is postulated, first and foremost, that airway hyperresponsiveness in swimmers arises from a combined osmotic and chemical insult to the epithelium rather than from either exposure acting alone. A second postulate holds that symptom-based assessment substantially misclassifies affected swimmers in both directions, so that objective bronchoprovocation is a prerequisite rather than a refinement. A third holds that environmental control of by-product generation is a legitimate therapeutic target, but one for which the supporting evidence is considerably thinner than for pharmacological management.

2. Materials and methods

Evidence from controlled clinical studies is set here alongside mechanistic, environmental and regulatory sources, and hypotheses for subsequent investigation are proposed on that footing. Adoption of a narrative rather than a systematic design was deliberate: the

constituent literature spans respiratory physiology, occupational hygiene, sports medicine and anti-doping science, and these fields differ so markedly in method and outcome definition that a single pooled estimate would misrepresent them. What is offered is an interpretive synthesis across those domains rather than the derivation of one pooled estimate of effect.

Interrogation of the PubMed/MEDLINE database spanned January 2020 to July 2026, subject to a small number of explicitly identified exceptions set out in the paragraph below. Exposure terminology (“trichloramine”, “nitrogen trichloride”, “chloramine”, “disinfection by-products”, “indoor swimming pool”) was paired within the search strings with respiratory-outcome terminology (“asthma”, “airway hyperresponsiveness”, “exercise-induced bronchoconstriction”, “epithelial injury”, “club cell protein”, “CC16”) and with regulatory terminology (“beta-2 agonist”, “anti-doping”, “therapeutic use exemption”), using Boolean operators. The review question was framed using a population–exposure–outcome structure. Full open access was applied as an inclusion criterion rather than a preference: every retained record is freely available to any reader, verified individually through PubMed Central deposition or an explicit open licence. Two records are exempted from this rule and are marked with an asterisk in the reference list; both are meta-analyses produced by subgroups of the International Olympic Committee consensus group, and no open-access equivalent reporting the same primary data exists. All records were written in English. Reference lists of the retrieved consensus statements and systematic reviews were hand-searched for further sources.

Priority in appraisal was given to systematic reviews with meta-analysis and to controlled clinical and environmental studies. Narrative commentaries, conference abstracts and non-peer-reviewed material were excluded. Two pre-2020 publications were retained because each constitutes the primary open-access source for a mechanism or a methodological standard that the later literature cites rather than restates: reference [1], establishing the inflammatory basis of exercise-induced bronchoconstriction, and reference [11], defining the methodological standard for eucapnic voluntary hyperpnoea. Both are freely available through PubMed Central. All remaining records were published from 2020 onwards. This limited departure from the date restriction is reported here in the interests of methodological transparency.

Because the constituent studies differ in design, exposure assessment and outcome definition, no single risk-of-bias instrument was applied across the corpus. Methodological limitations

relevant to interpretation are instead discussed at the point at which each body of evidence is presented, and revisited in Section 4.

3. Results

3.1. The Indoor Pool as a Respiratory Exposure Environment

3.1.1. Formation and Behaviour of Chlorination By-Products

Chlorine-based disinfectants react with nitrogenous organic matter introduced by bathers to produce a large family of by-products. Among the volatile species, the chloramines and the trihalomethanes dominate respiratory concern, and trichloramine has attracted the most attention because of its combination of volatility, irritant potency and characteristic odour [1]. Its physical behaviour is clinically relevant: being denser than air, trichloramine tends to accumulate close to the water surface and above the deck, which is where the breathing zone of a swimmer lies [2].

Exposure is not confined to inhalation. By-products may also be ingested with pool water and absorbed through the skin, so that the total internal dose in a competitive swimmer reflects several simultaneous routes [1]. For the airway, however, inhalation of the near-surface layer during high-ventilation exercise is the pathway of principal interest.

3.1.2. Determinants of Airborne Concentration and the Role of Ventilation

Airborne trichloramine concentration is not a fixed property of a facility. It varies with bather load, with the concentration of free chlorine and organic precursors in the water, with water and air temperature, and with the design and operation of the ventilation system [1,2]. Computational modelling of a pool enclosure has shown that outdoor air intake and the air-change rate are among the principal determinants of the concentration achieved in the occupied zone [2]. That analysis also exposes a genuine engineering conflict: pool halls are energy-intensive largely because of evaporation from the basin, and building codes therefore favour low air velocity above the water in order to limit evaporation and preserve thermal comfort, while dilution of trichloramine favours higher air movement [2]. Ventilation strategy is consequently a compromise rather than an optimisation, and this constrains what environmental control can realistically achieve.

3.2. Pathomechanism of Airway Injury

3.2.1. The Combined Osmotic and Chemical Insult

The prevailing mechanistic account of exercise-induced bronchoconstriction in elite athletes attributes airway narrowing to injury of the epithelium sustained during repeated high-volume ventilation, with water loss from the airway surface producing an osmotic stimulus for mediator release [5]. In swimmers this mechanism does not operate in isolation. The inhaled air is warm and humid, which should in principle attenuate the osmotic stimulus, yet aquatic athletes nevertheless show the highest prevalence of airway dysfunction of any discipline group [13]. The most economical explanation is that a second, chemical insult is superimposed on the mechanical one, and that the two act on the same target: the epithelial barrier [1].

Two features of swimming exercise intensify the delivered dose. The shift from nasal to oronasal breathing during exertion removes the conditioning and particle-trapping function of the nose, and the swimmer inhales from the layer immediately above the water in which volatile by-products are most concentrated [1,2]. Inhalation of hypotonic aerosol droplets generated at the water surface has been proposed as a further contributor to epithelial disturbance [1].

3.2.2. Epithelial Barrier Disruption and Club Cell Protein 16

Club cell protein 16, a low-molecular-weight anti-inflammatory secretoglobin produced by non-ciliated epithelial cells of the distal airways, has become the most frequently used peripheral marker of epithelial integrity in this field. Because of its small size it diffuses across the epithelial barrier into the circulation, and an acute increase in its serum or urinary concentration is interpreted as evidence of increased barrier permeability [1].

The evidence in swimmers requires careful reading. Exercise, but not pharmacological provocation with mannitol, has been shown to increase urinary club cell protein in elite swimmers, which locates the signal in the mechanical and ventilatory dimension of the exposure rather than in bronchial smooth-muscle responsiveness as such [1]. Serum concentrations of the protein have also been reported as elevated at baseline in competitive swimmers relative to controls, alongside markers of innate immune activation and neutrophilic inflammation [1].

Two interpretive cautions apply, and the present authors regard them as decisive for how this biomarker should be used clinically. First, the direction of change is not uniform: acute barrier leakage raises circulating concentrations, whereas chronic destruction or dedifferentiation of club cells lowers the capacity to secrete the protein at all, so that a normal value may conceal opposing processes in equilibrium [1]. Second, the protein is renally cleared, and both the intensity of preceding exercise and the timing of sampling therefore influence the value obtained. A single measurement without a defined exercise and sampling protocol is difficult to interpret, and comparisons across studies that differ in these respects should be made with corresponding reserve.

3.2.3. Inflammatory Response and Structural Change

Airway inflammation in competitive swimmers is characteristically mixed rather than purely eosinophilic. Studies of induced sputum have identified neutrophilic infiltration alongside eosinophilic markers, and eosinophil cationic protein in sputum supernatant has been shown to correlate with sputum eosinophil counts in elite swimmers, supporting its use as an accessible marker of the eosinophilic component [8]. The inflammatory basis of exercise-induced bronchoconstriction more generally involves mediator release from mast cells and eosinophils in response to the epithelial insult [5].

The natural history of these changes carries an important message for career counselling. A five-year prospective follow-up of forty-two highly trained swimmers found that airway inflammation and bronchial hyperresponsiveness differed according to whether athletes continued or ceased high-level competition, indicating that at least part of the airway phenotype in this population is a consequence of ongoing exposure rather than a fixed constitutional trait [1]. This observation is the strongest available argument that the disorder is, in the relevant sense, occupational [1].

3.2.4. Oxidative Stress and the Systemic Dimension

A further element of the mechanistic account concerns oxidative stress. Chlorine-derived oxidants are chemically capable of protein and lipid oxidation at the epithelial surface, and the inflammatory infiltrate itself generates reactive species, so that oxidative and inflammatory processes are difficult to separate in this setting [1,5]. The practical significance is that markers of oxidative stress rise and fall with training load as well as with exposure, which limits their specificity as indicators of the chemical component of the insult.

It is worth stating explicitly what the biomarker literature does not currently establish. No marker examined to date — club cell protein 16, eosinophil cationic protein, exhaled nitric oxide or measures of oxidative stress — has been shown to discriminate reliably between the osmotic and the chemical component of the exposure in an individual swimmer. Each responds to both. The consequence for clinical practice is that biomarkers may support a diagnosis of airway inflammation or epithelial disturbance but cannot at present attribute it to the pool environment specifically, and should not be presented to athletes as though they could.

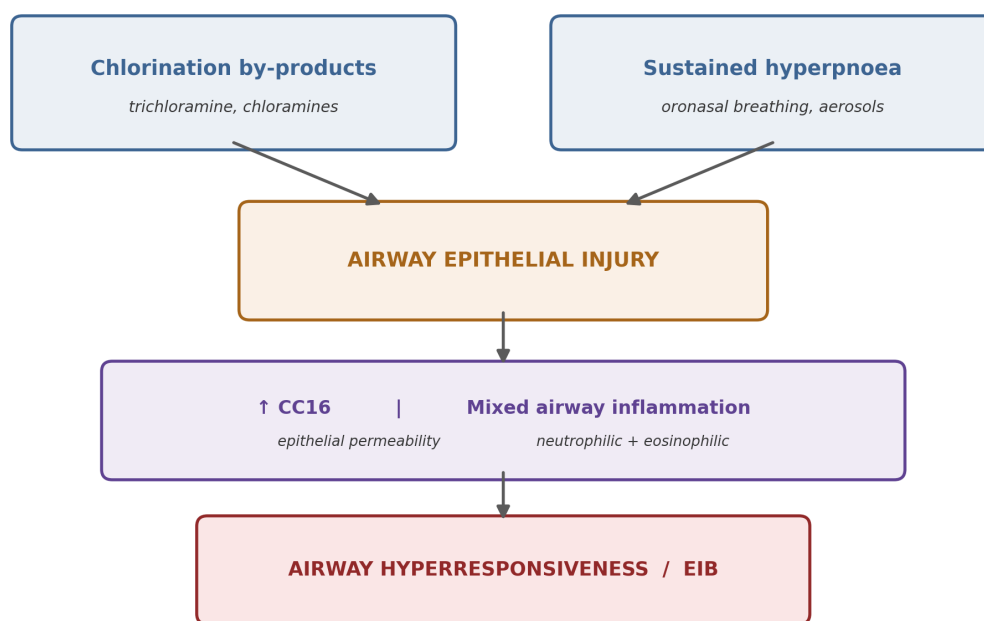


Figure 1. Proposed convergent pathway to airway hyperresponsiveness in the competitive swimmer. Two exposures act on a single target: inhaled volatile disinfection by-products, chiefly trichloramine, and the mechanical and osmotic stress of sustained hyperpnoea with oronasal breathing and inhalation of hypotonic aerosol from the water surface. Both converge on the airway epithelium, where barrier disruption produces two measurable consequences — leakage of club cell protein 16 into serum and urine, indicating increased permeability, and a mixed neutrophilic and eosinophilic inflammatory response detectable in induced sputum. Airway hyperresponsiveness, expressed clinically as exercise-induced bronchoconstriction, is the functional endpoint. Compiled by the authors as a schematic overview on the basis of references [1,5,8]; the diagram is illustrative, does not reproduce quantitative data from those sources, and represents a synthesis rather than a single validated model.

3.2.5. Why the Swimming Paradox Matters Mechanistically

The osmotic model of exercise-induced bronchoconstriction predicts that cold, dry inspired air constitutes the principal hazard, which is why cross-country skiers were long regarded as the paradigm case [5]. Swimmers breathe air that is warm and close to saturation, and on the osmotic model alone they should therefore be relatively protected. That they are not — that they in fact exceed winter-sport athletes in measured prevalence [13] — is the single most instructive observation in this literature, because it cannot be accommodated without invoking a second mechanism.

Three candidate explanations have been advanced. The first is chemical irritation by inhaled by-products acting directly on the epithelium [1,2]. The second is the sheer magnitude and duration of the ventilatory load in elite swimming, which may generate mechanical shear stress sufficient to injure the epithelium even when the inspired air is fully conditioned [5]. The third is the inhalation of hypotonic aerosol droplets from the water surface, which would deliver an osmotic insult by a route the conventional model does not consider [1]. These explanations are not mutually exclusive, and the available human data do not permit their relative contributions to be apportioned. The present authors note that this unresolved apportionment is precisely what limits the specificity of preventive recommendations: a facility-level intervention addresses the first mechanism only, while training-load management addresses the second.

3.3. Epidemiology in Aquatic Athletes

A systematic review and meta-analysis conducted by a subgroup of the International Olympic Committee consensus group, encompassing 64 studies and 37,643 athletes from more than 21 countries, reported an overall prevalence of lower airway dysfunction of 21.8% (95% CI 18.8% to 25.0%), a figure that has remained stable across three decades [13]. Disaggregation by discipline is the finding of principal relevance here: prevalence reached 39.9% (95% CI 23.4% to 57.1%) in aquatic sports, 29.5% (95% CI 22.5% to 36.8%) in winter-based sports and 25.1% (95% CI 20.0% to 30.5%) in elite endurance athletes [13].

Two qualifications should accompany these figures. Heterogeneity across the included studies was very high, and the confidence interval for the aquatic subgroup is correspondingly wide, spanning roughly a quarter to well over half of athletes [13]. Estimates also depend heavily on the method of ascertainment: among studies employing objective testing, the highest prevalence was observed where direct bronchial provocation was used, at 32.8% (95% CI 19.3% to 47.2%) [13]. Reported prevalence in swimmers should therefore be read as a function of methodology as much as of biology.

The stability of the overall figure across three decades is itself informative [13]. Awareness of airway dysfunction in athletes, the availability of objective testing and the sophistication of pool water treatment have all advanced considerably over that period, yet the aggregate prevalence has not fallen. Several readings are possible: that improvements in facility management have been offset by rising training volumes; that detection and true incidence have moved in opposite directions; or that the dominant driver is the ventilatory demand of

elite endurance training rather than the chemical environment. The present authors regard the third reading as insufficient on its own, since it would not explain why aquatic athletes exceed other endurance groups by a wide margin, but the data assembled here cannot adjudicate between the first two.

Prevalence estimates continue to be revised as new national cohorts are screened, and reported figures in elite populations outside the European and North American literature that dominates the existing meta-analysis are beginning to appear [12]. Readers should therefore treat the values in Table 1 as the best current synthesis rather than as settled parameters.

Table 1. Prevalence of lower airway dysfunction in athletes by discipline group and ascertainment method.

Subgroup	Prevalence	95% CI	Source
All athletes	21.8%	18.8–25.0%	[13]
Aquatic disciplines	39.9%	23.4–57.1%	[13]
Winter-based sports	29.5%	22.5–36.8%	[13]
Elite endurance athletes	25.1%	20.0–30.5%	[13]
Studies using direct bronchial provocation	32.8%	19.3–47.2%	[13]

3.4. Pulmonary Diagnostics

3.4.1. The Inadequacy of Symptom-Based Assessment

Reliance on reported symptoms misclassifies athletes in both directions. Under-recognition is well described: athletes frequently fail to identify or report respiratory symptoms, and screening studies have repeatedly uncovered previously undiagnosed cases [7,14]. Over-diagnosis is the mirror problem, since breathlessness, cough and chest tightness during heavy exertion are non-specific and occur in healthy athletes. The practical consequence is that a diagnosis of exercise-induced bronchoconstriction in a swimmer should not be made, and inhaled therapy should not be commenced, on symptom history alone [11,14].

The reasons for the mismatch between symptoms and objective findings are instructive. Elite swimmers are habituated to intense respiratory sensation and may normalise breathlessness that would prompt medical attention in a sedentary person. Conversely, the sensation of restricted breathing during maximal effort is universal, and attributing it to bronchoconstriction is a natural but frequently incorrect inference. Neither error is corrected by more careful history-taking, since both arise from the limits of introspective report rather than from inadequate questioning [7,14].

3.4.2. Bronchoprovocation Testing

Objective confirmation rests on bronchoprovocation, which may be direct, using an agent acting on bronchial smooth muscle, or indirect, using a stimulus that acts through mediator release from inflammatory cells. Eucapnic voluntary hyperpnoea has long been advanced as the reference indirect test in athletes, and its methodology and interpretation have been reviewed in detail [6].

The diagnostic accuracy data assembled by the IOC consensus subgroup temper any assumption that a single test settles the question. Against a clinical reference standard, eucapnic voluntary hyperpnoea confirmed the diagnosis with 46% sensitivity and 74% specificity, and exercise challenge testing with 51% sensitivity and 84% specificity [14]. Sensitivity of this order means that a negative indirect challenge in a symptomatic swimmer does not exclude the diagnosis, and that serial or complementary testing is often required. Reproducibility of eucapnic voluntary hyperpnoea has itself been the subject of dedicated investigation [7], and feasibility outside specialist centres is not guaranteed [6].

Field-based and sport-specific challenges have been proposed to improve ecological validity, on the reasoning that a laboratory ergometer challenge reproduces neither the workload pattern nor the environmental exposure of a swimming session [7]. For the swimmer specifically, this argument has particular force: a challenge performed in dry laboratory air omits the chemical component of the exposure entirely.

Table 2. Diagnostic instruments in the assessment of the symptomatic swimmer.

Instrument	Type	Reported performance / role	Source
Symptom history	Subjective	Insufficient alone; misclassifies in both directions	[7,11,14]
Eucapnic voluntary hyperpnoea	Indirect provocation	46% sensitivity, 74% specificity vs clinical reference	[6,14]
Exercise challenge test	Indirect provocation	51% sensitivity, 84% specificity vs clinical reference	[14]
Direct bronchial provocation	Direct	Highest prevalence yield among objective methods	[13,14]
Field / sport-specific challenge	Indirect provocation	Improves ecological validity; less standardised	[7]
Induced sputum (ECP, cell counts)	Inflammatory marker	Characterises eosinophilic component	[8]

Instrument	Type	Reported performance / role	Source
Serum / urinary CC16	Epithelial marker	Research tool; direction of change context-dependent	[1]

3.4.3. Differentiating the Phenotypes

The distinction between classical exercise-induced bronchoconstriction, chemically induced hyperresponsiveness and atopic asthma is clinically consequential but is not resolved by any single test. Several features assist. Atopic asthma is typically accompanied by sensitisation on skin-prick or specific IgE testing, by an eosinophilic inflammatory profile and by symptoms that persist outside the training environment. Chemically driven hyperresponsiveness would be expected to correlate with exposure intensity, to show a mixed or neutrophil-predominant inflammatory pattern, and to attenuate during periods away from the pool — an expectation consistent with the longitudinal observation that airway status differs between swimmers who continue and those who cease high-level training [1]. Exercise-induced bronchoconstriction in the narrow sense is episodic, tied to exertion, and need not be accompanied by persistent inflammation [11].

It should be stated plainly that this triad is a conceptual scheme rather than a validated diagnostic algorithm. The present authors are not aware of a prospective study that has assigned swimmers to these categories using predefined criteria and demonstrated differential treatment response. The scheme is offered as a structure for clinical reasoning and as a hypothesis for testing, not as a classification with established discriminant validity.

3.4.4. Differential Diagnosis Beyond the Lower Airway

Breathlessness in a swimmer is not invariably bronchial in origin. Diagnosis in this population is acknowledged to be difficult because aquatic athletes frequently display higher than normal baseline lung function, so that a clinically meaningful decrement may still fall within the predicted range, and because symptoms overlap with those of several non-bronchial conditions [4]. A structured differential is therefore required before inhaled therapy is commenced.

Two entities deserve particular mention in the aquatic context, and are raised here as established clinical background rather than as findings drawn from the sources reviewed above. Rhinitis and rhinosinusitis are common in swimmers and may account for symptoms reflexively attributed to the lower airway. Inducible laryngeal obstruction produces exertional

breathlessness and stridor that mimic bronchoconstriction, does not respond to inhaled bronchodilator, and is misdiagnosed as refractory asthma with some regularity. The practical rule that follows is one the present authors would emphasise: a swimmer who reports no benefit from an adequately dosed inhaler should prompt reconsideration of the diagnosis rather than escalation of the prescription — a point at which anti-doping prudence and sound clinical reasoning converge.

The biomarker approach has been taken furthest in a cohort drawn from an Olympic swimming team, where blood eosinophil count, serum total immunoglobulin E, fractional exhaled nitric oxide and skin-prick positivity were combined into a composite score. Serum immunoglobulin E and exhaled nitric oxide were higher in the asthmatic athletes, and positive prick testing was substantially more frequent, so that the composite was associated with a positive diagnosis [4]. The value of that work for the present argument is twofold: it demonstrates that a panel outperforms any single marker in this population, and it locates the discriminating signal in the atopic rather than the irritant dimension — which is consistent with the position taken in Section 3.2.4 that no available marker isolates the chemical component of the exposure.

3.5. Pharmacological Management and Anti-Doping Constraints

Asthma and exercise-induced bronchoconstriction do not preclude elite performance, and modern management allows affected athletes to compete at the highest level; the requirement is that treatment complies both with respiratory guidelines and with anti-doping regulation [11]. Untreated airway inflammation is itself harmful, carrying a risk of epithelial damage, remodelling and fibrosis, so therapeutic conservatism is not a neutral option [11].

All beta-2 agonists are prohibited substances, with defined exceptions for specified inhaled agents used below daily thresholds; within those limits no therapeutic use exemption is required [11]. The thresholds are set to correspond to normal treatment guidelines, and the regulatory framework has tightened over time — the 2023 revision restricted salbutamol to 600 micrograms in any eight-hour period, a change intended to prevent supratherapeutic bolus dosing while preserving ordinary clinical use [11]. Urinary thresholds operate in parallel with dosing limits, so that an athlete adhering to the permitted inhaled dose may nonetheless produce a reportable sample and must then demonstrate, through a controlled pharmacokinetic study, that the result followed therapeutic administration [11].

This regulatory architecture has a clinical corollary that deserves emphasis. Overuse of short-acting beta-2 agonists among elite athletes has been documented and analysed as a distinct phenomenon [10], and reliance on rescue medication is in any case a marker of inadequate control rather than of adequate treatment. In a swimmer whose salbutamol requirement approaches the permitted ceiling, the appropriate response is reassessment of inflammatory control and of environmental exposure, not titration towards the threshold.

A related practical difficulty concerns documentation. The burden of demonstrating that a reportable urinary concentration followed permitted therapeutic use falls on the athlete, and the controlled pharmacokinetic study required for that purpose is neither quick nor universally accessible [11]. Contemporaneous records of dose, timing and device — maintained prospectively rather than reconstructed after an adverse analytical finding — are therefore part of competent clinical care in this population, not an administrative afterthought. The treating physician who prescribes an inhaled beta-2 agonist to a tested athlete without establishing such a record has left the athlete materially exposed.

Because the Prohibited List is revised annually, the figures in Table 3 are presented as they are described in the cited literature and must be verified against the current List before any clinical application. The sources reviewed here do not constitute a regulatory reference.

Table 3. Inhaled beta-2 agonists permitted without therapeutic use exemption, as described in the cited literature. Current thresholds must be verified against the prevailing Prohibited List.

Agent	Permitted inhaled dose	Urinary threshold	Source
Salbutamol	1600 µg / 24 h; ≤600 µg / 8 h (2023 revision)	1000 ng/mL	[11]
Formoterol	54 µg / 24 h delivered dose	40 ng/mL	[11]
Salmeterol	200 µg / 24 h	Not established	[11]
Vilanterol	25 µg / 24 h	Not established	[11]
All other beta-2 agonists / non-inhaled routes	Prohibited; TUE required	—	[11]

The performance question that underlies the regulation is itself the subject of active investigation. Systematic appraisal of the evidence for performance enhancement by substances on the Prohibited List has been undertaken using formal inferential frameworks [11], and the ergogenic potential of inhaled beta-2 agonists beyond bronchodilation has been reviewed in detail, including the observation that formoterol at the permitted ceiling may exert effects extending to muscle hypertrophy [11]. Pharmacological management of

exercise-induced bronchospasm has recently been synthesised at the umbrella-review level [11].

One diagnostic avenue deserves separate mention because it addresses the ecological-validity problem directly. Unsupervised field-based exercise challenge testing, performed with application-based spirometry outside the laboratory, has been evaluated as a means of confirming lower airway dysfunction in the athlete's own training environment [7]. For the swimmer the attraction is obvious: a challenge conducted poolside reproduces both the workload pattern and the chemical exposure, neither of which a laboratory ergometer test reproduces. The approach also lowers the practical barrier to serial testing, which matters given the moderate sensitivity of single challenges discussed above.

Against this, field testing sacrifices the standardisation that makes laboratory results comparable between occasions and between athletes. Ambient conditions, effort regulation and device handling all vary, and an unsupervised test cannot verify that the target ventilation was reached. The present authors would position field challenge as complementary to, rather than a replacement for, laboratory bronchoprovocation — useful for confirming a suspected diagnosis when laboratory testing has been negative, and for monitoring an established one across a training season.

3.6. Preventive Management

Preventive strategy operates at three levels. At the level of the water, control of precursor loading — through pre-swim showering, bather-load management, filtration and the maintenance of appropriate free-chlorine concentrations — limits the substrate available for by-product formation [1]. At the level of the air, ventilation design determines the concentration reaching the breathing zone, and modelling indicates that outdoor air intake and air-change rate are the principal levers, subject to the energy and thermal-comfort constraints described in Section 3.1.2 [2]. At the level of the athlete, periodised exposure, attention to symptoms and objective monitoring form the clinical component.

Alternative disinfection approaches, including ultraviolet and ozone-assisted treatment and non-chlorine systems, are frequently proposed as a solution to the by-product problem. The present authors note that the evidence base for a respiratory benefit from such substitutions is substantially thinner than the mechanistic rationale would suggest, and that any

recommendation of this kind currently rests more on plausibility than on controlled outcome data in athletes.

Two further preventive avenues merit mention. Attenuating the airway response to a hostile inspired environment through a physical barrier has been demonstrated in a different setting, where a breathing mask reduced acute airway responses to exercise in sub-zero conditions [9]; whether an analogous approach is feasible or acceptable in swimming has not, so far as the present authors are aware, been established. Second, structured screening of adolescent and recreational athletes has been investigated as a means of earlier detection [3], which is relevant given that competitive swimming careers frequently begin in childhood.

A final observation concerns the distribution of responsibility. Almost every preventive lever identified above — bather load, water chemistry, filtration, ventilation rate, outdoor air fraction — lies with the facility operator rather than with the athlete or the treating physician. The clinical literature nonetheless frames management overwhelmingly in terms of what the athlete should take and how the athlete should be tested. The present authors suggest that this asymmetry is itself worth examining: an occupational model of the disorder [1] would imply a corresponding shift of preventive obligation towards the operator of the environment, as is standard in other occupational respiratory exposures.

4. Discussion

4.1. Interpretation and Practical Application

The evidence reviewed supports a coherent, if incompletely proven, account: swimmers develop airway hyperresponsiveness through the convergence of two exposures on a single vulnerable structure, the epithelial barrier. The strongest empirical pillars of that account are the epidemiological gradient favouring aquatic disciplines [13], the biomarker evidence of increased epithelial permeability associated with exercise in swimmers [1], the mixed inflammatory profile documented in induced sputum [8], and the longitudinal observation that airway status varies with continuation or cessation of training [1].

For the clinician, three practical positions follow. The first is diagnostic: given a sensitivity of 46% for eucapnic voluntary hyperpnoea against a clinical reference [14], a single negative challenge should not close the assessment of a symptomatic swimmer, and the limitations of laboratory testing in a chemically exposed population should be acknowledged explicitly in the report. The second is therapeutic: prescription should follow objective confirmation,

should aim at inflammatory control rather than at rescue-medication availability, and should be documented against current anti-doping thresholds at the point of prescription rather than retrospectively [10,11]. The third is environmental: a swimmer with poorly controlled symptoms warrants enquiry into the facility — bather load, ventilation, water chemistry — as part of the clinical assessment, an enquiry that is presently uncommon in respiratory practice.

A caution applies to the framing of the condition to athletes and coaches. Describing the disorder as ‘asthma’ without qualification risks two errors: it may imply a constitutional and permanent diagnosis where the evidence suggests a substantial exposure-dependent component [1], and it may direct management towards pharmacology alone when environmental modification is also available. The present authors regard the occupational framing advanced for elite athletes generally [1] as the more accurate description for this population, while acknowledging that this is an interpretive position rather than a settled classification.

The question of whether affected swimmers should modify or curtail training deserves a direct answer, since athletes ask it. The evidence does not support a general recommendation to leave the sport. It does support informing an athlete with established, poorly controlled airway disease that part of the process appears to be exposure-dependent and may improve with reduced exposure [1], while acknowledging that the magnitude and reversibility of that improvement in an individual case cannot presently be predicted. Presenting this honestly — as a real but unquantified prospect rather than as either a promise or a dismissal — is in the view of the present authors preferable to the confident advice in either direction that athletes frequently receive.

Honesty about the strength of the causal claim requires one further qualification, and the open-access literature supplies it directly. A systematic review of children exposed to chlorinated pools in early childhood, conducted to PRISMA standards with a registered protocol, found no association between that exposure and subsequent asthma [3]. Two readings are compatible with the evidence as a whole: that the dose experienced by a recreational child swimmer lies below any threshold of effect, while the cumulative dose of a competitive career does not; or that the association observed in elite swimmers reflects training intensity and selection into the sport rather than the chemical environment. The present authors favour the first, because it accommodates both bodies of evidence without discarding either, but the second cannot be excluded from the data reviewed here.

4.2. Limitations of the Present Review

This review is narrative rather than systematic. No protocol was registered, screening was not duplicated, and no quantitative synthesis was performed. The emphasis on records published from 2020 onwards, adopted to keep the synthesis current and verifiable, necessarily excluded relevant older primary literature, including much of the foundational epidemiology of pool exposure; the retention of six pre-2020 sources mitigates but does not remove this limitation. Restriction to English-language publications introduces a potential language bias, which is material here because a substantial body of European occupational-hygiene work on pool air quality is published in other languages. Several statements in Sections 3.2.2, 3.4.3, 3.6 and 4.1 are interpretive positions of the present authors rather than findings reported in the cited literature, and are identified as such where they appear.

A further limitation concerns the evidence itself rather than its synthesis. Direct human data linking measured individual trichloramine exposure to objectively confirmed airway outcomes in competitive swimmers remain sparse; much of the mechanistic argument is assembled from studies that measured exposure and outcome in different populations. The account presented in Figure 1 should be read with that in mind.

Two additional limitations concern generalisability. The meta-analytic prevalence figures reported here derive overwhelmingly from European and North American cohorts [13], and facility standards, water-treatment practice and regulatory limits on airborne trichloramine differ substantially between jurisdictions; extrapolation to settings with different infrastructure is therefore uncertain. Furthermore, the great majority of the mechanistic and diagnostic studies cited recruited adult athletes, whereas the exposure begins in childhood, and the developing airway may respond differently. The paediatric literature examined here [3,7] addresses screening rather than mechanism.

4.3. Recommendations for Future Research

Four priorities emerge. First, studies should pair individual-level exposure assessment — personal rather than stationary air sampling — with objectively confirmed respiratory outcomes in the same swimmers, since the current literature largely separates these measurements. Second, the club cell protein 16 signal requires standardisation: a defined exercise protocol, defined sampling times and simultaneous serum and urinary measurement would allow the opposing acute-leakage and chronic-depletion processes to be distinguished, which single time-point designs cannot achieve [1].

Third, the phenotypic triad set out in Section 3.4.3 should be tested prospectively, assigning symptomatic swimmers by predefined criteria and examining whether the categories predict treatment response and natural history. Fourth, intervention trials of environmental modification — ventilation regime, alternative disinfection, bather-load policy — with respiratory endpoints in athletes are needed to convert a plausible preventive rationale into evidence-based recommendation [2]. Given the practical difficulty of randomising facilities, well-designed interrupted time-series or stepped-wedge designs are likely to be the realistic approach.

A note on the evidence base of this review is warranted here rather than in the limitations, because it bears on how the conclusions should be weighted. The corpus was restricted to freely accessible publications, and this restriction is not neutral: open-access publishing is unevenly distributed across the specialties that contribute to this field. Environmental and sports-science journals are well represented in open repositories, whereas much of the clinical respiratory and allergy literature remains behind subscription. The synthesis presented above is therefore likely to be weighted towards the environmental and physiological dimensions of the problem relative to the clinical, and a reader with full institutional access would encounter a somewhat different balance of emphasis. The two exceptions retained in the reference list, both meta-analyses supplying the quantitative backbone of Sections 3.3 and 3.4.2, were admitted precisely because their exclusion would have removed the most reliable numbers in the review.

4.4. A Note on Terminology

The term ‘swimmer’s asthma’ is used widely in coaching and lay contexts and occasionally in the clinical literature. It is retained in the title of this review because it is the term under which readers will search, but the present authors would discourage its use in clinical documentation. It conflates at least three separable entities, it implies a settled diagnosis where objective confirmation may be absent, and it attributes causation to the sport in a way the evidence does not fully license. ‘Airway hyperresponsiveness in a competitive swimmer, confirmed by [named test]’ is longer but carries none of these liabilities, and it makes the evidential basis of the diagnosis visible to the next clinician who reads the record.

5. Conclusions

Aquatic athletes carry the highest documented burden of lower airway dysfunction of any competitive discipline group, with meta-analytic prevalence approaching 40% against 21.8% across athletes overall. The mechanism most consistent with the available evidence is a convergent one, in which sustained hyperpnoea and inhaled chlorination by-products act together on the airway epithelium, with barrier disruption reflected in club cell protein 16 and followed by a mixed inflammatory response and functional hyperresponsiveness.

The clinical implication is that symptom-based diagnosis is inadequate and that objective bronchoprovocation must precede prescription — while recognising that the sensitivity of the available tests is moderate, so that a negative result does not exclude the condition in a symptomatic swimmer. Pharmacological management is effective and compatible with elite competition, but operates within narrow and periodically revised anti-doping thresholds that must be checked at the time of prescribing rather than assumed.

The most under-developed element of current practice is environmental. Ventilation design and water chemistry demonstrably determine the concentration of irritant reaching the swimmer's breathing zone, yet these variables rarely enter the respiratory consultation, and controlled evidence that modifying them improves athlete outcomes is largely absent. Treating swimmers' airway disease as an occupational disorder — in which exposure control ranks alongside pharmacotherapy — is, in the view of the present authors, the framing most likely to advance both clinical care and research design.

Disclosure

Author's Contributions:

Conceptualization: Aleksandra Pacek, Jakub Bryl;

Methodology: Aleksandra Pacek, Jakub Bryl, Wioleta Pluta, Roksana Milaniuk;

Literature analysis: Aleksandra Pacek, Jakub Bryl, Wioleta Pluta, Roksana Milaniuk, Aleksandra Drzewiecka, Beata Surówka, Izabela Czerny, Szymon Daniszewski, Arkadiusz Ściubak, Aleksandra Plewa-Kijak

Writing - original draft: Aleksandra Pacek, Jakub Bryl, Aleksandra Drzewiecka, Beata Surówka, Izabela Czerny, Aleksandra Plewa-Kijak

Writing - review and editing: Aleksandra Pacek, Jakub Bryl, Wioleta Pluta,, Izabela Czerny, Aleksandra Plewa-Kijak, Piotr Tylec, Krzysztof Talarowski, Szymon Daniszewski,

Visualization: Piotr Tylec, Krzysztof Talarowski, Aleksandra Pacek, Arkadiusz Ściubak, Aleksandra Plewa-Kijak

All authors have read and agreed to the published version of the manuscript.

Funding: This review received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable. All data discussed are available in the cited published sources.

Acknowledgements: Thanks are due to the investigators whose published work made this synthesis possible.

Conflicts of Interest: The authors declare no conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

Claude (Anthropic) was employed during preparation of this manuscript to assist with identification of literature, organisation of structure and refinement of language. Every cited source was subsequently checked by the authors against the original publication, the content was revised where necessary, and responsibility for the scientific substance, the interpretation placed upon it and the final form of the manuscript rests entirely with the authors.

References

1. Boraczyński M, Balcerek T, Rożkiewicz N, Pabiszczak M, Harasymczuk M, Sławska A, Lutomski P. Pulmonary function in swimmers exposed to disinfection by-products: a narrative review. *Front Physiol.* 2025;15:1473302. <https://doi.org/10.3389/fphys.2024.1473302>
2. Proulx H, Hallé S. A numerical study of the impacts of outdoor air intake and air changes per hour on the trichloramine concentrations in a swimming pool enclosure. *Front Built Environ.* 2022;8:957973. <https://doi.org/10.3389/fbuil.2022.957973>
3. Silvestre CL, Freitas-Dias R, Costa EC, dos Santos MAM, Correia JL, Peixoto DM, Rodrigues EA, Correia MAV. Asthma risk in young people who attended chlorinated swimming pools in early childhood. *Rev Paul Pediatr.* 2025;43:e2024251. <https://doi.org/10.1590/1984-0462/2025/43/2024251>
4. Csoma B, Sydó N, Szűcs G, Seres É, Erdélyi T, Horváth G, Csulak E, Merkely B, Müller V. Exhaled and systemic biomarkers to aid the diagnosis of bronchial asthma in elite water sports athletes. *Med Sci Sports Exerc.* 2024;56(7):1256–1264. <https://doi.org/10.1249/MSS.0000000000003419>
5. Hallstrand TS, Moody MW, Wurfel MM, Schwartz LB, Henderson WR Jr, Aitken ML. Inflammatory basis of exercise-induced bronchoconstriction. *Am J Respir Crit Care Med.* 2005;172(6):679–686. <https://doi.org/10.1164/rccm.200412-1667OC>
6. Hull JH, Ansley L, Price OJ, Dickinson JW, Bonini M. Eucapnic voluntary hyperpnea: gold standard for diagnosing exercise-induced bronchoconstriction in athletes? *Sports Med.* 2016;46(8):1083–1093. <https://doi.org/10.1007/s40279-016-0491-3>
7. Bahr R, Ljungberg H, Reier-Nilsen T, Stang JS, Flatsetøy H, Isachsen M, Nordlund B. Unsupervised field-based exercise challenge tests to support the detection of exercise-induced lower airway dysfunction in athletes. *BMJ Open Sport Exerc Med.* 2023;9(3):e001680. <https://doi.org/10.1136/bmjsem-2023-001680>
8. Paciência I, Rocha AR, Farraia M, Sokhatska O, Delgado L, Couto M, Moreira A. Eosinophil cationic protein (ECP) correlates with eosinophil cell counts in the induced sputum of elite swimmers. *Porto Biomed J.* 2022;7(2):e155. <https://doi.org/10.1097/j.pbj.0000000000000155>

9. Stenfors N, Persson H, Tutt A, Tufvesson E, Andersson EP, Ainegren M, Hanstock HG. A breathing mask attenuates acute airway responses to exercise in sub-zero environment in healthy subjects. *Eur J Appl Physiol.* 2022;122(6):1473–1484.
<https://doi.org/10.1007/s00421-022-04939-x>
10. Vertadier N, Trzepizur W, Faure S. Overuse of short-acting beta-2 agonists (SABAs) in elite athletes: hypotheses to explain it. *Sports (Basel).* 2022;10(3):36.
<https://doi.org/10.3390/sports10030036>
11. Cricco R, Segreti A, Stirpe E, Ferro A, Ciano M, Cipriani F, Fossati C, Ussia GP, Pigozzi F, Grigioni F. Performance-enhancing effects of inhaled medications: implications for heart, muscle function, and doping detection in athletes. *J Funct Morphol Kinesiol.* 2025;10(4):462. <https://doi.org/10.3390/jfmk10040462>
12. Cao Z, et al. Prevalence of exercise-induced bronchoconstriction in elite Chinese summer sport athletes. *J Sport Health Sci.* 2026:101131.
<https://doi.org/10.1016/j.jshs.2026.101131>
13. * Price OJ, Sewry N, Schwellnus M, Backer V, Reier-Nilsen T, Bougault V, 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.
<https://doi.org/10.1136/bjsports-2021-104601>
14. * Reier-Nilsen T, Sewry N, Chenuel B, Backer V, Larsson K, Price OJ, 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. <https://doi.org/10.1136/bjsports-2022-106059>

** Access note. All references are freely available in full text without subscription, with the two exceptions marked by an asterisk. Both are meta-analyses prepared by subgroups of the International Olympic Committee consensus group and are available by institutional subscription only; they were retained because no open-access publication reports the same primary data.*