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### **► To cite this version:**

Martin Schwellnus, Paolo Emilio Adami, Valérie Bougault, Richard Budgett, Hege Havstad Clemm, et al.. International Olympic Committee (IOC) consensus statement on acute respiratory illness in athletes part 2: non-infective acute respiratory illness. *British Journal of Sports Medicine*, 2022, 56 (19), pp.1089-1103. 10.1136/bjsports-2022-105567. hal-03791952

**HAL Id: hal-03791952**

**<https://hal.science/hal-03791952>**

Submitted on 11 Apr 2024

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# **International Olympic Committee (IOC) Consensus Statement on Acute Respiratory Illness in Athletes Part 2: Non-infective acute respiratory illness**

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## **Word count:**

Abstract: 288

Main text (excl Tables and Figs): 8735

## **ABSTRACT**

Acute respiratory illness (ARill) is common and threatens the health of athletes. ARill in athletes forms a significant component of the work of Sport and Exercise Medicine (SEM) clinicians. The aim of this consensus is to provide the SEM clinician with an overview and practical clinical approach to non-infective ARill in athletes. The IOC Medical and Scientific Committee appointed an international consensus group to review ARill in athletes. Key areas of ARill in athletes were originally identified and six subgroups of the IOC Consensus group established to review the following aspects: 1) epidemiology / risk factors for ARill, 2) infective ARill, 3) non-infective ARill, 4) acute asthma/EIB and related conditions, 5) effects of ARill on exercise / sports performance, medical complications / return-to-sport, and 6) acute nasal/laryngeal obstruction presenting as ARill. Following several reviews conducted by subgroups, the sections of the consensus documents were allocated to “core” members for drafting and internal review. An advanced draft of the consensus document was discussed during a meeting of the main consensus core group, and final edits were completed prior to submission of the manuscript. This document (Part 2) of this consensus focusses on respiratory conditions causing non-infective ARill in athletes. These include non-inflammatory obstructive nasal, laryngeal, tracheal or bronchial conditions, or non-infective inflammatory conditions of the respiratory epithelium that affect the upper and/or lower airways, frequently as a continuum. The following aspects of more common as well as lesser-known non-infective ARill in athletes are reviewed: epidemiology, risk factors, pathology / pathophysiology, clinical presentation and diagnosis, management, prevention, medical considerations, and risks of illness during exercise, effects of illness on exercise / sports performance, and return-to-sport (RTS) guidelines.

## **KEY POINTS:**

- Acute illness accounts for up to ~50% of all medical consultations at major sporting events with ~50% of cases involving the respiratory system
- Acute respiratory illness (ARill) includes acute respiratory infections and non-infective ARill
- Causes of non-infective ARill in athletes can involve predominantly the upper (e.g. structural nasal obstruction, acute allergic and non-allergic rhinitis / rhinosinusitis, and

exercise-induced laryngeal obstruction - EILO) or the lower airways (lower airway dysfunction, and rarer conditions such as excessive dynamic airway collapse - EDAC) and tracheobronchomalacia - TBM)

- In this consensus, we provide the sport and exercise medicine (SEM) clinician with an overview and practical clinical approach to the diagnosis, management, return-to-sport (RTS) decision making and prevention of these non-infective causes of acute respiratory illness in athletes

## **1. INTRODUCTION**

Protection of the health of athletes and illness prevention have been formally identified as high priorities by the International Olympic Committee (IOC) since 2009.(1) Acute illness that threatens athlete health is a significant component of Sport and Exercise Medicine (SEM) clinicians' work at international single sport (2-4) and multisport events, including the Olympic (5, 6) and Paralympic Games.(7) The IOC Medical and Scientific Committee identified "acute respiratory illness (ARill) in the athlete" as a high priority area to protect the health of athletes and is the focus of this IOC Consensus paper.

Acute illness can account for ~50% of all medical consultations at major sporting events (2, 4-7), and in ~50% of cases involve the respiratory system.(2, 4-7) Although the majority of ARill in athletes are suspected acute respiratory infections (ARinf), mostly by clinical diagnosis,(2-4, 6, 8) ARill can have a non-infective cause.(4)

The aim of this consensus is to provide the SEM clinician with an overview and practical clinical approach to acute non-infective respiratory illness in athletes. This consensus document complements Part 1, which focuses on ARinf.(9) The specific focus of Part 2 is a review of epidemiology, risk factors, pathology / pathophysiology, clinical presentation and diagnosis, management, prevention, medical considerations, risks of illness during exercise, effects of illness on exercise / sports performance, and return-to-sport (RTS) guidelines. This consensus group recognises that there are international guidelines for several of the respiratory conditions. In this consensus statement we aim to be consistent with these guidelines, but we specifically highlight unique aspects of non-infective ARill in athletes.

## **2. METHODS AND CONSENSUS GROUP WORKFLOW**

The IOC Medical and Scientific Committee appointed a chair (MS) for this consensus group in July 2019. This consensus statement process took a unique approach, following key steps as detailed in Part 1.(9) In brief, subgroups of the IOC Consensus group were originally established to review the literature in the following six areas: 1) epidemiology / risk factors for ARill, 2) infective ARill, 3) non-infective ARill, 4) acute asthma/EIB and related conditions, 5) effects of ARill on exercise / sports performance, medical complications / return-to-sport, and 6) acute nasal/vocal cord dysfunction presenting as ARill. A chair, who

was a “core” member of the consensus group, was identified for each subgroup. The six subgroups were created, and subgroup chairs nominating and adding content-specific expert “corresponding” members to their group. Following the work of the subgroups, the sections of the consensus documents were allocated to “core” members for drafting and internal review. An advanced draft of the consensus document was discussed during a meeting of the main consensus core group in Lausanne, Switzerland on 11-12 October 2021. Final edits were completed in the weeks after the meeting, prior to submission of the manuscript.

### **3. GENERAL APPROACH TO NON-INFECTIVE ACUTE RESPIRATORY ILLNESS IN ATHLETES**

ARill in the athlete can present with a variety of symptoms or signs, with considerable overlap in the predisposing factors, pathophysiology, and clinical presentation. ARill in athletes can be broadly classified by: 1) an anatomical classification predominantly affecting the upper or lower airways, and 2) a pathological classification of infective and non-infective ARill. However, both these classifications have limitations. For example, non-infective conditions presenting predominantly in the upper airways in athletes, such as allergic rhinitis, are frequently associated with lower airway dysfunction (e.g., asthma).(10) Similarly, non-infective conditions such as allergies can predispose individuals to acute respiratory infections (ARinf). It is important that the SEM clinician is aware of these limitations in the classifications. If concurrent or overlapping ARill in athletes are not recognised, this shortcoming may compromise recognition, diagnosis and management. With these qualifications, and for the purposes of this consensus statement, the following terminology, and classifications for ARill in athletes were agreed upon by the consensus group.

#### **3.1. Anatomical classification of ARill in athletes**

The consensus group recognises that in many conditions causing ARill, including allergy, asthma, infection, and other inflammatory conditions,(11, 12) there is an anatomical and pathological continuum given the interaction between upper and lower airways. However, the usage of the terms “*upper*” or “*lower*” airways disease is still common in the literature when referring to both infective and non-infective causes of ARill. In this context, “*upper*” refers to symptoms, signs, and pathology arising from the larynx and above, while “*lower*” refers to symptoms, signs, and pathology arising from below the level of the larynx. In this consensus statement the group have referred to “predominantly” upper or lower airway conditions,

based on a predominant cluster of upper or lower airway symptoms, signs or pathological involvement.

### **3.2. Pathological classification of acute respiratory illness in athletes**

Acute respiratory illness (ARill) in an athlete can present with general non-specific symptoms. In many studies among athletes the pathology of ARill could not be attributed specifically to an infection or a non-infective cause. In these studies, the ARill in athletes was classified as “*undiagnosed*”. Acute respiratory infection (ARinf) in athletes is defined and discussed in Part 1 of this consensus statement.<sup>(9)</sup> Non-infective ARill in athletes is the focus of this consensus paper and was defined as an illness not caused by an infection. There are several conditions that cause non-infective ARill and a broad pathological sub-classification of either “*inflammatory*” or “*non-inflammatory*” causes can be applied.

In summary, the consensus group classified non-infective ARill based on the anatomical and pathological classification of ARill (Table 1) and these conditions will now be reviewed.

**[Insert Table 1]**

**Table 1: Classification of non-infective acute respiratory illness (ARill) in athletes**

Anatomical classification	Pathological classification	Conditions in athletes
Predominantly upper airways	<ul style="list-style-type: none"> <li>Inflammatory non-infective (ARill)</li> </ul>	<ul style="list-style-type: none"> <li>Non-infective rhinitis / rhinosinusitis *                             <ul style="list-style-type: none"> <li>Allergic rhinitis / rhinosinusitis</li> <li>Non-allergic rhinitis / rhinosinusitis (chemically / mechanically mediated e.g. pollution, gases, air temperature)</li> </ul> </li> <li>Non-infective pharyngitis / laryngitis (chemically / mechanically mediated e.g. Gastroesophageal reflux disease (GORD), pollution, gases, air temperature)</li> </ul>
	<ul style="list-style-type: none"> <li>Non-inflammatory non infective (ARill): Upper airway obstruction (anatomical / mechanical)</li> </ul>	<ul style="list-style-type: none"> <li>Structural (anatomical) nasal obstruction**                             <ul style="list-style-type: none"> <li>Static</li> <li>Dynamic (non-inflammatory)</li> </ul> </li> <li>Oropharyngeal obstruction</li> <li>Laryngeal obstruction                             <ul style="list-style-type: none"> <li>Exercise induced laryngeal obstruction (EILO)</li> <li>Static (anatomical) laryngeal obstruction</li> </ul> </li> </ul>
Predominantly lower airways	<ul style="list-style-type: none"> <li>Inflammatory non-infective (ARill)</li> </ul>	<ul style="list-style-type: none"> <li>Lower airway dysfunction (allergy mediated, chemically / mechanically mediated e.g. pollution, gases, air temperature, etc)                             <ul style="list-style-type: none"> <li>Asthma</li> <li>Exercise-induced bronchoconstriction (EIB)</li> <li>Airway hyper-responsiveness (AHR)</li> </ul> </li> </ul>
	<ul style="list-style-type: none"> <li>Non-inflammatory non infective (ARill): Lower airway obstruction (anatomical / mechanical)</li> </ul>	<ul style="list-style-type: none"> <li>Expiratory / Excessive Central Airway Collapse (ECAC)                             <ul style="list-style-type: none"> <li>Excessive Dynamic Airway Collapse (EDAC)</li> <li>Tracheobronchomalacia (TBM)</li> </ul> </li> </ul>

\*: Can cause nasal obstruction due to mucosal swelling (inflammatory)

\*\* : Can cause nasal obstruction due to anatomic / structural causes and can be static (fixed) or dynamic (non-inflammatory e.g. nasal valve collapse)



#### **4. GENERAL APPROACH TO ACUTE NON-INFECTIVE RHINITIS / RHINOSINUSITIS IN ATHLETES**

Rhinosinusitis is defined as inflammation of the nasal (rhinitis) and paranasal sinus (sinusitis) mucosa and characterised by: 1) two or more symptoms, one of which should be either nasal blockage/obstruction/congestion/discharge, facial pain/pressure, reduction/loss of smell, and 2) endoscopic nasal examination of nasal polyps/mucopurulent discharge/mucosal obstruction and/or computerised tomography (CT) changes.(11, 13) Acute rhinosinusitis can be either infective or non-infective. Acute infective rhinosinusitis in the athlete is reviewed in Part 1 of the IOC Consensus on “acute respiratory illness in the athlete”.(9) Non-infective rhinosinusitis can be subclassified as either allergic or non-allergic. In non-allergic rhinitis, mucosal inflammation is caused by a trigger, irritant or exposure, which together with nasal hyperreactivity (NHR) leads to mucosal swelling and nasal obstruction. Mucosal inflammation can lead to nasal obstruction. A detailed review of nasal obstruction in the athlete was specifically commissioned for this IOC Consensus statement and has been published.(13) A clinical diagnostic approach to an athlete presenting with symptoms or signs of nasal obstruction is summarised in Figure 1. In this diagnostic approach, nasal obstruction can originate from either structural (anatomical) causes or mucosal causes (usually swelling). Causes of mucosal swelling can be subclassified into infective (acute infective rhinitis / rhinosinusitis) or non-infective (non-infective acute rhinitis / rhinosinusitis).(13). The structural causes of nasal obstruction and the non-infective causes of acute rhinitis / rhinosinusitis in athletes will be reviewed first.

**[insert Figure 1 here]**

#### **5. NASAL OBSTRUCTION (STRUCTURAL / ANATOMICAL)**

##### **5.1. Introduction and definition**

Athletes with ARill can present with symptoms and signs of nasal obstruction. Nasal obstruction can be defined as either permanent anatomical “*narrowing of the nasal cavity*” or “*the subjective feeling*” of reduced nasal airflow. Structural causes of nasal obstruction are subclassified as static or dynamic. Generally, static obstruction is a permanent anatomical cause of obstruction whereas dynamic obstruction is intermittent.

## **5.2. Epidemiology**

The incidence of nasal trauma leading to static structural nasal obstruction in athletes has not been thoroughly investigated. Nasal injuries potentially resulting in static obstruction are more common in competitive sports, adolescent and young adult athletes, and males. The majority of nasal injuries affect the nasal bone and septum.(14-16) The prevalence and incidence of structural static and dynamic nasal obstruction in athletes is not well documented and this requires investigation.(16)

## **5.3. Risk factors**

A history of previous trauma to the nose is the most frequent risk factor associated with a structural static (permanent) anatomical cause of nasal obstruction. Risk factors associated with dynamic nasal obstruction in athletes are not well documented but include cranial nerve injury (after previous surgery or trauma), which can lead to nasal valve insufficiency during forceful nasal breathing.(17)

## **5.4. Pathology and pathophysiology**

The pathophysiology of static and dynamic nasal obstruction in athletes varies. Static obstruction is a permanent anatomical abnormality narrowing the nasal airway not influenced by airflow and pressure changes. Dynamic obstruction is influenced by airflow and pressure changes due to a weakness in the soft tissue of the nose. Collapse occurs when the breathing rate or intensity increases, given the Venturi effect where increased flow results in higher negative pressures in the nasal cavity.(18)

## **5.5 Clinical presentation and diagnosis**

A comprehensive clinical approach to the diagnosis of nasal obstruction in athletes is described in the commissioned published review for this IOC Consensus statement.(13) A key element in the diagnosis is a good history and the most common acute symptoms are blocked nose, nasal congestion, or nasal discharge. More chronic symptoms are obligate mouth breathing, snoring, poor sleeping habits, and a dry throat in the morning. There are a variety of validated questionnaires adopted by otorhinolaryngologists to screen for nasal conditions, but no athlete-specific screening tool exists. In athletes that present with nasal symptoms, the following stepwise approach to the diagnosis of static and dynamic nasal obstruction is proposed:(13) 1) screen for possible nasal obstruction (NOSE score), 2) obtain a detailed history of nasal symptoms, followed by a general medical history, 3) conduct a

systematic examination of the nose to determine the causes of obstruction, and 4) decide on the use of special investigations (such as cone beam sinus CT scans) to determine the nature, severity and cause of nasal obstruction [**Insert video link 1 here**].

## **5.6 Management**

In athletes with structural dynamic causes nasal obstruction, temporary relief with various internal or external nasal dilating devices during training, competition, recovery or during sleep can be considered.(19-21) Early referral to an experienced otorhinolaryngologist for further surgical management is recommended.(22, 23) In general, surgery is the only permanent solution for structural static causes of nasal obstruction.

## **5.7 Prevention**

Reducing the risk of nasal trauma during sport is the most important primary prevention strategy. If acute nasal trauma does occur, secondary prevention strategies to reduce the risk of complications are important. Nasal trauma should first be treated according to advanced trauma life support (ATLS) principles.(24) Immediate reduction of a displaced nasal fracture is recommended and surgical reduction within 7 days. A septal haematoma requires urgent intervention given the risk of infection, or destruction of the septum with saddle nose deformity resulting in complete nasal obstruction.(25)

## **5.8 Medical considerations and risks**

In athletes, permanent structural nasal obstruction can lead to several health problems. These include poor quality of life, poor sleep and inadequate recovery from training or competition. Mild symptoms of structural obstruction can worsen when athletes are exposed to triggers from various sports-specific environments that cause additional mucosal swelling (rhinitis) such as cold, pollution, irritants, chlorinated water, and allergens.(26)

## **5.9 Effect on exercise and sports performance**

There is little evidence that alterations in nasal airflow negatively impact exercise or sports performance, or that nasal airflow is reduced by exercise.(27, 28) Two main reasons for this are: 1) as exercise intensity increases, ventilation increases concomitantly and breathing switches from nasal to oral, usually at a minute ventilation of approximately 35-45 L/min, resulting in a reduced resistance to the airflow,(29) and 2) the sympathetic nervous system

response during exercise results in vasoconstriction of the vessels in the nasal valve region, which increases airflow through the nose and nasal passages.(30)

### **5.10. Return-to-sport considerations**

In general, athletes with mild- to moderate nasal obstruction due to structural abnormality can continue training even though they may experience mild symptoms during exercise.

However, sleep disturbance and inadequate recovery may cause reductions in sport or exercise performance. Return-to-sport after acute nasal trauma is dependent on the extent of the injury, which should be discussed with an otolaryngologist. If there is a delay of more than 7 days after the acute injury, the recommendation is to delay surgical repair for at least 3 months. In general, contact sports can resume after ~ 6 weeks after surgery and the timing of surgery to coincide with the off-season period is advisable.

## **6. ALLERGIC RHINITIS / RHINOSINUSITIS**

### **6.1. Definition**

Allergic rhinitis is a symptomatic IgE-mediated inflammatory mucosal nasal condition resulting from allergen introduction in a sensitised individual.(31, 32) Where allergens are also the cause of inflammation in the mucosa of the paranasal sinuses, the condition is referred to as allergic rhinosinusitis.(11) In this consensus document, allergic rhinitis / rhinosinusitis will be discussed as a single clinical syndrome (and abbreviated as AR), unless otherwise stated.

### **6.2. Epidemiology**

A recent systematic review reported that 20-60% of athletes can be classed as having allergic rhinitis(33) based on a diagnosis of nasal symptoms and a positive skin prick test to at least one allergen. This prevalence is similar to the general population.(33) Except for a few studies in swimmers, the prevalence of allergic rhinosinusitis in athletes is not reported.(13)

### **6.3. Risk factors**

Athletes with a predisposition to allergy and atopy are at increased risk for AR. A few studies indicate a linear association between the volume of training per week in elite endurance sport

athletes and allergic symptoms, Th1 lymphocytes, and raised serum IgE levels.(34, 35) In some sports with environment-specific exposure, e.g. swimmers training in a chlorinated pool, there may be a higher risk of developing AR than other sports, but this remains to be determined.(36, 37)

#### **6.4. Pathology and pathophysiology**

Increased ventilation during exercise results in a greater volume and a deeper penetration of aeroallergens into the respiratory tract, potentially compromising the immunoregulatory function of the epithelial barrier.(13, 38) Atopy, which can be a strong predictor of allergic symptoms in the general population, is observed in many endurance athletes but does not appear to be directly linked to allergic symptoms(34, 35). Exercise-induced changes in immune function observed during and after intense exercise may predispose, at least in endurance athletes, to atopy and allergic symptoms.(34, 39) For example, atopic athletes, compared with non-atopic athletes, have a lower Th1/Th2 lymphocyte type balance which can increase susceptibility to upper ARinf.(34, 39) Environmental conditions (i.e. chlorinated water, polluted air and cold air) can also increase allergenic sensitisation, via reaction with airway surface lining fluid components, airway oxidative stress, and associated inflammatory cascade.(40-42)

#### **6.5. Clinical presentation and diagnosis**

The classical symptoms of AR are nasal obstruction due to mucosal swelling, itching, sneezing, rhinorrhoea and post-nasal drip.(32) Athletes with allergies may also complain of associated ocular itching, erythema and/or tearing, oral cavity or pharyngeal pruritis, sore throat, aural itching, or congestion, wheezing or cough, and/or asthma.(32) A clinical diagnosis of AR is made when athletes present with a history consistent with an allergic cause and at least one of the classical symptoms.(32, 43) The diagnosis can be confirmed by determining serum IgE as a general indicator of allergies, and by performing skin prick testing or in vitro testing methods(32, 43) for specific allergens. In cases of suspected rhinosinusitis a definitive diagnostic is made by a computerised tomography (CT) scan and/or nasal endoscopy.(13) In resource scarce settings, where skin prick tests or laboratory testing are not available, validated questionnaires such as the allergy questionnaire for athletes (AQUA) can be used to predict allergic disease.(44, 45) However, due to the potentially multiple causes of rhinitis in athletes, it is important to: 1) consider other causes such as those of infectious origin, and 2) provide evidence of allergies. If a diagnosis of AR is made,

pulmonary function testing (spirometry) should also be considered to diagnose concomitant LAD. Rhinitis is a risk factor for, and associated with, lower airway dysfunction, specifically asthma.(39, 43)

## **6.6. Management**

Management decisions are based on making a correct diagnosis and identifying the causative allergen/s. In the first instance, the causative allergen/s should be avoided if possible, and nasal rinsing advised, even if evidence is limited.(46) A 10 point visual analogue scale (VAS) from 0-10 can be used to assess and track symptomatic relief.(13, 47) If allergen avoidance and nasal rinsing is not fully effective to bring sufficient symptom relief, pharmacotherapy should be considered. When AR is acute and/or symptoms are mild (VAS<5), either antihistamines (oral or topical) or intranasal corticosteroids (with or without antihistamines) can be used.(48) When symptoms are more severe (VAS≥5), or experienced for more than a week, intranasal corticosteroids should be considered with or without anti-histamines.(13, 47) In addition, if AR impairs sports performance, negatively affects quality of life in general, or where regular treatment is insufficient to control symptoms, other treatment options may be considered.(47) These include anticholinergics, leukotriene receptor antagonists, mast cell stabilisers, or mucolytics. For bouts of acute exacerbation of rhinosinusitis, decongestants may be considered. The SEM clinician should refer to World Anti-Doping Agency (WADA) current standards when prescribing medications such as oral pseudoephedrine.(49) Finally, patient preferences, treatment availability and costs will determine the choice of treatment among first line medication.(48) Athletes should be followed up and if the VAS remains ≥5, despite correct use of intranasal corticosteroids, good compliance and adherence, the SEM clinician should consider referral of the athlete to an otorhinolaryngologist or an allergy specialist.(13)

## **6.7. Prevention**

The only known prevention for sensitisation of allergens is avoidance, at home and/or during sport. Assessing allergen levels in ambient air and pollutants' concentration through official websites may be useful when advising athletes where or when to practice their sport. Alpine regions above 1600m generally show markedly lower exposure to pollens, moulds and mites, which may reduce risk of AR in athletes and improve lung function in asthmatic subjects.(50)

### **6.8. Medical considerations and risks**

The main risk during exercise for untreated athletes suffering from allergic rhinitis is lower airway dysfunction, which includes exercise-induced bronchoconstriction (EIB) and asthma.(51, 52) Of interest, there are recent data showing an association between allergies in general and gradual onset musculoskeletal injuries in long-distance runners and cyclists.(53, 54) However, a causal relationship between allergies and gradual onset injuries remains to be established, and the potential mechanisms for this relationship are not clear. For example, it is not known if this may be because of allergies or medications used to treat allergies such as antihistamines or corticosteroids.(55, 56)

### **6.9. Effect on exercise and sports performance**

Allergic rhinitis causing nasal obstruction and post-nasal drip can disturb sleep and impair quality of life, thereby reducing exercise or sports performance.(26, 57-62) A decrease in endurance sports performance has been reported in healthy endurance athletes treated with histamine receptor blockers (selective H1- and H2-receptor blocker).(56, 63) However, to the best of our knowledge, there is no comparative study on the effects on performance with untreated symptomatic allergies vs. those treated with oral anti-histamine (H2-receptor blocker) treatment. While intranasal corticosteroids can be effective in improving quality of life and performance in elite athletes,(58) a recent study showed that 80% of athletes (endurance sports and football) with rhinitis reported impaired performance, including those under treatment.(59) In this study, 13% could not exercise at all due to allergic symptoms and among athletes using treatments, ~ 80% still had impaired training capabilities.(59) Therefore, the relationship between AR, allergies in general and exercise /sport performance in athletes requires further investigation.

### **6.10. Return-to-sport considerations**

In most cases of AR in athletes, training can continue but the SEM clinician may suggest, in individual athletes, decreases in training volume and intensity based on AR symptom severity. An important clinical consideration in RTS is that in case of a new diagnosis of AR, the lower airways should also be investigated to exclude lower airway dysfunction before an athlete continues with normal training.

## **7. NON-ALLERGIC RHINITIS / RHINOSINUSITIS**

### **7.1. Introduction and definition**

Non-allergic rhinitis / rhinosinusitis (NAR) is defined as acute or chronic rhinitis / rhinosinusitis in the absence of clinical symptoms and signs of infection, without evidence of allergies (normal systemic allergen specific IgE and negative skin prick tests for allergens).(64) This definition includes a variety of clinical syndromes (phenotypes) that vary according to severity, aetiology and type of inflammation.(64) For athletes, exercise itself can be a trigger as well as exposure to environmental irritants in certain sports (e.g. chlorine in swimmers, cold air in winter athletes, and changes in environmental conditions such as humidity, dry air and ambient air pollution that athletes may be exposed to).(26, 65, 66)

### **7.2. Epidemiology**

The incidence / prevalence of NAR in athletes is poorly documented and good evidence that unfavourable environmental conditions cause ARill in athletes is limited. The exceptions are cold air-induced rhinitis in winter athletes(67) and chemically-induced rhinitis in swimmers, linked at least partly to the chlorine disinfection.(37, 68)

### **7.3. Risk factors**

The type of sport and exposure to certain environmental conditions are risk factors for NAR. Predominantly upper airway respiratory symptoms, especially rhinorrhoea, are common in water sport and winter sport athletes.(37, 69) There is evidence that time spent in a chlorinated swimming pool, and the level of chloramines in the air are two risk factors associated with swimming-induced rhinitis.(37, 70, 71) Some early studies reported a dose-dependent effect of pollution during exercising on the frequency and severity of upper respiratory symptoms in healthy or asthmatic athletes, especially pharyngeal irritation and nasal congestion.(72)

### **7.4. Pathology and pathophysiology**

A detailed review of each of the clinical syndromes of NAR is beyond the scope of the consensus document, but the differing pathophysiological mechanisms that influence management principles are summarised in Figure 2. There are two important clinical consideration for athletes: Firstly, athletes may present with NAR syndromes not related to sport. Secondly, there may be several pathophysiological mechanisms responsible for a clinical syndrome in an athlete including: various types of inflammation (e.g. non-allergic



rhinitis with eosinophilia syndrome, with eosinophils and mast cells, neutrophilic rhinitis, neurogenic inflammation), as well as neuronal imbalance or atrophic rhinitis (Figure 2).(26, 32, 36, 73) The physiological response to air pollutants and chlorine likely includes stimulation of cell and neurogenic airway inflammation as well as epithelial cell damage through the oxidative stress provoked by the reaction of pollutants with the airway surface lining content.(65, 74, 75)

**[Insert Figure 2 here]**

### **7.5. Clinical presentation and diagnosis**

It is important to make a specific diagnosis of the clinical syndrome of NAR in the athlete because this will influence decision making on management. The diagnosis of NAR is made by clinical examination and appropriate tests, and based on: 1) typical recurrent nasal respiratory symptoms when exposed to specific triggers, and 2) the exclusion of other or co-existing causes of allergic and infective rhinitis or respiratory disease.(13) It is important to note that, as in the general population, mixed rhinitis may occur in some athletes i.e. rhinitis symptoms that have both allergic and non-allergic components.(32) A diagnosis of a mixed rhinitis should be considered in cases where an athlete has an ineffective or partial response to medication.

### **7.6. Management**

There is limited research on the management of athletes with NAR caused by environmental factors. Therefore, management of NAR in athletes should align with the position paper of the European Academy of Allergy and Clinical Immunology.(64) The principles of management are both non-pharmacological and pharmacological. The mainstay of non-pharmacological treatment is avoidance of environmental and other conditions that cause NAR. Nose rinsing with saline before or after swimming, may help some swimmers exposed to chlorine-related products in pools. If environmental avoidance fails to suppress symptoms, pharmacotherapy may be considered. The clinical phenotype of NAR could influence decision making on pharmacological management using topical intranasal corticosteroid or antihistamines (Figure 2).(76) In athletes with underlying inflammation topical intranasal corticosteroid is the treatment of choice.(76, 77) In cases where there is no suspected inflammatory component, ipratropium is the preferred treatment for e.g. watery rhinorrhoea,(77) and cold-induced rhinitis.(69) Intranasal capsaicin can be useful if

ipratropium is insufficient.(77) In cases of suspected mixed rhinitis (allergic and non-allergic), a combination of anti-inflammatory and ipratropium is an option.

### **7.7. Prevention**

The key to prevention of NAR is avoiding environmental stimuli by reducing exposure.(78) During swimming, a nose clip may be effective in relieving nasal symptoms related to the chlorinated environment, or decrease their severity.(36) Facemasks may be advised to humidify and heat the inhaled air (cold air)(79) or partly filter at least particles, but not gases (air pollution).(80)

### **7.8. Medical considerations and risks**

There are no studies on acute or long-term risks associated with environmentally-induced NAR in athletes. It is not known if athletes with acute NAR are at higher risk of developing acute medical complications during exercise. Athletes with chronic NAR may be at higher risk of allergic disease or asthma, but this requires further investigation. Any long-term risk is likely related to the environmental trigger, the pathophysiology, and the frequency of exposure.

### **7.9. Effect on exercise / sports performance**

There are no data on the effects of NAR related to pollution, cold air or chlorine on exercise / sports performance and this requires further investigation. In general, detrimental effects on exercise / sports performance are more likely if there is exposure to pollution or if lower airway dysfunction co-exists with NAR.

### **7.10. Return-to-sport considerations**

In most instances, there are no contraindications to training with environmentally-induced NAR. However, as with AR, the lower airways should also be investigated to exclude lower airway dysfunction before an athlete with NAR continues with normal training.

## **8. EXERCISE-INDUCED LARYNGEAL OBSTRUCTION (EILO)**

### **8.1. Introduction and definition**

Exercise-induced laryngeal obstruction (EILO) is defined as a paradoxical inspiratory narrowing of the laryngeal structures during exercise in an otherwise normal larynx at rest. EILO can mimic lower airway dysfunction (asthma and/or EIB), may impair exercise performance(81) and is therefore an important cause of non-infective ARill in athletes. A detailed review of EILO in the athlete was specifically commissioned for this IOC Consensus statement and has been published.(82)

## **8.2. Epidemiology**

There are only a few studies reporting the prevalence of EILO in athletes. In most studies the diagnosis was not confirmed using the gold-standard diagnostic test of continuous laryngoscopy during exercise (CLE) testing.(83) In the general paediatric population, the prevalence of EILO is approximately 5-10%.(84, 85) In the most extensive study in high-level (Olympic) athletes, the point prevalence of EILO, based only on clinical assessments of stridor without CLE, was 5%.(86) This contrasts with data from one study where EILO was confirmed by CLE-testing in 24 out of 89 (27%) elite cross-country skiers, with only one athlete reporting stridor.(87)

## **8.3. Risk factors**

The precise causes and risk factors associated with EILO are still unknown. Several factors have been suggested as both causes or contributory factors, including: asthma, environmental conditions (exposure to humid or cold air), anatomic factors related to the upper airway in females, “high achiever” personality, and a genetic factor.(88) EILO seems to be more prevalent in athletes,(89, 90) and this has been attributed to interactions between the high airflows required in some sports and anatomical and/or psychological characteristics.(91-93) There is evidence that lower airway dysfunction (asthma, EIB) and EILO can co-exist in some individuals(90, 94) and that symptoms of EILO may worsen when exercising in cold and humid air(83) or other adverse environmental conditions.(86) Although gastroesophageal reflux disease might increase the risk of EILO, treating EILO patients with proton pump inhibitors has not proven to be effective in reducing symptoms.(95) An anatomical contributor to EILO may explain the observed female preponderance(84, 88, 90) in pubertal years and beyond, as females typically have smaller upper airways than males.(85) A genetic predisposition to EILO has been suggested,(88, 96) but to date, there is no definite proof of a hereditary component.

#### **8.4. Pathology and pathophysiology**

The larynx is a highly complex 'valve' that protects the lower airway from aspiration, permits vocalisation and abducts fully (opens) during exercise, to optimise airflow. Typically, a patient with EILO has a normal laryngeal structure and no symptoms at rest. In individuals with EILO the laryngeal structures adduct (close) during inspiration and reach maximum obstruction at the peak of intense exercise. Symptoms typically resolve rapidly (<5 min) on exercise cessation. When viewed from a physical airflow perspective, the Venturi effect arising from a narrowed structure results in higher negative pressure and induces obstruction in the larynx during exercise.

The anatomical area of obstruction in EILO can be either supraglottic or glottic (vocal cords), or a combination.(97) Supraglottic obstruction followed by glottic obstruction is common, while pure glottic obstruction is rare (i.e. seen in <5% of cases). A scoring system is widely used to grade the severity of EILO and ranges from no obstruction (complete patency=0), mild, moderate, or severe obstruction (almost complete closure=3). The grading is based on direct observation of the obstruction at moderate to maximum intensity of exercise.(98-100)

#### **8.5. Clinical presentation and diagnosis**

The diagnosis of EILO is based on a high index of suspicion when an athlete presents with symptoms during exercise that follow a specific time-course. The key elements of a typical presentation are wheeze, whistle/stridor when breathing in that: 1) are usually most evident during the inspiratory phase of the respiratory cycle, 2) are most prominent at peak exercise, when ventilation is at its greatest, and 3) typically resolve rapidly (within 1-5 min) after cessation of exercise unless there is co-existent (ongoing) panic or hyperventilation.(101)

This pattern is in contrast to EIB, where symptoms are related to expiratory limitation and are worst 3-15 min post-exercise. These differentiating features can distinguish suspected EILO from EIB in athletes (Figure 3). It is important that the SEM clinician makes the correct diagnosis to avoid treatment failure.(90, 94)

**[insert Figure 3 here]**

Athletes may find it difficult to attribute their exertional respiratory symptoms to a specific phase of the respiratory cycle. They may present with other exercise-related symptoms such as breathing that simply feels 'harder', throat tightness, choking sensations, upper chest tightness and chest pain, noisy breathing, stridor, hoarseness, voice changes, cough, and

panic reactions.(97) Typical symptoms of EILO however, may also occur in athletes with diseases such as asthma, structural airway abnormalities, cardiac diseases, vascular anomalies, tumours / cancer, primary hyperventilation / breathing pattern disorders, poor breathing techniques, low physical fitness, and psychological causes. Given the difficulties inherent in discerning the features of exercise-related phenomena in a clinical consultation at rest, it may be helpful to: 1) seek a collateral witness report describing the symptoms, 2) obtain a video recording of the athlete during a symptomatic period, or 3) directly assess an athlete when they are symptomatic (e.g. during an exercise test).

If the diagnosis of EILO is suspected, referral to a centre where CLE can be performed is recommended. The CLE test involves placement of a flexible laryngoscope to allow real-time visualisation of the larynx throughout a complete exercise session. Typically, moderate obstruction defined by a CLE-score  $\geq 2$  at the glottic or supraglottic level is considered pathological and is diagnostic for EILO.(98-100)

[Insert video link 2 here]

## **8.6. Management**

Treatment for EILO is non-surgical in the first instance and includes behavioural therapies (breathing advice, speech therapy, laryngeal control therapy),(102, 103) biofeedback,(104) and inspiratory muscle training (IMT).(105, 106) The aim of these strategies is to provide athletes with tools to better control their larynx during exercise, and enable them to continue exercising without experiencing EILO. The CLE test can be used as a biofeedback tool to guide athletes in using breathing control techniques during exercise. There is little scientific evidence on the efficacy of these treatment strategies, and most are based on anecdotal experiential evidence or single centre intervention studies. Surgery is an option, and the CLE test is also used to select those in need of surgery.(107-109) Surgery should be limited to athletes with moderate to severe obstruction caused by the supraglottic structures, with little or no effect from conservative treatments.

## **8.7. Prevention**

To date, there are no evidence-based preventative strategies for EILO. However, anecdotal experiential evidence indicates that if athletes employ effective breathing techniques and have knowledge of laryngeal function, they may have a lower risk of some types of EILO. This area requires further study.

## **8.8. Medical considerations and risks**

An important medical consideration is that EILO can be incorrectly diagnosed as lower airway dysfunction (asthma/EIB). This may lead to over-prescription of asthma medication with subsequent side effects and unnecessary doping implications.(49, 110) A more widespread understanding of the key clinical features that distinguish EILO from asthma/EIB will improve assessment of athletes with exercise-related breathing problems. There is a clinical impression from the author group (not based on scientific evidence) that athletes and young individuals who have a prolonged (years) history of EILO appear more difficult to treat than those who seek advice early (months).

## **8.9. Effect on exercise and sports performance**

EILO may lead to airflow limitation and excessive breathlessness, which are all contributory factors to impairment of exercise and sports performance.(81) Performance decrements can lead to loss of motivation and willingness to participate in competitive sports. Several studies from the general population indicate that EILO left untreated or undertreated can negatively affect a healthy lifestyle through the avoidance of exercise.(99, 100)

## **8.10 Return-to-sport considerations**

The general recommendation for return-to-sport (RTS) in an athlete with ongoing severe EILO causing distress +/- complications (e.g., syncope / dizziness) is to only exercise below or up to the threshold of intensity where symptoms of EILO develop. Once an athlete with EILO is treated and has a working strategy to control the larynx during exercise, RTS is recommended. In highly symptomatic athletes, a more cautious RTS protocol is recommended. Unsupervised or aquatic-based sports, with a risk of injury, are to be avoided until symptoms are better controlled.

# **9. LOWER AIRWAY DYSFUNCTION**

## **9.1. Introduction, definition and classification**

A variety of terms are used to describe clinical entities causing asthma-related issues in athletic individuals. Terms used include exercise-induced asthma (EIA), exercise-induced bronchoconstriction (EIB) and airway hyper-responsiveness (AHR). These terms are often

used interchangeably in real-life practice to describe a similar clinical entity. In this consensus document the term “lower airway dysfunction” (LAD) is used as an umbrella term to encompass the clinical entities causing asthma-related issues in athletes. Where indicated, definitive reference is made to a specific condition, e.g., studies specifically evaluating asthma, EIB or AHR.

## **9.2. Epidemiology of lower airway dysfunction in athletes**

The prevalence of LAD is greater in athletes than in the general population.(111) A recent systematic review and meta-analysis, specifically commissioned for this IOC consensus statement, re-evaluated the prevalence of LAD in athletes. In this review, studies employing different diagnostic approaches, revealed an overall prevalence of LAD in ~20% of athletes.(112) The highest prevalence rates were reported in elite-level endurance athletes and in those participating in aquatic and winter-based sports. A higher prevalence of LAD was also evident in studies utilising objective testing methodology (Figure 4).

**[Insert Figure 4 here]**

Key deficits in the available epidemiological data in this area include a lack of robust data from developing nations, detail on LAD in para-athletes, and insight regarding the incidence and impact of acute asthma-related events during training or competition in the athletic population.

## **9.3. Risk factors**

Several risk factors are associated with the development of LAD, reflecting both host and environmental influences. For example, as in the general population, an atopic disposition in athletes is associated with a 25- to 75-fold increased likelihood of EIB.(51) In the context of sport, the training environment in which exercise is undertaken is further important risk factor.(113, 114) For example, in the aquatic environment, exposure to high levels of trichloramines (i.e. a chlorine by-product) is associated with AHR (115-117). When endurance exercise is performed in cold air, as witnessed in elite cross-country skiers, there is a high prevalence of respiratory symptoms and AHR.(118-121) Other environmental risk factors include exposure to pollution e.g. emissions from fossil-fuelled ice resurfacing machines in sports arenas and air pollutants such as ozone and particulate matter.(122)

## **9.4. Pathology and pathophysiology**

Over the past forty years, there has been considerable progress in understanding the pathophysiological pathways leading the development of EIB in athletes.(123, 124) In this context, two questions are particularly pertinent; 1) how EIB develops acutely, and 2) why there is a heightened prevalence of LAD in endurance athletes. The development of airway dysfunction may be viewed as being akin to an ‘occupational lung disease’ in athletes.(125)

The key pathophysiological mechanism driving acute EIB is the release of local mediators in response to evaporative water loss from the airway surface. Specifically, hyperpnoea during exercise places stress on the airway surface to replenish evaporative water loss. In susceptible individuals, failure to replenish evaporative water loss at the airway surface leads to cellular changes and activation of sensory pathways that release local mediators, which promote bronchoconstriction.(126, 127) This process is amplified by the following factors: dry and cold air exposure, air pollution,(122) allergic asthma, and the extent of epithelial damage and inflammatory status of the airway at baseline.(127) A recent acute viral infection may also act to sensitize airway responses, with some reports indicating development of a post-viral AHR syndrome. Generally, this condition resolves spontaneously within 12 weeks.(128)

The reason elite-level endurance athletes appear to have a higher prevalence of lower dysfunction remains to be fully established. Repeated and prolonged episodes of hyperpnoea, specifically in endurance athletes, particularly when they exercise in potentially noxious environments, may drive airway structural, cellular and inflammatory changes, in a pattern similar to that witnessed pathologically following injury.(38, 123) Epithelial damage may facilitate sensitisation to circulating allergic and inflammatory mediators, cause sensory irritation and a local hyper-inflammatory response, thereby precipitating heightened AHR.(123) Increased parasympathetic nervous system may also be relevant in the development of AHR in endurance athletes.(129-131) Certainly, there are key differences in the phenotypic and inflammatory pattern of LAD in some groups of elite athletes, and this may be relevant for treatment but also for strategies to protect airway health.(132)

## **9.5. Clinical presentation and diagnosis**

There are two main clinical scenarios where a SEM clinician will need to follow an approach to make a diagnosis of LAD. In the first instance, there may be a need to screen asymptomatic athletes as part of a periodic health assessment, and in the second instance a



diagnosis is required for athletes that may present with symptoms suggestive of LAD. An approach to these two scenarios is presented in Figure 5.

**[Insert Figure 5 here]**

Symptoms suggestive of LAD, in athletes, typically include breathlessness, chest tightness, wheeze and cough during or after exercise.(132, 133) Other less distinct symptoms are “difficulty obtaining a satisfying breath” or “catching their breath”, particularly at the onset of vigorous exercise, and exercising in cold environments.

In athletes, there is a poor relationship between clinical features suggestive of LAD and proof of these diagnoses when objective testing is undertaken.(134-136) This is because conditions such as EILO or dysfunctional breathing may mimic or co-exist with asthma,(10, 84, 90) or respiratory symptoms may arise because of sensory irritation (e.g. as is seen in cough hypersensitivity).(137) A careful history of the clinical features can help discern EILO from EIB (Figure 3).(138)

The diagnosis of LAD in an athlete must be robust and supported by objective tests; a position supported by The Global Initiative for Asthma (GINA), prior IOC-Medical Commission statements(139) and the WADA.(140) Moreover, WADA recommend that a childhood diagnosis of asthma should be re-confirmed in adulthood.(49)

The key first line objective test in the assessment of an athlete with suspected LAD is spirometry at rest. If expiratory airflow limitation / obstruction is evident at rest (based on appropriate predicted normative values), then bronchodilator reversibility testing should be undertaken (e.g., following administration of a short acting beta-2 agonist [SABA]). This process may provide immediate evidence of reversible airflow obstruction, supporting a diagnosis of LAD. In most cases however, athletic individuals typically have normal resting spirometry values and thus some form of additional testing will be required.

A broad range of additional tests have been used in this context, including: 1) sport-specific / field-based exercise challenge tests (ECTs) with spirometry performed before and after exercise, and 2) other bronchoprovocation-based tests that can be performed and interpreted in line with international guidelines.(141) The optimal test to detect LAD in athletes remains debated and will most often be dictated by factors such as local availability and cost.

Generally, it is recommended that test replicates the process that precipitates symptoms during exercise, i.e., one that promotes relevant physiological changes at the airway surface. Accordingly, an approach that utilises either exercise or an alternative ‘indirect’ form of bronchoprovocation testing (e.g., eucapnic voluntary hyperpnoea [EVH] or mannitol provocation testing) is recommended in preference to direct bronchoprovocation tests (e.g. using methacholine or histamine for bronchoprovocation).

A recent systematic review, specifically commissioned for this IOC consensus statement, revealed the following: 1) a symptom-based approach / clinical diagnosis by a physician was sensitive but very non-specific for diagnosis of LAD in athletes, and 2) EVH, followed by a sport specific ECT, had the highest specificity when compared to other forms of provocation tests to confirm the diagnosis of LAD in athletes (*Reier-Nilsen et al., 2022, unpublished*). Therefore, in a resource-scarce setting where EVH testing is not an option, the alternative test is a sports-specific ECT that is conducted with appropriate consideration of the environmental conditions and type of activity to obtain precise results.(119, 142, 143) The sensitivity / specificity of ECT is enhanced if testing is: 1) performed in cold air,(51, 144) 2) at a very high exercise intensity (>85% of age predicted maximum heart rate) for 6-8 minutes,(142) and 3) in an environment and time of year when the athlete is symptomatic,(51) e.g. during a grass pollen season. Finally, repeat testing can increase sensitivity, specifically in athletes where there are initial negative or inconclusive test findings but there is an ongoing high suspicion of LAD (i.e. without features of a differential diagnosis such as EILO).(145)

A further key component in the modern-day assessment of an athlete with suspected LAD, is the detection and an assessment of airway inflammation.(146) This can be undertaken with measurement of fractional exhaled nitric oxide (FeNO), and interpretation of results can be guided by international guidelines (see GINA). In addition, a comprehensive assessment of LAD in athletes includes a full blood count to detect eosinophilia and tests for atopy (e.g. serum IgE and skin prick testing).

## **9.6 Management**

Both pharmacological and non-pharmacological strategies play a role in a successful holistic approach to the treatment of LAD in athletes. The overall goal of treatment is to ensure good

symptom control and minimise risk of acute exacerbations. In athletes, a key aim is to ensure that participation in sport is not hampered by respiratory symptoms. Different phenotypes of asthma in athletes have been described, and identification of biomarkers of inflammation (i.e. termed Type-2) are now considered relevant to guide management.(147) The evidence-based management of EIB was reviewed in 2013.(148)

There are several non-pharmacological strategies to help to manage LAD in athletes. The first strategy is to reduce exposure to cold and dry air, allergens, pollution, or other airway irritants. Other strategies include wearing a heat and moisture exchange face mask,(149) alterations to dietary intake with vitamin D,(150) omega-3 polyunsaturated fatty acids,(151-154) low-salt diet,(155, 156) and use of prebiotics.(157) An attenuation of EIB has also been reported after short high-intensity intervals and variable intensity pre-exercise warm-ups.(158)

The pharmacological approach to management of asthma is constantly evolving. In this consensus, we recommend SEM clinicians refer to [www.ginasthma.org](http://www.ginasthma.org) for latest updates and management advice. At the current time, there is no contemporary and robust evidence, to indicate that management of asthma in athletes should deviate from these international asthma guidelines.

The classical pharmacological approach of recommending a short-acting  $\beta$ 2-adrenoceptor agonists (SABA) alone, before sport for an individual with EIB, has been challenged by changes to international guidelines on the management of asthma. Specifically, as outlined in the GINA guidelines ([www.ginasthma.org](http://www.ginasthma.org)), it is now recommended that all patients with asthma, even with mild intermittent asthma, should use an inhaled corticosteroid (ICS) daily to prevent exacerbations and control symptoms. This recommendation represents an important change to the general treatment of asthma, and SEM clinicians should consider this updated guidance when caring for asthmatic athletes.

Some clinicians may choose to consider the use anti-cholinergic bronchodilators or leukotriene antagonists (e.g., montelukast), based on their expertise when managing a specific athletic population, and the environmental conditions in which sport is being undertaken. Approach to therapy should be modified by baseline lung function (i.e., if an athlete has spirometry at rest that indicates airflow obstruction) and the presence of

biomarkers of Type 2 (e.g., raised FeNO +/- blood eosinophilia) inflammation; i.e., highlighting a potentially steroid responsive process.

Treatment should be regularly reviewed with evaluation of adherence and inhaler technique. When taken correctly and with use of a spacer or volumetric device, it is not necessary to use nebulised preparations of inhaler therapy; this type of treatment should be restricted to management of severe or life-threatening asthma, when administered in an emergency scenario. It is also important that athletes with asthma are aware of how to manage an acute exacerbation, and for competitive athletes, there is a discussion regarding anti-doping regulations.(159) In this respect various web-based resources including [www.wada-ama.org](http://www.wada-ama.org), [www.globaldro.com](http://www.globaldro.com), international sports federations and national antidoping agencies are important sources of information to provide clarity regarding anti-doping regulations on asthma medication.

### **9.7. Prevention**

A key strategy to prevent LAD in athletes is to ensure that the air an athlete breathes is as clean as possible.(160) Environmental exposure factors such as cold air, pollution (carbon monoxide, nitrogen dioxide, ozone, and fine particulate matter), and allergens may contribute to development of LAD. It is recommended that sporting organisations and facilities should endeavour to improve air quality in the sporting arena, and where possible limit athletes' exposure to polluted areas. Airway health should be considered when sports authorities develop extreme weather protocols (e.g., if air temperature is very cold). It is also important to consider strategies to reduce allergen exposure in atopic individuals.(160) This may include advice on how to modify training plans (i.e. time of day and duration of session), according to the local environmental conditions. In scenarios where it is not possible to modify exposure to a noxious environment (e.g. polluted or in very cold air), it may be appropriate to consider use of a face mask or filter,(80) both at rest and during exercise, although it is acknowledged that this is impractical in many sporting scenarios. More work is needed to assess climatic conditions and the concentration of pollutants through various official monitoring sites or individual sensor systems, with the aim of helping inform clinicians involved with the care of athletes.

### **9.8. Medical considerations and risks**

In the general population, asthma is associated with recognised morbidity (e.g. exacerbations) and mortality.(146) However, data on medical complications in athletes with LAD is currently limited. Most athletes with LAD will only develop or report symptoms in the context of sporting participation, i.e., they do not generally report other classical complications of asthma, such as frequent exacerbations or night waking with symptoms. In one study the presence of asthma / EIB in athletes was associated with a 2-fold increased likelihood of more frequent ARinf in the preceding 18 months.(10) However other factors, respiratory issues, such as the presence of laryngeal dysfunction or breathing pattern irregularities, were also relevant predictors of risk of ARinf. With respect to asthma-related mortality, very few studies have evaluated asthma as a cause of sudden death in athletes or in the context of sport. A recent study found that sudden death due to asthma in athletes is uncommon when compared to cardiac causes, accounting for 0.8–4.9% of all causes.(161) A further potential medical consideration in athletes with asthma, relates to potential complications arising from treatment with  $\beta$ 2-adrenoceptor agonists (e.g. tachyarrhythmia).(162, 163)

### **9.9. Effect on exercise and sports performance**

LAD may impact breathing and gas exchange efficiency and limit performance secondary to the development of dyspnoea. Data from athletes competing at international level events suggest that asthmatic athletes are still capable of excelling in their chosen sport, and in some studies appear to outperform their competitors without asthma.(164) A systematic review of ten studies evaluating the impact of LAD on athletic performance measures could not draw any firm conclusions because of several methodological limitations within the included studies.(165) Further work is needed to study the impact of LAD in sport-specific settings and with environmental conditions likely to be provocative to the lower airways.(165) A diagnosis of LAD, when managed appropriately, should not be perceived as a negative consequence for athletic performance nor an athlete's aspirations. Finally, although often raised in discussion with athletes, the best available evidence to date indicates that inhaled asthma therapy, when taken at routinely prescribed doses, does not enhance endurance-based athletic performance.(166, 167)

### **9.10. Return-to-sport considerations**

There are currently no dedicated studies informing athletic RTS following an acute exacerbation of asthma. In the general population, it is recommended that vigorous exercise

is avoided completely in the period immediately following an acute exacerbation of asthma.(110) It would appear logical to extend this recommendation to athletes, i.e. an athlete requiring maximal treatment for an acute exacerbation of asthma (e.g. with oral glucocorticoid therapy) should therefore not partake in vigorous exercise. This recommendation is made to mitigate the risk of an acute serious adverse event; e.g. worsening respiratory tract infection, development of hypoxaemia +/- arrhythmias. Some sporting activities likely present a greater risk than others (e.g., high intensity exercise in a cold environment) and thus decision making regarding RTS should be guided by the identification of features prompting a need for a cautious approach to RTS. These include clinical features such as recent hospitalisation, ongoing respiratory symptoms during low intensity physical activity / activities of daily living, and ongoing significant need for bronchodilator medication (i.e., in excess of typical utilisation). The SEM clinician should also consider utilising both clinical and physiological assessment tools, to help assess and accurately appraise an athlete's recovery. It is recommended that specialist input and advice is sought for more complex scenarios.

## **10. OTHER CONDITIONS CAUSING NON-INFECTIVE ARill**

### **10.1. Differential diagnoses of acute breathing problems in athletes**

The SEM clinician should also consider the broader range of differential diagnoses when an athlete presents with acute breathing problems. These conditions include the following: cardiac-related diseases, acute pulmonary emboli, acute pneumothorax (acute dyspnoea with chest pain, often related to strenuous activity), altitude-related pulmonary oedema (acute dyspnoea and other features of altitude related illness)(168) and exercise-induced pulmonary oedema (dyspnoea and haemoptysis, in relation to endurance sports generally free-water swimming, often with wetsuits in cold conditions).(169, 170) These conditions should be assessed and managed appropriately. More recently, focus has been placed on the potential for respiratory symptoms that arise from excessive dynamic airway collapse (EDAC) or dysfunctional breathing in athletic individuals.

### **10.2. Excessive dynamic airway collapse (EDAC) and tracheobronchomalacia (TBM)**

Excessive dynamic airway collapse (EDAC) and tracheobronchomalacia (TBM) are conditions characterised by excessive inward movement of the trachea and/or main bronchi during the expiratory phase of the respiratory cycle.(171) The terms are often used

interchangeably, both defined as >50% collapse of the airway lumen.(172) Clinically, these conditions present with unexplained breathlessness, a barking or honking-type cough and difficulty clearing respiratory secretions with or without recurrent lower respiratory tract infections.

### **10.3 Dysfunctional breathing / breathing pattern disorders**

Dysfunctional breathing can be defined as an altered biomechanical pattern of breathing, leading to intrusive symptoms that can impact athletic performance and quality of life.(173, 174) Several terms have been used to describe this issue, including exercise-associated hyperventilation or a breathing pattern disorder. This condition can occur in the presence or absence of other comorbidities including EIB, EILO, and/or anxiety, and can overlap or amplify symptoms making them refractory to treatment.

Dysfunctional breathing is characterised by the following features: frequent sighing, apical / upper chest movement during respiration, asynchrony between upper and lower chest, excessive mouth breathing, and limited lateral movement of abdominal rib cage. A suspected diagnosis of dysfunctional breathing can be supported by affirmative responses to a questionnaire (e.g. Nijmegen questionnaire) or breathing pattern assessment tools that are used in the general population.(175) Ideally, a breathing pattern assessment should be conducted in a sport-specific context by an individual with expertise in the area. Assessment and subsequent treatments are usually undertaken by respiratory therapists. The focus is optimised breathing patterns but also to identify and address relevant psychophysiological and musculoskeletal factors, including core stability.

## **11. FUTURE DIRECTIONS**

In this IOC consensus statement, we provide an evidence-based overview of common and lesser-known causes of non-infective ARill in athletes. However, in this process, several unanswered questions were identified by the group translating into future research priorities (Table 2). The group encourages researchers and institutions to consider these as research priorities so that knowledge can be expanded and contribute to the protection of respiratory health in athletes.

**[Insert Table 2 here]**

**Table 2: Summary of unanswered questions and potential research priorities in non-infective acute respiratory illness in athletes**

	<b>Unmet need / unanswered questions</b>	<b>Future Research priorities</b>
<b>Nasal conditions in athletes (e.g. nasal obstruction, rhinitis / rhinosinusitis)</b>	<ul style="list-style-type: none"> <li>• Epidemiological data on structural nasal obstruction</li> <li>• Data on impact of nasal obstruction on training capability, resuscitation</li> <li>• Epidemiological data from developing countries and para-athlete populations.</li> <li>• Data on the impact of various severities of rhinitis/rhinosinusitis and anti-allergic treatment on training capability and return to play.</li> <li>• Impact of pollution on the nose and possible treatments.</li> </ul>	<ul style="list-style-type: none"> <li>• Effect on health and performance following correction of structural nasal obstruction</li> <li>• RCT on nasal obstruction treatment</li> <li>• More inclusive epidemiological studies to help characterise global burden of disease.</li> <li>• Characterization of the physiopathology of nasal symptoms during exercise in various type of sports.</li> <li>• The effects of immunotherapy on allergic rhinitis in athletes, comparatively to acute treatments</li> <li>• Various treatments or prevention measures efficacy to manage NAR according to the environment</li> </ul>
<b>Exercise-Induced Laryngeal Obstruction (EILO)</b>	<ul style="list-style-type: none"> <li>• Epidemiological data from developing countries and para-athlete populations.</li> <li>• Data on the impact of EILO on training capability.</li> <li>• Impact of cold, chlorine, pollution and thermal stress</li> <li>• Evidence based treatment guidelines</li> <li>• Pathophysiological studies on EILO – why is EILO more prevalent in athletes?</li> </ul>	<ul style="list-style-type: none"> <li>• RCT on EILO treatment</li> <li>• Impact from environmental factors on EILO</li> <li>• More available diagnostic options for EILO</li> <li>• More inclusive epidemiological studies on EILO to help characterise global burden of disease</li> <li>• Prevention and risk considerations in different types of sports</li> </ul>
<b>Lower Airway Dysfunction (LAD)</b>	<ul style="list-style-type: none"> <li>• Epidemiological data from developing countries and para-athlete populations.</li> <li>• Data on the impact of asthma exacerbations on training capability and return to play.</li> <li>• Impact of pollution and thermal stress on airway health in athletes.</li> <li>• Data on the effect of nebulized isotonic saline post strenuous exercise on pulmonary function, inflammation and on respiratory symptoms.</li> </ul>	<ul style="list-style-type: none"> <li>• More inclusive epidemiological studies to help characterise global burden of disease.</li> <li>• Establish prospective longitudinal monitoring systems to evaluate and ensure athlete respiratory health is protected from early in an athlete’s career.</li> <li>• Prospective evaluation of lower airway function, inflammatory biomarkers during exacerbations / evaluating the interplay with acute respiratory infections (ARinf).</li> <li>• Mechanistic studies to evaluate ‘airway injury’ and interaction with environmental conditions.</li> <li>• Placebo-controlled studies on nebulized isotonic saline.</li> </ul>

RCT: Randomised controlled trial



**Contributors:** All authors confirmed the final version to be published.

**Acknowledgements:** The core members of the IOC consensus group and authors of this manuscript would like to acknowledge the following subgroup corresponding members of the IOC consensus group on Acute Respiratory Illness in Athletes, who contributed to the generating and publishing review material that formed part of this consensus statement:

Marelise Badenhorst, Maaïke Eken, Josu Gomez-Eceiza, Jane Fitzpatrick, Maree Gleeson, Lovemore Kunorozva, Katja Mjosund, Margo Mountjoy, Christopher Carlsten, Beat Villiger, Oliver Price, Vibeke Backer, Bruno Chenuel, Kjell Larsson, Carolette Snyders, Kelly Kaulback, Tod Olin, Thomas Halvorsen, Harald Hrubos-Strom

**Funding:** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

**Competing interests:** None declared.

**Data sharing statement** There is no unpublished data.

## References

1. Rogge J. An ounce of prevention? *Br J Sports Med.* 2009;43(9):627-.
2. Dvorak J, Junge A, Derman W, et al. Injuries and illnesses of football players during the 2010 FIFA World Cup. *Br J Sports Med.* 2011;45(8):626-30.
3. Mountjoy M, Junge A, Alonso JM, et al. Sports injuries and illnesses in the 2009 FINA World Championships (Aquatics). *Br J Sports Med.* 2010;44(7):522-7.
4. Schwellnus M, Derman W, Page T, et al. Illness during the 2010 Super 14 Rugby Union tournament – a prospective study involving 22 676 player days. *Br J Sports Med.* 2012;46(7):499-504.
5. Engebretsen L, Steffen K, Alonso JM, et al. Sports injuries and illnesses during the Winter Olympic Games 2010. *Br J Sports Med.* 2010;44(11):772-80.
6. Soligard T, Steffen K, Palmer D, et al. Sports injury and illness incidence in the Rio de Janeiro 2016 Olympic summer games: a prospective study of 11274 athletes from 207 countries. *Br J Sports Med.* 2017;51(17):1265-71.
7. Derman W, Runciman P, Jordaan E, et al. Incidence rate and burden of illness at the Pyeongchang 2018 Paralympic Winter Games. *Br J Sports Med.* 2019;bjsports-2018-100096.
8. Engebretsen L, Soligard T, Steffen K, et al. Sports injuries and illnesses during the London Summer Olympic Games 2012. *Br J Sports Med.* 2013;47(7):407-14.
9. Schwellnus M, al..... e. IOC Consensus Statement on Acute Respiratory Illness in Athletes - Part 1: Acute respiratory infections *Br J Sports Med.* 2021;xx(xx).
10. Hull JH, Jackson AR, Ranson C, et al. The benefits of a systematic assessment of respiratory health in illness-susceptible athletes. *Euro Respir J.* 2021;57(6):2003722.
11. Fokkens WJ, Lund VJ, Hopkins C, et al. European position paper on rhinosinusitis and nasal polyps 2020. *Rhinology.* 2020;58:1-464.
12. Hens G, Hellings PW. The nose: gatekeeper and trigger of bronchial disease. *Rhinology.* 2006;44(3):179-87.
13. Mcintosh C, Clemm HH, Sewry N, et al. Diagnosis and management of nasal obstruction in the athlete: a narrative review by subgroup 6 of the IOC Consensus Group on "Acute Respiratory Illness in the Athlete". *J Sports Med Phys Fitness.* 2021.
14. Cannon CR, Cannon R, Young K, et al. Characteristics of nasal injuries incurred during sports activities: analysis of 91 patients. *Ear, Nose Throat J.* 2011;90(8):E8-E12.
15. Frenguelli A, Ruscito P, Bicciolo G, et al. Head and neck trauma in sporting activities: Review of 208 cases. *J Cranio-Maxillofacial Surg.* 1991;19(4):178-81.
16. Marston AP, O'Brien EK, Hamilton GS. Nasal injuries in sports. *Clinics Sports Med.* 2017;36(2):337-53.
17. Spielmann PM, White PS, Hussain SSM. Surgical techniques for the treatment of nasal valve collapse. *Laryngoscope.* 2009;119(7):1281-90.
18. Apaydin F. Nasal valve surgery. *Facial Plast Surg.* 2011;27(02):179-91.
19. Dinardi RR, de Andrade CR, Ibiapina CdC. External nasal dilators: definition, background, and current uses. *Int J Gen Med.* 2014;7:491-504.
20. Gelardi M, Porro G, Accettura D, et al. The role of an internal nasal dilator in athletes. *Acta Biomed.* 2019;90(2-S):28-30.
21. Kiyohara N, Badger C, Tjoa T, et al. A comparison of over-the-counter mechanical nasal dilators. *JAMA facial Plastic Surg.* 2016.
22. Fischer H, Gubisch W. Nasal valves-importance and surgical procedures. *Facial Plast Surg.* 2006;22(04):266-80.
23. Kasperbauer JL, Kern EB. Nasal valve physiology. Implications in nasal surgery. *Otolaryngol Clin North Am.* 1987;20(4):699-719.

24. Subcommittee AACoSCo, T., & International, A. w. g. Advanced trauma life support (ATLS®): the ninth edition. *J Trauma Acute Care Surg.* 2013;74(5):1363-6.
25. Lennon PF, Jaber SM, Fenton JEFF. Functional and psychological impact of nasal bone fractures sustained during sports activities: A survey of 87 patients. *Ear, Nose Throat J.* 2016;95(8):324-6,8,30,32.
26. Hox V, Beyaert S, Bullens D, et al. Tackling nasal symptoms in athletes: Moving towards personalized medicine. *Allergy.* 2021;76(9):2716-29.
27. Saketkhou K, Kaplan I, Sackner MA. Effect of exercise on nasal mucous velocity and nasal airflow resistance in normal subjects. *J Appl Physiol.* 1979;46(2):369-71.
28. Surda P, Walker A, Limpens J, et al. Nasal changes associated with exercise in athletes: systematic review. *J Laryngol Otol.* 2018;132(3):191-7.
29. Niinimaa V, Cole P, Mintz S, et al. The switching point from nasal to oronasal breathing. *Respir Physiol.* 1980;42(1):61-71.
30. Walker AC, Surda P, Rossiter M, et al. Nasal disease and quality of life in athletes. *J Laryngol Otol.* 2018;132(9):812-5.
31. Bousquet J, Van Cauwenberge P, Khaltaev N. Allergic rhinitis and its impact on asthma. *J Allerg Clin Immunol.* 2001;108(5):S147-S334.
32. Dykewicz MS, Wallace DV, Amrol DJ, et al. Rhinitis 2020: A practice parameter update. *J Allerg Clin Immunol.* 2020;146(4):721-67.
33. Surda P, Walker A, Putala M, et al. Prevalence of rhinitis in athletes: systematic review. *Int J Otolaryngol.* 2017;2017:8098426.
34. Teixeira RN, dos Santos Leite G, Gorjao R, et al. Immune and inflammatory response in atopic elite endurance athletes. *Int J Sports Med.* 2018;39(09):720-5.
35. Teixeira RN, Leite GdS, Bonini M, et al. Atopy in elite endurance athletes. *Clin J Sport Med.* 2018;28(3).
36. Gelardi M, Ventura MT, Fiorella R, et al. Allergic and non-allergic rhinitis in swimmers: clinical and cytological aspects. *Br J Sports Med.* 2012;46(1):54-8.
37. Surda P, Putala M, Siarnik P, et al. Rhinitis and its impact on quality of life in swimmers. *Allergy.* 2018;73(5):1022-31.
38. Kippelen P, Anderson SD. Airway injury during high-level exercise. *Br J Sports Med.* 2012;46(6):385-90.
39. Bonini S, Bonini M, Bousquet J, et al. Rhinitis and asthma in athletes: an ARIA document in collaboration with GA2LEN. *Allergy.* 2006;61(6):681-92.
40. Bernard A. Chlorination products: emerging links with allergic diseases. *Curr Medic Chemist.* 2007;14(16):1771-82.
41. D'amato G, Pawankar R, Vitale C, et al. Climate change and air pollution: effects on respiratory allergy. *Allergy, Asthma Immunol Res.* 2016;8(5):391-5.
42. Huff RD, Carlsten C, Hirota JA. An update on immunologic mechanisms in the respiratory mucosa in response to air pollutants. *J Allerg Clin Immunol.* 2019;143(6):1989-2001.
43. Brozek JL, Bousquet J, Agache I, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines: 2016 revision. *J Allergy Clin Immunol.* 2017.
44. Bonini M, Braidò F, Baiardini I, et al. AQUA: allergy questionnaire for athletes. Development and validation. *Med Sci Sports Exer.* 2009;41(5):1034-41.
45. Teixeira RN, Mendes FAR, Martins MA, et al. AQUA© as predictor of allergy in elite marathon runners. *World Allerg Organiz J.* 2014;7:7.
46. Chong LY, Head K, Hopkins C, et al. Saline irrigation for chronic rhinosinusitis. *The Cochrane Database Systematic Rev.* 2016(4).
47. Bousquet J, Schünemann HJ, Togias A, et al. Next-generation Allergic Rhinitis and Its Impact on Asthma (ARIA) guidelines for allergic rhinitis based on Grading of

- Recommendations Assessment, Development and Evaluation (GRADE) and real-world evidence. *J Allerg Clin Immunol*. 2020;145(1):70-80.e3.
48. Bousquet J, Schünemann HJ, Hellings PW, et al. MACVIA clinical decision algorithm in adolescents and adults with allergic rhinitis. *J Allerg Clin Immunol*. 2016;138(2):367-74.e2.
  49. WADA. International standard prohibited list 2021 2021 [Available from: [https://www.wada-ama.org/sites/default/files/resources/files/2021list\\_en.pdf](https://www.wada-ama.org/sites/default/files/resources/files/2021list_en.pdf)].
  50. Vinnikov D, Khafagy A, Blanc PD, et al. High-altitude alpine therapy and lung function in asthma: systematic review and meta-analysis. *ERJ Open Res*. 2016;2(2):00097-2015.
  51. Helenius IJ, Tikkanen HO, Haahtela T. Occurrence of exercise induced bronchospasm in elite runners: dependence on atopy and exposure to cold air and pollen. *Br J Sports Med*. 1998;32(2):125-9.
  52. Helenius IJ, Tikkanen HO, Sarna S, et al. Asthma and increased bronchial responsiveness in elite athletes: Atopy and sport event as risk factors. *J Allerg Clin Immunol*. 1998;101(5):646-52.
  53. Mokwena PL, Schwellnus MP, Van Rensburg AJ, et al. Chronic disease, allergies, and increased years of running are risk factors predicting gradual onset running-related injuries in ultramarathon runners—SAFER XIX study in 29 585 race entrants. *Clin J Sport Med*. 2021.
  54. Viljoen CT, Sewry N, Schwellnus MP, et al. Independent risk factors predicting gradual onset injury in 2824 trail running race entrants: SAFER XVIII study. *Wilderness Environ Med*. 2021.
  55. Doh H-W, Stebbins CL, Choi H-M, et al. Histamine H2 receptor blockade augments blood pressure responses to acute submaximal exercise in males. *Appl Physiol, Nutr, Metabol*. 2016;41(6):605-10.
  56. Ely MR, Romero SA, Sieck DC, et al. A single dose of histamine-receptor antagonists before downhill running alters markers of muscle damage and delayed-onset muscle soreness. *J Appl Physiol*. 2017;122(3):631-41.
  57. Katelaris CH, Carrozzi FM, Burke TV, et al. A springtime Olympics demands special consideration for allergic athletes. *J Allerg Clin Immunol*. 2000;106(2):260-6.
  58. Katelaris CH, Carrozzi FM, Burke TV, et al. Effects of intranasal Budesonide on symptoms, quality of life, and performance in elite athletes with allergic rhinoconjunctivitis. *Clin J Sport Med*. 2002;12(5):296-300.
  59. Salem L, Dao V-A, Shah-Hosseini K, et al. Impaired sports performance of athletes suffering from pollen-induced allergic rhinitis: a cross-sectional, observational survey in German athletes. *J Sports Med Phys Fitness*. 2019;59(4):686-92.
  60. Silvers WS, Poole JA. Exercise-induced rhinitis: a common disorder that adversely affects allergic and nonallergic athletes. *Annals Allergy, Asthma Immunol*. 2006;96(2):334-40.
  61. Dijkstra HP, Robson-Ansley P. The prevalence and current opinion of treatment of allergic rhinitis in elite athletes. *Curr Opin Allerg Clin Immunol*. 2011;11(2):103-8.
  62. Katelaris CH, Carrozzi FM, Burke TV. Allergic rhinoconjunctivitis in elite athletes. *Sports medicine (Auckland, NZ)*. 2003;33(6):401-6.
  63. Ely MR, Sieck DC, Mangum JE, et al. Histamine-receptor antagonists slow 10-km cycling performance in competitive cyclists. *Med Sci Sports Exer*. 2019;51(7):1487-97.
  64. Hellings PW, Klimek L, Cingi C, et al. Non-allergic rhinitis: Position paper of the European Academy of Allergy and Clinical Immunology. *Allergy*. 2017;72(11):1657-65.
  65. Bougault V, Turmel J, Levesque B, et al. The respiratory health of swimmers. *Sports Med*. 2009;39(4):295-312.

66. Salonen RO, Pennanen AS, Vahteristo M, et al. Health risk assessment of indoor air pollution in Finnish ice arenas. *Environ Int.* 2008;34(1):51-7.
67. Togias A, Naclerio RM. Cold air-induced rhinitis. *Clin Allerg Immunol.* 2007;19:267-81.
68. Alves A, Martins C, Delgado L, et al. Exercise-induced rhinitis in competitive swimmers. *Am J Rhinol Allerg.* 2010;24(5):e114-e7.
69. Bonadonna P, Senna G, Zanon P, et al. Cold-induced rhinitis in skiers—clinical aspects and treatment with Ipratropium Bromide nasal spray: a randomized controlled trial. *Am J Rhinol.* 2001;15(5):297-301.
70. Gomà A, de Lluís R, Roca-Ferrer J, et al. Respiratory, ocular and skin health in recreational and competitive swimmers: Beneficial effect of a new method to reduce chlorine oxidant derivatives. *Environ Res.* 2017;152:315-21.
71. Lévesque B, Duchesne J-F, Gingras S, et al. The determinants of prevalence of health complaints among young competitive swimmers. *Int Archives Occup Environ Health.* 2006;80(1):32-9.
72. Adams WC. Effects of Ozone exposure at ambient air pollution episode levels on exercise performance. *Sports Med.* 1987;4(6):395-424.
73. Meng Y, Wang C, Zhang L. Diagnosis and treatment of non-allergic rhinitis: focus on immunologic mechanisms. *Expert Rev Clin Immunol.* 2021;17(1):51-62.
74. Rundell KW, Smoliga JM, Bougault V. Exercise-induced bronchoconstriction and the air we breathe. *Immunol Allerg Clinics.* 2018;38(2):183-204.
75. Steelant B, Hox V, Van Gerven L, et al. Nasal symptoms, epithelial injury and neurogenic inflammation in elite swimmers. *Rhinology.* 2018;56(3):279-87.
76. Fokkens WJ. Thoughts on the pathophysiology of nonallergic rhinitis. *Curr Allerg Asthma Rep.* 2002;2(3):203-9.
77. Scadding GK, Kariyawasam HH, Scadding G, et al. BSACI guideline for the diagnosis and management of allergic and non-allergic rhinitis (Revised Edition 2017; First edition 2007). *Clin Experiment Allerg.* 2017;47(7):856-89.
78. Rundell KW, Caviston R, Hollenbach AM, et al. Vehicular air pollution, playgrounds, and youth athletic fields. *Inhalat Toxicol.* 2006;18(8):541-7.
79. Hanstock HG, Ainegren M, Stenfors N. Exercise in sub-zero temperatures and airway health: implications for athletes with special focus on heat-and-moisture-exchanging breathing devices. *Front Sports Act Liv.* 2020;2(34).
80. Pacitto A, Amato F, Salmatonidis A, et al. Effectiveness of commercial face masks to reduce personal PM exposure. *Sci Total Environ.* 2019;650:1582-90.
81. Welch JF, Archiza B, Sheel AW. Respiratory limitations to exercise. *Complete Guide to Respiratory Care in Athletes: Routledge; 2020.* p. 16-26.
82. Clemm HH, Olin JT, Mcintosh C, et al. Exercise-Induced Laryngeal Obstruction (EILO) in Athletes: A narrative review by a subgroup of the IOC Consensus on “Acute Respiratory Illness in the Athlete”. *Br J Sports Med.* 2022.
83. Halvorsen T, Walsted ES, Bucca C, et al. Inducible laryngeal obstruction: an official joint European Respiratory Society and European Laryngological Society statement. *Euro Respir J.* 2017;50(3):1602221.
84. Christensen PM, Thomsen SF, Rasmussen N, et al. Exercise-induced laryngeal obstructions: prevalence and symptoms in the general public. *Euro Arch Otorhinolaryngol.* 2011;268(9):1313.
85. Johansson H, Norlander K, Berglund L, et al. Prevalence of exercise-induced bronchoconstriction and exercise-induced laryngeal obstruction in a general adolescent population. *Thorax.* 2015;70(1):57-63.

86. Rundell KW, Spiering BA. Inspiratory stridor in elite athletes. *Chest*. 2003;123(2):468-74.
87. Irewall T, Bäcklund C, Nordang L, et al. High prevalence of exercise-induced laryngeal obstruction in a cohort of elite cross-country skiers. *Med Sci Sports Exer*. 2021;53(6):1134-41.
88. Walsted ES, Hvedstrup J, Eiberg H, et al. Heredity of supraglottic exercise-induced laryngeal obstruction. *Euro Respir J*. 2017;50(2):1700423.
89. Buchvald F, Phillipsen LD, Hjuler T, et al. Exercise-induced inspiratory symptoms in school children. *Pediatr Pulmonol*. 2016;51(11):1200-5.
90. Nielsen EW, Hull JH, Backer V. High prevalence of exercise-induced laryngeal obstruction in athletes. *Med Sci Sports Exer*. 2013;45(11):2030-5.
91. Landwehr LP, Wood RP, Blager FB, et al. Vocal cord dysfunction mimicking exercise-induced bronchospasm in adolescents. *Pediatrics*. 1996;98(5):971-4.
92. Olin JT, Westhoff E. Exercise-induced laryngeal obstruction and performance psychology: using the mind as a diagnostic and therapeutic target. *Immunol Allerg Clinics*. 2018;38(2):303-15.
93. McFadden J, Zawadski D. Vocal cord dysfunction masquerading as exercise-induced asthma. a physiologic cause for "choking" during athletic activities. *Am J Respir Crit Care Med*. 1996;153(3):942-7.
94. Low K, Lau KK, Holmes P, et al. Abnormal vocal cord function in difficult-to-treat asthma. *Am J Respir Crit Care Med*. 2011;184(1):50-6.
95. Woolnough K, Blakey J, Pargeter N, et al. Acid suppression does not reduce symptoms from vocal cord dysfunction, where gastro-laryngeal reflux is a known trigger. *Respirology*. 2013;18(3):553-4.
96. Hilland M, Røksund OD, Sandvik L, et al. Congenital laryngomalacia is related to exercise-induced laryngeal obstruction in adolescence. *Arch Dis Childhood*. 2016;101(5):443-8.
97. Røksund OD, Maat RC, Heimdal JH, et al. Exercise induced dyspnea in the young. Larynx as the bottleneck of the airways. *Respir Med*. 2009;103(12):1911-8.
98. Christensen P, Thomsen SF, Rasmussen N, et al. Exercise-induced laryngeal obstructions objectively assessed using EILOMEA. *Euro Arch Otorhinolaryngol*. 2010;267(3):401-7.
99. Maat RC, Røksund OD, Halvorsen T, et al. Audiovisual assessment of exercise-induced laryngeal obstruction: reliability and validity of observations. *Euro Arch Otorhinolaryngol*. 2009;266(12):1929-36.
100. Norlander K, Johansson H, Emtner M, et al. Differences in laryngeal movements during exercise in healthy and dyspnoeic adolescents. *Int J Pediatr Otorhinolaryngol*. 2020;129:109765.
101. Christopher KL, Morris MJ. Vocal cord dysfunction, paradoxical vocal fold motion, or laryngomalacia? Our understanding requires an interdisciplinary approach. *Otolaryngol Clinics N Am*. 2010;43(1):43-66.
102. De Guzman V, Ballif CL, Maurer R, et al. Validation of the dyspnea index in adolescents with exercise-induced paradoxical vocal fold motion. *JAMA Otolaryngol Head Neck Surg*. 2014;140(9):823-8.
103. Shaffer M, Litts JK, Nauman E, et al. Speech-language pathology as a primary treatment for exercise-induced laryngeal obstruction. *Immunol Allergy Clin North Am*. 2018;38(2):293-302.
104. Olin JT, Deardorff EH, Fan EM, et al. Therapeutic laryngoscopy during exercise: A novel non-surgical therapy for refractory EILO. *Pediatr Pulmonol*. 2017;52(6):813-9.

105. Clemm HSH, Sandnes A, Vollsæter M, et al. The heterogeneity of exercise-induced laryngeal obstruction. *Am J Respir Crit Care Med*. 2018;197(8):1068-9.
106. Sandnes A, Andersen T, Clemm HH, et al. Exercise-induced laryngeal obstruction in athletes treated with inspiratory muscle training. *BMJ Open Sport Exerc Med*. 2019;5(1):e000436.
107. Maat RC, Hilland M, Røksund OD, et al. Exercise-induced laryngeal obstruction: natural history and effect of surgical treatment. *Euro Arch Oto-rhino-laryngol*. 2011;268(10):1485-92.
108. Sandnes A, Hilland M, Vollsæter M, et al. Severe exercise-induced laryngeal obstruction treated with supraglottoplasty. *Front Surg*. 2019;6:44.
109. Siewers K, Backer V, Walsted ES. A systematic review of surgical treatment for supraglottic exercise-induced laryngeal obstruction. *Laryng Inv Otolaryngol*. 2019;4(2):227-33.
110. Hull JH, Pavord ID. Treating asthma exacerbations in athletes: TUE or not TUE? *Lancet Respir Med*. 2018;6(1):8-10.
111. Voy RO. The U.S. Olympic Committee experience with exercise-induced bronchospasm, 1984. *Med Sci Sports Exerc*. 1986;18(3):328-30.
112. Price OJ, Sewry N, Schwellnus M, et al. Prevalence of lower airway dysfunction in athletes: a systematic review and meta-analysis by subgroup of the IOC consensus group on 'acute respiratory illness in the athlete'. *Br J Sports Med*. 2021:bjsports-2021-104601.
113. Rundell KW, Jenkinson DM. Exercise-induced bronchospasm in the elite athlete. *Sports medicine (Auckland, NZ)*. 2002;32(9):583-600.
114. 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):1391-6.
115. Massin N, Bohadana AB, Wild P, et al. Respiratory symptoms and bronchial responsiveness in lifeguards exposed to nitrogen trichloride in indoor swimming pools. *Occupat Environ Med*. 1998;55(4):258-63.
116. Bougault V, Turmel J, Boulet L-P. Airway hyperresponsiveness in elite swimmers: Is it a transient phenomenon? *J Allerg Clin Immunol*. 2011;127(4):892-8.
117. Bougault V, Boulet L-P. Is there a potential link between indoor chlorinated pool environment and airway remodeling/inflammation in swimmers? *Expert Rev Respir Med*. 2012;6(5):469-71.
118. Heir T, Oseid S. Self-reported asthma and exercise-induced asthma symptoms in high-level competitive cross-country skiers. *Scand J Med Sci Sports*. 1994;4(2):128-33.
119. Stensrud T, Mykland KV, Gabrielsen K, et al. Bronchial hyperresponsiveness in skiers: field test versus methacholine provocation? *Med Sci Sports Exerc*. 2007;39(10):1681-6.
120. Mäki-Heikkilä R, Karjalainen J, Parkkari J, et al. Higher prevalence but later age at onset of asthma in cross-country skiers compared with general population. *Scand J Med Sci Sports*. 2021;31(12):2259-66.
121. 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):1963-81.
122. Rundell KW. Effect of air pollution on athlete health and performance. *Br J Sports Med*. 2012;46(6):407-12.
123. Anderson SD, Kippelen P. Airway injury as a mechanism for exercise-induced bronchoconstriction in elite athletes. *J Allerg Clin Immunol*. 2008;122(2):225-35.
124. Kippelen P, Anderson SD, Hallstrand TS. Mechanisms and biomarkers of exercise-induced bronchoconstriction. *Immunol Allerg Clinics*. 2018;38(2):165-82.
125. Price OJ, Ansley L, Menzies-Gow A, et al. Airway dysfunction in elite athletes – an occupational lung disease? *Allergy*. 2013;68(11):1343-52.

126. Daviskas E, Gonda I, Anderson SD. Local airway heat and water vapour losses. *Respir Physiol*. 1991;84(1):115-32.
127. Hallstrand TS, Moody MW, Wurfel MM, et al. Inflammatory Basis of Exercise-induced Bronchoconstriction. *Am J Respir Crit Care Med*. 2005;172(6):679-86.
128. Ostransky D, Blais FX. Postviral bronchial hyperreactivity syndrome: recognizing asthma's great mimic. *J Am Osteopath Assoc*. 1991;91(5):465-8, 71-5.
129. Filipe JAC, Falcao-Reis F, Castro-Correia J, et al. Assessment of autonomic function in high level athletes by pupillometry. *Autonom Neurosci*. 2003;104(1):66-72.
130. Knöpfli BH, Bar-Or O, Araújo CGS. Effect of ipratropium bromide on EIB in children depends on vagal activity. *Med Sci Sports Exer*. 2005;37(3):354-9.
131. Moreira A, Delgado L, Carlsen K-H. Exercise-induced asthma: why is it so frequent in Olympic athletes? *Expert Rev Respir Med*. 2011;5(1):1-3.
132. 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.
133. Hahtela T, Malmberg P, Moreira A. Mechanisms of asthma in Olympic athletes – practical implications. *Allergy*. 2008;63(6):685-94.
134. Simpson AJ, Romer LM, Kippelen P. Self-reported symptoms after induced and inhibited bronchoconstriction in athletes. *Med Sci Sports Exer*. 2015;47(10):2005-13.
135. Weiler JM, Hallstrand TS, Parsons JP, et al. Improving screening and diagnosis of exercise-induced bronchoconstriction: a call to action. *J Allerg Clin Immunol: In Prac*. 2014;2(3):275-80.e7.
136. Rundell KW, Im J, Mayers LB, et al. Self-reported symptoms and exercise-induced asthma in the elite athlete. *Medicine and science in sports and exercise*. 2001;33(2):208-13.
137. Ternesten-Hasséus E, Johansson E-L, Bende M, et al. Dyspnea from exercise in cold air is not always asthma. *J Asthma*. 2008;45(8):705-9.
138. Price OJ, Hull JH, Ansley L, et al. Exercise-induced bronchoconstriction in athletes – A qualitative assessment of symptom perception. *Respir Med*. 2016;120:36-43.
139. Fitch KD, Sue-Chu M, Anderson SD, et al. Asthma and the elite athlete: Summary of the International Olympic Committee's Consensus Conference, Lausanne, Switzerland, January 22-24, 2008. *J Allerg Clin Immunol*. 2008;122(2):254-60.e7.
140. Anderson SD, Argyros GJ, Magnussen H, et al. Provocation by eucapnic voluntary hyperpnoea to identify exercise induced bronchoconstriction. *Br J Sports Med*. 2001;35(5):344-7.
141. Hallstrand TS, Leuppi JD, Joos G, et al. ERS technical standard on bronchial challenge testing: pathophysiology and methodology of indirect airway challenge testing. *Euro Respir J*. 2018;52(5):1801033.
142. Carlsen KH, Engh G, MØRK M. Exercise-induced bronchoconstriction depends on exercise load. *Respir Med*. 2000;94(8):750-5.
143. Rundell KW, Anderson SD, Spiering BA, et al. Field exercise vs laboratory Eucapnic Voluntary Hyperventilation to identify airway hyperresponsiveness in elite cold weather athletes. *Chest*. 2004;125(3):909-15.
144. Kennedy MD, Steele AR, Parent EC, et al. Cold air exercise screening for exercise induced bronchoconstriction in cold weather athletes. *Respir Physiol Neurobiol*. 2019;269:103262.
145. Price OJ, Ansley L, Hull JH. Diagnosing exercise-induced bronchoconstriction with Eucapnic Voluntary Hyperpnea: is one test enough? *J Allerg Clin Immunol Pract*. 2015;3(2):243-9.
146. GINA. Global initiative for asthma 2020 [Available from: [www.ginasthma.org](http://www.ginasthma.org)].
147. Lund TK, Pedersen L, Anderson SD, et al. Are asthma-like symptoms in elite athletes associated with classical features of asthma? *Br J Sports Med*. 2009;43(14):1131-5.



148. Parsons JP, Hallstrand TS, Mastronarde JG, et al. An official American Thoracic Society clinical practice guideline: exercise-induced bronchoconstriction. *Am J Respir Crit Care Med.* 2013;187(9):1016-27.
149. Jackson AR, Hull JH, Hopker JG, et al. The impact of a heat and moisture exchange mask on respiratory symptoms and airway response to exercise in asthma. *ERJ Open Res.* 2020;6(2):00271-2019.
150. Price OJ, Hull JH, Howatson G, et al. Vitamin D and omega-3 polyunsaturated fatty acid supplementation in athletes with exercise-induced bronchoconstriction: a pilot study. *Exp Rev Respir Med.* 2015;9(3):369-78.
151. Kumar A, Mastana SS, Lindley MR. EPA/DHA dietary supplementation attenuates exercise-induced bronchoconstriction in physically active asthmatic males. *Cogent Med.* 2016;3(1):1172696.
152. Mickleborough TD, Lindley MR, Ionescu AA, et al. Protective effect of fish oil supplementation on exercise-induced bronchoconstriction in asthma. *Chest.* 2006;129(1):39-49.
153. Mickleborough TD, Murray RL, Ionescu AA, et al. Fish oil supplementation reduces severity of exercise-induced bronchoconstriction in elite athletes. *Am J Respir Crit Care Med.* 2003;168(10):1181-9.
154. Williams NC, Hunter KA, Shaw DE, et al. Comparable reductions in hyperpnoea-induced bronchoconstriction and markers of airway inflammation after supplementation with 6.2 and 3.1 g/d of long-chain n-3 PUFA in adults with asthma. *Br J Nutr.* 2017;117(10):1379-89.
155. Gotshall RW, Mickleborough TD, Cordain L. Dietary salt restriction improves pulmonary function in exercise-induced asthma. *Med Sci Sports Exer.* 2000;32(11):1815-9.
156. Mickleborough TD, Lindley MR, Ray S. Dietary salt, airway inflammation, and diffusion capacity in exercise-induced asthma. *Med Sci Sports Exer* 2005;37(6):904-14.
157. Williams NC, Johnson MA, Shaw DE, et al. A prebiotic galactooligosaccharide mixture reduces severity of hyperpnoea-induced bronchoconstriction and markers of airway inflammation. *Br J Nutr.* 2016;116(5):798-804.
158. Stickland MK, Rowe BH, Spooner CH, et al. Effect of warm-up exercise on exercise-induced bronchoconstriction. *Med Sci Sports Exer.* 2012;44(3):383-91.
159. Allen H, Backhouse SH, Hull JH, et al. Anti-doping policy, therapeutic use exemption and medication use in athletes with asthma: a narrative review and critical appraisal of current regulations. *Sports Med.* 2019;49(5):659-68.
160. Boulet L-P, O'Byrne PM. Asthma and exercise-induced bronchoconstriction in athletes. *New Eng J Med.* 2015;372(7):641-8.
161. Price OJ, Kucera KL, Price HM, et al. Asthma-related sudden death in athletes: A retrospective analysis of the U.S. NCCSIR database (1982–2018). *Euro Respir J.* 2021:2100088.
162. Abramson MJ, Walters J, Walters EH. Adverse effects of  $\beta$ -agonists. *Am J Respir Med.* 2003;2(4):287-97.
163. Warnier MJ, Rutten FH, Kors JA, et al. Cardiac arrhythmias in adult patients with asthma. *J Asthma.* 2012;49(9):942-6.
164. Fitch KD. An overview of asthma and airway hyper-responsiveness in Olympic athletes. *Br J Sports Med.* 2012;46(6):413-6.
165. Price OJ, Hull JH, Backer V, et al. The impact of exercise-induced bronchoconstriction on athletic performance: a systematic review. *Sports Med.* 2014;44(12):1749-61.

166. Riiser A, Stensrud T, Stang J, et al. Aerobic performance among healthy (non-asthmatic) adults using beta2-agonists: a systematic review and meta-analysis of randomised controlled trials. *Br J Sports Med*. 2021;55(17):975-83.
167. Pluim BM, de Hon O, Staal JB, et al.  $\beta_2$ -Agonists and physical performance: a systematic review and meta-analysis of randomized controlled trials. *Sports Med*. 2011;41(1):39-57.
168. Swenson ER, Bärtzsch P. High-altitude pulmonary edema. *Comprehen Physiol*. 2012;2(4):2753-73.
169. Spencer S, Dickinson J, Forbes L. Occurrence, risk factors, prognosis and prevention of swimming-induced pulmonary oedema: a systematic review. *Sports Med - Open*. 2018;4(1):43.
170. Zavorsky GS. Evidence of pulmonary oedema triggered by exercise in healthy humans and detected with various imaging techniques. *Acta physiologica (Oxford, England)*. 2007;189(4):305-17.
171. Mitropoulos A, Song W-J, Almaghlouth F, et al. Detection and diagnosis of large airway collapse: a systematic review. *ERJ Open Res*. 2021;7(3):00055-2021.
172. Kalra A, Abouzgheib W, Gajera M, et al. Excessive dynamic airway collapse for the internist: new nomenclature or different entity? *Postgrad Med J*. 2011;87(1029):482-6.
173. Barker N, Everard ML. Getting to grips with 'dysfunctional breathing'. *Paediatr Respir Rev*. 2015;16(1):53-61.
174. Boulding R, Stacey R, Niven R, et al. Dysfunctional breathing: a review of the literature and proposal for classification. *Euro Respir Rev*. 2016;25(141):287-94.
175. Todd S, Walsted ES, Grillo L, et al. Novel assessment tool to detect breathing pattern disorder in patients with refractory asthma. *Respirology*. 2018;23(3):284-90.

**Figure 1: A clinical diagnostic approach to the athlete presenting with symptoms and signs of nasal obstruction**

**Figure 2: Phenotypes, pathophysiology and treatment of non-allergic rhinitis [adapted from Hellings et al. 2017]**

**Figure 3: Key differences in the clinical presentation of Exercise Induced Laryngeal Obstruction (EILO) and Exercise Induced Bronchoconstriction (EIB)**

**Figure 4. Prevalence of lower airway dysfunction according to athletic standard and sporting discipline [adjusted from Price et al. 2021]**

**Figure 5. A clinical approach to the detection and evaluation of lower airway dysfunction (LAD) in athletes**

\*: AQUA: Allergy Questionnaire for Athletes [Bonini et al. 2009]

\*\* : AQUA screening uses 5 as the cut-off score for normal

\*\*\*: Objective testing is recommended to ensure diagnosis is robust, to be guided by local availability of tests; sport-specific exercise testing and/or another indirect bronchoprovocation testing protocols are recommended.

Definitions of abbreviations: IgE – Immunoglobulin E; EIB - Exercise-Induced Bronchoconstriction; LAD - Lower Airway Dysfunction