Prevalence of lower airway dysfunction in athletes: a systematic review and meta-analysis by a subgroup of the IOC consensus group on ‘acute respiratory illness in the athlete’

Oliver J Price,1,2 Nicola Sewry,3,4 Martin Schwellnus,3,4 Vibeke Backer,5 Tonje Reier-Nilsen,6 Valerie Bougault,7 Lars Pedersen,8 Bruno Chenuel,9,10 Kjell Larsson,11 James H Hull12,13

ABSTRACT

Objective To report the prevalence of lower airway dysfunction in athletes and highlight risk factors and susceptible groups.

Design Systematic review and meta-analysis.

Data sources PubMed, EBSCOhost and Web of Science (1 January 1990 to 31 July 2020).

Eligibility criteria Original full-text studies, including male or female athletes/physically active individuals/military personnel (aged 15–65 years) who had a prior asthma diagnosis and/or underwent screening for lower airway dysfunction via self-report (ie, patient recall or questionnaires) or objective testing (ie, direct or indirect bronchial provocation challenge).

Results In total, 1284 studies were identified. Of these, 64 studies (n=37,643 athletes) from over 21 countries (81.3% European and North America) were included. The prevalence of lower airway dysfunction was 21.8% (95% CI 18.8% to 25.0%) and has remained stable over the past 30 years. The highest prevalence was observed in elite endurance athletes at 25.1% (95% CI 20.0% to 30.5%) (Q=293, I²=91%), those participating in aquatic (39.9%) (95% CI 23.4% to 57.1%) and winter-based sports (29.5%) (95% CI 22.5% to 36.8%). In studies that employed objective testing, the highest prevalence was observed in studies using direct bronchial provocation (32.8%) (95% CI 19.3% to 47.2%). A high degree of heterogeneity was observed between studies (I²=98%).

Conclusion Lower airway dysfunction affects approximately one in five athletes, with the highest prevalence observed in those participating in elite endurance, aquatic and winter-based sporting disciplines. Further longitudinal, multicentre studies addressing causality (ie, training status/dose–response relationship) and evaluating preventative strategies to mitigate against the development of lower airway dysfunction remain an important priority for future research.

INTRODUCTION

The respiratory tract is frequently affected by acute and chronic illness in athletic individuals1 with disorders often classified by their involvement of the upper (ie, laryngeal region), large central (ie, trachea and main bronchi) and lower/small airways.2 It is now recognised that high-intensity exercise leads to a shift from nasal to predominantly oral breathing; thus ‘bypassing’ the upper airway (nasal and nasopharyngeal) structures and exposing the lower airways to significant physical, thermal and/or chemical stress.2 This can precipitate acute lower airway narrowing in susceptible individuals, leading to respiratory symptoms, such as cough, wheeze and dyspnoea.3

Historically, various clinical definitions have been used to describe this condition, including exercise-induced asthma (EIA), exercise-induced bronchoconstriction (EIB) and/or airway hyper-responsiveness (AHR). Irrespective of the terminology or definitions employed, research published over the past 50 years indicates that some form of ‘lower airway dysfunction’ is an important and relevant issue in both elite and recreational athletes.4 5

To date, the best available data concerning the prevalence and epidemiological characteristics of lower airway dysfunction in the athletic population primarily arise from cross-sectional studies in highly selected cohorts.6–9 Moreover, prior studies have used a diverse range of diagnostic approaches, including variable use of self-report and/or clinical or physician-based diagnosis and/or objective direct bronchial provocation (ie, histamine and methacholine) or indirect bronchial provocation (ie, laboratory and field-based exercise challenge tests, eucapnic voluntary hyperpnoea (EVH) or inhaled mannitol). While it is now widely recommended that some form of objective testing is used to secure a diagnosis,10 11 it is common for studies to provide evidence detailing the prevalence of lower airway dysfunction in elite athletes based on prior medication prescription data, often arising from submitted therapeutic use exemption (TUE) requests prior to major competition (eg, Olympic Games and World Championships).12 13 Overall, this broad range of methodological approaches makes it difficult to accurately quantify prevalence estimates and limits the ability to identify epidemiological risk factors.

This acknowledged, in recent years there have been a significant number of additional studies addressing this issue, published in both elite and recreational athletes. The primary aim of this systematic review and meta-analysis was therefore to provide contemporary insight into the prevalence of lower airway dysfunction in the athletic population and to characterise and describe findings based
on sex, test methodology, athletic standard, sporting discipline and geographic location. A secondary aim was to highlight relevant risk factors and susceptible groups and to evaluate temporal change over the study period (1990–2020).

**METHODOLOGY**

**Protocol and registration**

This systematic review was performed in accordance with the 2020 Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. The review was registered prospectively with the PROSPERO database (registration number: CRD42020167691). In September 2019, an IOC consensus statement core panel on ‘acute respiratory illness in athletes’ was convened on behalf of the IOC Medical and Scientific Commission and chaired by MS. A subgroup (number 4 out of 7) of this core panel, consisting of 10 members (OJP, NS, MS, VBa, TR-N, VBo, LP, BC, KL, JHH), focused on lower airway dysfunction and was chaired by JHH. The members of subgroup 4 conducted this systematic review and meta-analysis.

**Study selection and eligibility criteria**

The search strategy was developed by members of IOC subgroup 4. PubMed, EBSCOhost and Web of Science (core collection) databases were used to search for published articles between 1 January 1990 and 31 July 2020, in order to capture relevant contemporary literature concerning the diagnosis of lower airway dysfunction in athletes. A combination of search terms was used to identify studies focusing on the prevalence of lower airway dysfunction in athletes (eg, exercise-induced asthma (EIA) OR exercise-induced bronchoconstriction (EIB) OR exercise-induced bronchospasm OR asthma AND athlete OR active population) and relevant exclusions. For the full search string for each database, see online supplemental file 1. The results of these searches were combined, and duplicate articles removed. Any additional relevant articles identified by the authors or sourced from the reference list of identified studies were included. All article screening and selection was undertaken using the online tool CADIMA.

**Inclusion and exclusion criteria**

Studies were required to meet the following criteria for inclusion: (1) study participants were male or female athletes/physically active individuals/military personnel, aged 15–65 years; (2) participants had received a prior clinical or physician-based asthma diagnosis or underwent screening for lower airway dysfunction via self-report (ie, patient recall or questionnaires) or objective testing (ie, direct or indirect bronchial provocation challenge); (3) original full-text studies (ie, not research correspondence or case studies) of observational, prospective, retrospective, cross-sectional, longitudinal or intervention design, written in English. Animal or non-human studies were excluded. Articles were also excluded if the study was a review article, expert opinion or consensus position statement. The articles were screened independently by two reviewers (OJP, JHH) first by title/abstract and then full text, and any conflicts resolved through discussion or via a third researcher (NS).

**Data extraction**

The data extracted from the studies were clustered into three groups: (1) quality assessment of the studies (modified Downs and Black score, and Oxford Level of Evidence, 2009); (2) descriptive characteristics of the studies (study design, cohort number, sex, sport and level of participation); and (3) study outcome measures (diagnostic method, test outcome and the prevalence of lower airway dysfunction in the cohort). For interventional studies (clinical, nutrition, pharmacological), only data from the control group(s) were extracted. The geographical location of each study was also recorded. All data were extracted by two reviewers independently (OJP, JHH) and checked by a third researcher during analysis (NS) until consensus was reached.

**Quality assessment and risk of bias**

A modified Downs and Black checklist was used to determine the quality of the articles (see online supplemental file 2 for full checklist with relevant domains). Two reviewers (OJP, JHH) scored the articles independently and reached consensus on the final score after discussion. A third reviewer (NS) was consulted to resolve any inconsistencies. The Downs and Black checklist was adjusted to remove questions pertaining to a randomised controlled trial (RCT). The modified checklist included components of reporting, external and internal validity (bias and selection bias), and yielded a final score for each article out of a possible 13 points. The quality assessment score for each article was determined against the following criteria: 11–13: Excellent; 9–10: Good; 7–8: Fair; ≤6: Poor. The level of evidence was also determined using the 2009 Oxford Centre for Evidence-Based Medicine (OCEBM) Levels of Evidence. The OCEBM is a hierarchical system, grading studies on a scale of 1 (highest level of evidence) to 5 (lowest level of evidence), including subsections for levels 1, 2 and 3.

**Criteria and definitions of subcategories and outcome measures**

The primary categorisation of the entities of lower airway dysfunction (EIB, asthma and AHR) was performed to determine the overall prevalence. The following outcomes were included in further subgroup analysis, and if multiple different domains were reported in subgroups within a study, all were included in the analysis. The categories included: prevalence of lower airway dysfunction excluding studies rated as ‘poor’, decade of publication (1990–2000, 2001–2010, 2011–2020), diagnostic method (physician diagnosed, questionnaire only, bronchial provocation test, combination), type of provocation test (methacholine, histamine, exercise, EVH, inhaled mannitol), provocation test (direct, indirect), athletic standard (Olympic, elite or recreational), sex (male, females), sporting discipline (endurance, power; aquatic, non-aquatic; team, individual), season (summer, winter) and TUE studies. All studies that reported on these outcomes were included in the analysis. No direct contact was made with authors to determine if further data were available. If data were not differentiated for specific subgroups, it was not included in the analysis (ie, mixed data). For sex, the study had to include both sexes to be included in the analysis (ie, male-only studies were not included for this subanalysis).

**Data synthesis and analysis**

The pooled prevalence of lower airway dysfunction was determined by dividing the number of cases of disease observed by the total number of athletes and was estimated using a DerSimonian and Laird random effects model to account for the heterogeneity in the cohorts (eg, differences in diagnostic method and provocation test, etc) and weighting of studies. Heterogeneity was measured using $I^2$ and Cochran’s Q statistics. Pooled prevalence analysis was performed using MetaXL V5.3 (EpiGear International). Data are reported as prevalence (%) and 95% CI. The latter were compared to determine significant differences.
between subgroups for prevalence data (except sex) (ie, 95% CIs were considered significantly different if they did not overlap). For the comparison of sex, these data were extracted from within studies and analysed using a DerSimonian and Laird binary random effects model using OpenMeta-Analyst (Metafor) to determine the OR (95% CIs) of males having lower airway dysfunction in comparison to females (p<0.05 was considered statistically significant). Publication bias statistics, funnel and digital object identifier (DOI) plots are presented in online supplemental file 3.

RESULTS
Included studies and quality characteristics
In total, 1284 studies were identified. Of these, 64 studies,6–9 12 13 18–75 from over 21 countries were considered eligible for inclusion in the qualitative synthesis and meta-analysis (none of the studies included in this review were RCTs) (figures 1 and 2). Study characteristics and sample sizes, according to subgroups, are summarised in table 1 and online supplemental table S1. The Oxford Level of Evidence ranged from 1b (n=51) to 2b (n=13) and included both prospective (n=56) and retrospective (n=8) studies. Downs and Black Quality Assessment Scores ranged from 2 to 12 and studies were rated as poor (n=10); fair (n=9); good (n=21); and excellent (n=24) (online supplemental table S2 and online supplemental file 3).

Overall prevalence of lower airway dysfunction
The 64 eligible studies resulted in a combined study population of n=37 643: elite (n=7898) (21.0%); recreational (n=12 767) (33.9%); Olympic (n=16 978) (45.1%) athletes. Detail regarding athlete sex was available in 52 studies (n=16 474 athletes) (43.8% of total athletes included in the population) but revealed a slightly greater proportion of male athletes (62.3%). The overall mean prevalence of lower airway dysfunction (ie, those with confirmed EIB and/or asthma and/or AHR) for all studies was 21.8% (95% CI 18.8% to 25.0%) (Q=2711, I²=98%) (online supplemental figure S1). This remained unchanged when (n=10) ‘poor’ studies were excluded from the analysis (23.0%; 95% CI 19.1% to 27.1%) (Q=2254, I²=98) (online supplemental figure S2). The prevalence remained similar over the study period: 1990–2000 (23.5%; 95% CI 16.4% to 31.1%) (Q=169, I²=94%); 2001–2010 (21.6%; 95% CI 16.9% to 26.6%) (Q=1564, I²=98%); 2011–2020 (21.0%; 95% CI 17.2% to 25.0%) (Q=305, I²=93%) (figure 3). A high degree of heterogeneity was however observed between studies (Q=2711, I²=98%). When stratified according to the original terminology reported in the respective published paper, the highest prevalence was observed in AHR: 38.2% (95% CI 26.9% to 49.8%) (Q=92, I²=89%), followed by EIB: 21.0% (95% CI 15.4% to 27.0%) (Q=1201, I²=97%) and asthma: 17.8% (95% CI 14.6% to 21.2%) (Q=831, I²=97%) (online supplemental figure S3).
In the 23 studies that compared sex, the prevalence of lower airway dysfunction was significantly higher in females (15.5%) in comparison to males (11.5%) (OR 0.75; 95% CI 0.62 to 0.91) (Q=46, I²=52%) (p=0.003).

### Prevalence analysis based on diagnostic methodology

Thirty-six studies (56.3%) included at least one form of objective test methodology (ie, bronchial provocation test), 12 studies relied on a physician-based diagnosis (18.8%), 9 studies used questionnaires (14.1%) and 9 employed combined methods (14.1%) (two studies employing combined methods also included a bronchial provocation test). The prevalence of lower airway dysfunction was highest when using combined methods: 25.8% (95% CI 16.8% to 35.3%) (Q=365, I²=98%), followed by objective testing: 23.2% (95% CI 19.1% to 27.5%) (Q=606, I²=92%), physician diagnosed: 16.8% (95% CI 14.7% to 19.1%) (Q=91, I²=84%) and questionnaires: 14.9% (95% CI 6.9% to 24.1%) (Q=470, I²=98%) (figure 4). Three studies reported the prevalence of lower airway dysfunction based on TUE application data for Olympic competition (n=13869 athletes), revealing an overall prevalence estimate of 8.0% (95% CI 3.6% to 13.8%) (Q=136, I²=99%).

In studies that employed bronchial provocation testing, a higher prevalence was observed in studies using direct test (ie, histamine or methacholine challenge): 32.8% (95% CI 19.3% to 47.2%) (Q=99, I²=93%) in comparison to indirect test methodology (ie, exercise, EVH, inhaled mannitol): 22.3% (95% CI 17.9% to 26.9%) (Q=472, I²=92%) (online supplemental figure S4). Of the indirect tests, the highest prevalence of lower airway dysfunction was observed in response to EVH: 29.2% (95% CI 21.3% to 37.6%) (Q=215, I²=93%), followed by inhaled mannitol: 25.0% (95% CI 0.0% to 59.9%) (Q=34, I²=94%) and exercise: 16.8% (95% CI 12.0% to 22.0%) (Q=186, I²=89%) (online supplemental figure S5).

### Prevalence analysis based on sporting discipline and athletic standard

The prevalence of lower airway dysfunction, classified according to sporting discipline and athletic standard, is summarised in figure 5. A higher prevalence of lower airway dysfunction was observed in athletic groups partaking in individual sports: 27.5% (95% CI 21.7% to 33.5%) (Q=204, I²=91%) when compared with team sports: 17.3% (95% CI 9.6% to 26.0%) (Q=88, I²=91%) (online supplemental figure S6).

The highest prevalence of lower airway dysfunction was observed in athletes participating in aquatic disciplines (39.9%) (95% CI 23.4% to 57.1%) (Q=128, I²=96%) (online supplemental figure S7) and winter-based sports (29.5%) (95% CI 22.5% to 36.8%) (Q=453, I²=97%) (online supplemental figure S8). Likewise, when a single study of low numbers (<20) was excluded, the prevalence of lower airway dysfunction was higher in endurance athletes at 25.1% (95% CI 20.0% to 30.5%) (Q=293, I²=91%) in comparison to those partaking in power-based sports (18.7%) (95% CI 11.8% to 26.3%) (Q=13, I²=69%) (online supplemental figure S9). The prevalence also varied according to athletic standard with lower airway dysfunction most commonly reported in elite-level athletes of 28.2% (95% CI 22.4% to 34.3%) (Q=1032, I²=97%) (online supplemental figure S10). A high degree of publication bias was observed for the overall analysis when evaluating the DOI plots (and asymmetrical funnel plots); however, this decreased when accounting for the specific form of bronchial provocation test (ie, the only subgroup analysis where no asymmetry was observed) (online supplemental file 4).

### DISCUSSION

It has long been reported that lower airway dysfunction is the most common chronic medical condition encountered in elite and recreational athletes. In this comprehensive systematic review and meta-analysis that included data from over 37 000 athletes...
Table 1  Summary of key study variables, athlete characteristics and prevalence statistics

| Athlete breakdown (%) | Lower airway dysfunction (%) |
|-----------------------|-----------------------------|
| **Standard**          |                             |
| Elite                 | 21.0                        | 28.2                        |
| Recreational          | 33.9                        | 16.7                        |
| Olympian              | 45.1                        | 16.1                        |
| **Sex**               |                             |
| Male                  | 60.9                        | 11.5                        |
| Female                | 39.1                        | 15.5                        |
| **Diagnostic method** |                             |
| Bronchial provocation | 13.7                        | 23.2                        |
| Physician diagnosed   | 38.8                        | 16.8                        |
| Questionnaire         | 12.3                        | 14.9                        |
| Combined              | 35.2                        | 25.8                        |
| **Type of bronchial provocation** |             |
| Direct challenge      | 14.2                        | 32.8                        |
| Indirect challenge    | 85.8                        | 22.3                        |
| **Direct challenge**  |                             |
| Methacholine          | 64.0                        | 32.8                        |
| Histamine             | 36.0                        | 25.0                        |
| **Indirect challenge**|                             |
| Exercise              | 54.9                        | 16.8                        |
| EVH                   | 41.8                        | 29.2                        |
| Inhaled mannitol      | 3.3                         | 25.0                        |
| **Sporting discipline**|                            |
| Endurance             | 96.8                        | 25.1                        |
| Power                 | 3.2                         | 18.7                        |
| Aquatic               | 5.5                         | 39.9                        |
| Non-aquatic           | 94.5                        | 20.7                        |
| Team                  | 7.7                         | 17.3                        |
| Individual            | 92.3                        | 27.5                        |
| **Season**            |                             |
| Summer                | 70.1                        | 19.6                        |
| Winter                | 29.9                        | 29.5                        |
| **Studies (n)**       |                             |
| **Studies (%)**       |                             |
| Europe                | 36                          | 56.3                        |
| North America         | 16                          | 25.0                        |
| South America         | 1                           | 1.6                         |
| Africa                | 2                           | 3.1                         |
| Asia                  | 3                           | 4.7                         |
| Australasia           | 2                           | 3.1                         |
| Global                | 4                           | 6.3                         |

*Percentage breakdown presented according to available data reported in studies. EVH, eucapnic voluntary hyperpnoea.

individuals and from over 21 countries, we confirm that approximately one in five athletes are affected by lower airway dysfunction (including asthma and/or EIB and/or AHR). In keeping with asthma prevalence estimates from the general population in developed countries,177 the high prevalence of lower airway dysfunction in athletes has remained relatively stable over the past 30 years and appears particularly common in elite endurance athletes and those partaking in aquatic and winter-based sporting disciplines.

The evaluation of the epidemiology or prevalence of a clinical condition depends on several key factors including definitions, diagnostic methodology, test protocols and cut-off criteria employed.87 In this respect, it is apparent that a broad range of diagnostic approaches have been used to assess the prevalence of lower airway dysfunction in the athletic population over the past three decades. It is widely recommended that a form of objective testing should be conducted to secure a diagnosis of lower airway dysfunction in athletes.10 11 76 However, this approach was only employed in approximately half of the studies (n=36) included in this review, with a large number (n=28) relying solely on either physician diagnosis and/or symptom-based questionnaires.

Our data indicate that the choice of diagnostic test significantly impacts prevalence estimates. For example, in studies that employed a form of direct bronchial provocation testing (ie, methacholine or histamine), approximately one in three athletes were found to have evidence of lower airway dysfunction. In contrast, in studies that used indirect bronchial provocation, the prevalence was closer to one in five. Likewise, in studies that reported a physician or symptom-based approach to diagnosis, the prevalence was closer to one in six. Importantly, even when comparing objective methods, test selection appears to influence the reported prevalence. Specifically, studies that used exercise testing (ie, often considered the most intuitive approach to detect lower airway dysfunction in athletes)10 11 actually resulted in the lowest prevalence (16.8%). While this may appear counterintuitive, exercise testing is recognised to be highly specific (ie, ability to rule in) but less sensitive (ie, ability to rule out), given the type, duration and intensity of exercise and the temperature and water content of inspired air are recognised to be important determinants of the airway response.79 80 For that reason, indirect ‘surrogate’ tests such as EVH and inhaled mannitol are often recommended in an attempt to improve diagnostic sensitivity and specificity when screening athletes.33 81

It is important to note that, from an epidemiological perspective, it does not appear to be appropriate to use objective methods interchangeably, given a higher prevalence of lower airway dysfunction was observed when using surrogate tests (EVH: 29.2%; inhaled mannitol: 25.0%) in comparison to exercise. This observation is in keeping with studies conducted over the past two decades that consistently reveal poor diagnostic agreement when directly comparing exercise, EVH and/or inhaled mannitol.27 33 82–84 Accordingly, while surrogate tests may reduce the risk of underdiagnosis (ie, false-negative outcome), there remains concern regarding the potential for overdiagnosis (ie, false-positive outcome).85 Indeed, recent studies have questioned the suitability of current diagnostic thresholds when using surrogate tests in this setting. Specifically, Price et al86 have previously observed a greater reduction in lung function post-EVH in “healthy” (defined as entirely asymptomatic, with no history of asthma or inhaler medication use) elite versus recreational athletes, indicating that the ‘normative’ airway response may differ according to the athletic population tested. This also presents challenges with respect to the most appropriate diagnostic ‘cut-off’ value according to the form of bronchial provocation challenge employed.85 86

Historically, many studies that report the prevalence of lower airway dysfunction in Olympic athletes have arisen from mandatory evidence of inhaled beta-2 agonist use when TUE certificates were required for this type of medication (for review see Allen et al87). Indeed, large retrospective studies in this area reveal a consistent prevalence of lower airway dysfunction (ie, asthma medication use) of approximately 8% in Olympic-level athletes over sequential major competitions (1996–2004).12 13 The reason this figure is lower than the overall prevalence in
Figure 3  Prevalence of lower airway dysfunction in athletes between 1990 and 2020.
Figure 4  Prevalence of lower airway dysfunction according to diagnostic method.

Price OJ, et al. Br J Sports Med 2022;56:213–222. doi:10.1136/bjsports-2021-104601
our analysis is unclear, but certainly challenges the widely held supposition that many athletes report ‘asthma symptoms’ or use inhalers to potentially enhance performance87–89; that is, studies objectively confirming lower airway dysfunction actually suggest that a greater number of athletes should be using inhaler therapy to optimise and maintain their respiratory health.

A secondary aim of this meta-analysis was to evaluate ‘risk’ factors and highlight susceptible groups. To achieve this objective, we subclassified athlete populations according to sporting discipline and athletic standard. It has long been recognised that endurance sport is associated with the highest prevalence of AHR and our systematic review substantiates this, with a higher incidence observed in endurance (25.1%) versus power athletes (18.7%), aquatic (39.9%) versus non-aquatic disciplines (20.7%) and winter (29.5%) versus summer sports (19.6%). The pathophysiological mechanism(s) underpinning this remains to be fully determined; however, it has been proposed that high-intensity repeated periods of hyperpnoea particularly when conducted in noxious environments (eg, high aeroallergen, exposure to chlorine derivatives, cold dry air, particulate matter, etc) may act to sensitise or potentially damage the small airways, akin to an airway injury, thus driving a predisposition to AHR.5 90 91 This theory is supported by the finding that discontinuing exercise is associated with a fairly rapid resolution of heightened AHR on discontinuing vigorous training.92 In support of this concept, Helenius et al93 previously observed a reduction in airway inflammation and attenuation or disappearance of AHR in elite swimmers who stopped high-level training. The fact that environmental exposure appears to be relevant in terms of aetiology, yet the prevalence has remained unchanged over time, supports a need for closer scrutiny regarding this issue and development of effective preventative strategies moving forward.

Ideally, it would have been informative to assess biological risk factors in our analysis, such as the impact of allergenic profiling. Indeed, prior studies have shown that AHR and asthma are strongly associated with atopic disposition in athletes.30 31 However, our ability to analyse this type of data was limited on the basis that few studies completed skin prick testing and/or included specific statistics with respect to subgroup prevalence. Accordingly, logistic regression or ORs are often not reported and therefore this does not permit extraction of true prevalence data which limited our ability to analyse atopic versus non-atopic athletes. Similarly, differentiating between type 2 and non-type 2 ‘asthma’ was not possible in this analysis. Likewise, there are also limited data available regarding relevant biomarkers of airway inflammation such as fractional exhaled nitric oxide.

It is also important to highlight that very few studies have reported the prevalence of acute lower airway dysfunction (‘asthmatic’ events) and thus we did not systematically evaluate the literature to address this issue. It is recognised that acute respiratory illness is highly prevalent in athletes and the most common reason an athlete seeks acute medical attention during major competition.94 It is likely that a proportion of these acute events are exacerbations of lower airway dysfunction, and a 4-year prospective study found an incidence of 0.18 per 1000 athletes required treatment for acute asthma.98

**Figure 5** Prevalence of lower airway dysfunction according to athletic standard and sporting discipline. TUE, therapeutic use exemption.
What is already known

- Lower airway dysfunction (including asthma and/or exercise-induced bronchoconstriction and/or airway hyper-responsiveness) is often cited as the most common chronic medical condition in athletes.
- The reported prevalence data in athletes typically arise from cross-sectional studies in highly selected cohorts.
- A contemporary systematic appraisal of evidence is required to provide insight regarding the prevalence of lower airway dysfunction in athletes, associated risk factors and temporal change over the past 30 years.

What are the new findings

- Lower airway dysfunction affects approximately one in five athletes across a broad range of sporting disciplines and abilities, with highest prevalence rates observed in those participating in elite endurance, aquatic and winter-based sporting disciplines.
- The prevalence of lower airway dysfunction in athletes has not changed significantly over the past 30 years.
- The majority of evidence arises from European countries or North America, with a paucity of evidence arising from other geographical areas, including developing nations.

Methodological limitations and future research

Several methodological limitations are worthy of consideration. First, we recognise that the nomenclature pertaining to ‘asthma in athletes’ remains debated, and thus we opted to use the term ‘lower airway dysfunction’ to encompass and capture all relevant prevalence-based studies in athletes. Furthermore, as with any epidemiological evaluation, a true prevalence estimate is dependent on appropriate and robust capture of the population of interest. It is important that, as close as possible, the whole population is included to provide an accurate denominator (ie, asymptomatic and symptomatic athletes). Despite our best efforts to exclude studies with potential biased inclusion criteria, it seems likely that in most of the studies there is inadequate capture of the whole study population. Specifically, the nature of any study with a self-report or questionnaire-based approach response will be associated with a degree of self-selection bias; that is, it is likely that symptomatic individuals may be preferentially included thus potentially artificially increasing prevalence rates in some studies.

The wide range of diagnostic methods employed over the past 30 years resulted in a high level of heterogeneity between studies included in this review ($I^2=98\%$) (even when accounting for subgroups analysis). The publication bias was also high (major asymmetry in all analyses except for the specific form of bronchial provocation test subgroup analysis), and therefore all prevalence estimates should be interpreted with caution. Furthermore, the risk of bias failed to account for the observed heterogeneity (ie, when analysing the data excluding ‘poor’ studies, the asymmetry was still present, and the prevalence was not significantly different).

It is also important to acknowledge that while some groups have previously reported good test–retest repeatability following objective testing in athletes, others have highlighted that a single bronchial provocation challenge (ie, exercise and EVH) has the potential to result in misdiagnosis (particularly in athletes with mild severity disease or a borderline diagnosis). In this respect, none of the included studies performed multiple assessments (ie, in/out of season testing) in the same athlete to confirm or refute a diagnosis. While repeat assessment is not a current requirement, it is important to note that airway calibre fluctuates over short-term periods and thus any change in training status or environmental exposure (eg, seasonal variation due to high allergen exposure) has the potential to impact test outcome.

In addition, the lack of longitudinal studies limits the ability to draw robust conclusions concerning the development of lower airway dysfunction (ie, training status/dose–response relationship) over the course of a sporting career. Also, a key deficiency in the field is the paucity of data with respect to racial differences in prevalence figures and individuals participating in Paralympic sport which remains an important avenue for future research.

A further consideration when evaluating the epidemiology of a condition is the availability of resources to screen athletes (ie, access to diagnostic tests), to ensure that best practice is upheld to rule in/rule out a diagnosis (ie, adhering to established test protocols in accordance with international guidelines). Despite the large number of athletes in our analysis, the majority of available data were sourced from European countries (n=36 studies) or North America (n=16 studies), with a paucity of evidence arising from other geographical areas, including developing nations. The reason(s) for this remain to be fully established and thus further epidemiological research is required moving forward to provide a globally inclusive prevalence estimate of lower airway dysfunction in athletes.

Finally, despite conducting a robust and comprehensive search strategy, there is vast literature on this topic, and thus it is possible that some studies may not have been identified in the initial search. Irrespective of this potential limitation, the current analysis included a combined study population of over 37 000 athletes and thus we feel that this analysis provides a reliable representation of the current epidemiological characteristics of the condition within this population.

Clinical implications and practical application

The clinical implications and practical application of our findings can be considered twofold. First, improved epidemiological insight enables sport and exercise medicine clinicians and support personnel to conduct targeted screening and assessment in high-risk athletic cohorts (eg, elite endurance, aquatic and winter-based sports) moving forward. Second, the ability to identify susceptible groups provides the opportunity to conduct focused longitudinal research to establish the underlying pathophysiological mechanism(s) associated with disease onset and progression and to evaluate the efficacy of preventative strategies to protect and maintain airway health.

CONCLUSION

In summary, lower airway dysfunction occurs in approximately one in five athletes, with a higher prevalence in those participating in elite endurance, winter and aquatic disciplines. This estimate appears to be unchanged over the past three decades, with studies consistently revealing that objective testing results in a higher incidence in comparison to a physician or symptom-based approach. Further longitudinal, multicentre studies addressing causality (ie, training status/dose–response relationship) and evaluating preventative strategies to mitigate against the development of lower airway dysfunction remain an important priority for future research.
Author affiliations

1School of Biomedical Sciences, Faculty of Biological Sciences, University of Leeds, Leeds, UK
2Leeds Institute of Medical Research at St. James’s, University of Leeds, Leeds, UK
3Sport, Exercise Medicine and Lifestyle Institute (SEMLI), Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa
4IOC Research Centre, Pretoria, South Africa
5Centre for Physical Activity Research, Righhopitalet, Copenhagen University, Denmark, Copenhagen, Denmark
6The Norwegian Olympic Sports Centre, Oslo, Norway
7Laboratoire Motricité Humaine Expertise Sport Santé, Université Côte d’Azur, Nice, France
8Department of Respiratory Medicine, Bispebjerg Hospital, Copenhagen, Denmark
9Centre Hospitalier Régional Universitaire de Nancy, Centre Universitaire de Médecine du Sport et Activité Physique Adaptée, Service des Explorations de la Fonction Respiratoire, Université de Lorraine, Nancy, France
10Medical Physiology, Université de Lorraine, Nancy, France
11Institute of Environmental Medicine, Karolinska Institute, Stockholm, Sweden
12Department of Respiratory Medicine, Royal Brompton and Harefield NHS Foundation Trust, London, UK
13Division of Surgery and Interventional Science, Institute of Sport, Exercise and Health (SSEH), University College London, London, UK

Correction notice

This article has been corrected since it published Online First. The title has been corrected.

Twitter

Oliver J Price @olivejprice, Valerie Bougault @VBougault and James H Hull @Breathe_to_win

Contributors

Conception and design: OJP, NS, MS, VBa, TR-N, VBo, LP, BC, KL, JHJ. Analysis and interpretation: OJP, NS, MS, JHH. Drafting the manuscript for important intellectual content: OJP, NS, MS, VBa, TR-N, VBo, LP, BC, KL, JHJ. OJP and JHH confirm full responsibility for the content of the manuscript.

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.

Patient consent for publication

Not required.

Provenance and peer review

Not commissioned; externally peer reviewed.

Supplemental material

This content has been supplied by the author(s). It has not been vetted by BMJ Publishing Group Limited (BMJ) and may not have been peer-reviewed. Any opinions or recommendations discussed are solely those of the author(s) and are not endorsed by BMJ. BMJ disclaims all liability and responsibility arising from any reliance placed on the content. Where the content includes any translated material, BMJ does not warrant the accuracy and reliability of the translations (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages), and is not responsible for any error and/or omissions arising from translation and adaptation or otherwise.

ORCID iDs

Oliver J Price http://orcid.org/0000-0001-8596-4949
Martin Schwellenius http://orcid.org/0000-0003-3647-0429
Valerie Bougault http://orcid.org/0000-0002-2258-6562
James H Hull http://orcid.org/0000-0003-4697-1526

REFERENCES

1 Fitch KD. An overview of asthma and airway hyper-responsiveness in Olympic athletes. Br J Sports Med 2012;46:413–6.
2 Price OJ, Walsted ES, Hull JH. Understanding the total airway response to exercise: a review. Br J Sports Med 2022;56:213–222. doi:10.1136/bjsports-2021-104601
3 Price OJ, Hull JH. Asthma in elite athletes: who cares? Br J Sports Med 2013;47:163–6.
4 Weiler JM, Brannan JD, Randolph CC, et al. Exercise-induced bronchoconstriction update-2016. J Allergy Clin Immunol 2016;138:1292–5.
5 Anderson SD, Sue-Chu M, Perry CP, et al. Bronchial challenges in athletes applying to inhale a beta-2 agonist at the 2004 summer Olympics. J Allergy Clin Immunol 2006;117:767–73.
6 Anderson SD, Fitch K, Perry CP, et al. Responses to bronchial challenge submitted for approval to use inhaled beta-2-agonists before an event at the 2002 winter Olympics. J Allergy Clin Immunol 2003;111:445–50.
7 Page MJ, McKenzie JE, Bosuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71.
8 Kohi C, McIntosh EJ, Unger S. Online tools supporting the conduct and reporting of systematic reviews and systematic maps: a case study on CADIMA and reviewing existing tools. Environmental Evidence 2018;7.
9 Downs SH, Black N. The feasibility of creating a checklist for the assessment of the methodological quality both of randomised and non-randomised studies of health care interventions. J Epidemiol Community Health 1998;52:37–84.
10 Phillips B, Ball C, Sackett D. Oxford centre for evidence-based medicine: levels of evidence. Available: https://www.cebm.net/2009/06/oxford-centre-evidence-based-medicine-levels-evidence-march-2009/ [Accessed March 2009].
11 Ahmad A, Sandlia MP, Siddiqui NA, et al. Exercise-induced bronchospasm in male athletes at karachi. J Ayub Med Coll Abbottabad 2003;15:29–33.
12 Alaranta A, Alaranta H, Palmu P, et al. Asthma medication in Finnish Olympic athletes: no signs of inhaled beta2-agonist overuse. Med Sci Sports Exerc 2004;36:919–24.
13 Batista C, Soares JM. Are former athletes more prone to asthma? J Asthma 2013;50:403–9.
14 Bonini M, Lapucci G, Petrelli G, et al. Predictive value of allergy and pulmonary function tests for the diagnosis of asthma in elite athletes. Allergy 2007;62:1166–70.
15 Bonini M, Gramiconi C, Fioretti D, et al. Asthma, allergy and the Olympics: a 12-year survey in elite athletes. Curr Opin Allergy Clin Immunol 2015;15:184–92.
16 Bougault V, Drouard F, Legall F, et al. Allergies and exercise-induced bronchoconstriction in a youth Academy and reserve professional soccer team. Clin J Sport Med 2017;27:450–6.
17 Burnett DM, Vardiman JR, Dickert JA, et al. Perception of exercise-induced bronchoconstriction in college athletes. Respi Care 2016;61:897–901.
18 Burns J, Mason C, Mueller N, et al. Asthma prevalence in olympic summer athletes and the general population: an analysis of three European countries. Respir Med 2015;109:813–20.
19 Dickinson JW, Whyte GP, McConnell AK, et al. Impact of changes in the IOC-MC asthma criteria: a British perspective. Thorax 2005;60:629–32.
20 Dickinson JW, Whyte GP, McConnell AK, et al. Screening elite winter athletes for exercise induced asthma: a comparison of three challenge methods. Br J Sports Med 2006;40:179–82.
21 Durand F, Kippelen P, Ceugnet F, et al. Undiagnosed exercise-induced bronchoconstriction in ski-mountaineers. Int J Sports Med 2005;26:233–7.
22 Ekland UM, Irewall T, Lindberg A, et al. Prevalence, age at onset, and risk factors of self-reported asthma among Swedish adolescent elite cross-country skiers. Scand J Med Sci Sports 2018;28:180–6.
23 Helenius IJ, Tikkanen HO, Haanheita T. Occurrence of exercise-induced bronchospasm in elite runners: dependence on atopy and exposure to cold air and pollen. Br J Sports Med 1998;32:125–9.
24 Helenius IJ, Tikkanen HO, Sarna S, et al. Asthma and increased bronchial responsiveness in elite athletes: atopy and sport event as risk factors. J Allergy Clin Immunol 1998;101:646–52.
25 Holzer K, Anderson SD, Douglass J. Exercise in elite summer athletes: challenges for diagnosis. J Allergy Clin Immunol 2002;110:374–80.
26 Holzer K, Anderson SD, Chan H-K, et al. Mannitol as a challenge test to identify exercise-induced bronchoconstriction in elite athletes. Am J Respir Crit Care Med 2003;167:534–7.
27 Hunt EB, Murphy B, Murphy C, et al. A study to assess the prevalence of exercise-induced bronchoconstriction in inter-county hurling. Ir J Med 2017;110:655.
28 Jackson AR, Hull JH, Hopker JG, et al. Impact of detecting and treating exercise-induced bronchoconstriction in elite footballers. Eur Respir Rev 2018;4:doi:10.1183/23152829.00122-2017. [Epub ahead of print: 20 04 2018].
29 Kippelen P, Caillaud C, Coste Q, et al. Asthma and exercise-induced bronchoconstriction in amateur endurance-trained athletes. Int J Sports Med 2018;25:130–2.
30 Kukafka DS, Lang DM, Porter S, et al. Exercise-induced bronchospasm in high school athletes via a free running test: incidence and epidemiology. Chest 1998;114:1613–22.
31 Kukafka DS, Lang DM, Porter S, et al. Exercise-induced bronchospasm in high school athletes via a free running test: incidence and epidemiology. Chest 1998;114:1613–22.
32 Koski K, Juntunen P, Koski K, et al. Bronchial response to exercise in Olympic-level endurance athletes. Am J Sports Med 2002;30:1126–31.
