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SERIES: "HOT TOPICS IN PAEDIATRIC ASTHMA"
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The breathless adolescent asthmatic athlete

K-H. Carlsen

ABSTRACT: This article concerns physical activity and sports in asthmatic adolescents. Exercise-induced asthma (EIA) is found in 8–10% of the normal child population and in ~35% of children with current asthma, as reported in a population-based birth cohort study.

The mechanisms of EIA are related to markedly increased ventilation during exercise, which causes increased heat and water loss through respiration, leading to bronchial constriction. In athletes and especially in endurance athletes, the repeated daily physical activity during training will, over time, cause epithelial damage and increase inflammation in the respiratory mucosa. With increased exposure to environmental agents, such as as cold air in skiers and chlorine compounds in swimmers, the athlete may contract symptoms and signs of asthma and bronchial hyperresponsiveness, either worsening existing asthma or causing symptoms in a previous healthy adolescent athlete.

There are several causes of breathlessness in adolescents, including EIA, vocal cord dysfunction, poor physical fitness and others, which are important to consider in the diagnostic procedure.

The asthmatic athlete should follow the same guidelines for treating their asthma as an ordinary asthmatic patient, with careful consideration of the special diagnostic rules given for the use of asthma drugs in sports, especially for inhaled β_2-agonists.

KEYWORDS: Adolescence, athletes, bronchial hyperresponsiveness, children, exercise-induced asthma, treatment

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Sports and physical activity in general are important for the majority of schoolchildren and adolescents. Physical activity has an effect on development, psychological function and growth [1]. In 1989, STRUNK *et al.* [2] reported that fitness was related to psychological function in asthmatic children. Children usually continuously vary their activity between vigorous and low-intensity activity: BAILEY *et al.* [3] reported that children engage in low-intensity activity 77.1% of the time and high-intensity 3.1% of the time, but vary continuously between the two. For the asthmatic child, it is important to control exercise-induced asthma (EIA) without being dependent upon planned pre-medication before planned exercise training. Thus, anti-inflammatory treatment controlling asthma becomes important for the daily life activities of children and adolescents with asthma. Recognising this, treating and preventing EIA has become one of the main objectives of treating asthma among all international and national guidelines for the treatment of childhood asthma [4–6]. This also has obvious relevance to the asthmatic adolescent actively participating in sports.

PREVALENCE OF EIA AND EXERCISE-INDUCED BRONCHOCONSTRICTION

We define exercise-induced symptoms and signs of asthma occurring after intensive physical exercise as EIA. The reduction in lung function (forced expiratory volume in 1 s (FEV1)) occurring after a

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standardised exercise test is called exercise-induced bronchoconstriction (EIB) [7]. A $\geqslant$10% reduction in FEV_1 after exercise is the common definition of EIB [7]. EIA has been reported to occur frequently in asthmatic children and adults without anti-inflammatory treatment, and reported in 70–80% of untreated asthmatics [8]. In the Environment and Childhood Asthma birth cohort study in Oslo (Norway), 36.7% of 10-yr-old children with current asthma had a positive exercise test and thus suffered from EIB regardless of type of treatment, whereas in the entire population-based cohort, 8.6% had a positive EIB test [9].

WHY DOES EXERCISE CAUSE ASTHMA SYMPTOMS?

Physical activity increases the need for oxygen, thus increasing ventilation and, therefore, the amount of air passing through the airways to and from the lungs. On its passage through the airways, the incoming air is heated to 37°C and is fully saturated with water vapour. This gives rise to increased water and heat loss *via* respiration. Cooling of the airways causes reflex parasympathetic nerve stimulation leading to bronchoconstriction through stimulation of the vagal nerve, initially causing reflex vasoconstriction of bronchial venules to conserve heat, and then, at the end of the exercise and increased ventilation, causing vasodilatation. This causes a combination of smooth muscle constriction and mucosal oedema in susceptible individuals [10], reducing the size of the bronchial lumen, leading to increased airway resistance [11]. However, the increased water loss due to the rise in ventilation is considered to be more important than the heat loss, as it increases the osmolality of the extracellular fluid of the bronchial mucosal membranes. ANDERSON and DAVISKAS [12] have shown that the osmotic gradient caused by water loss from the bronchial mucosa induces water to move extracellularly and, through regulatory volume increase, causes the intracellular concentration of ions to rise, as demonstrated for calcium and inositol triphosphate [13]. This process may lead to the release of both newly formed mediators, through lysis of phospholipids releasing eicosanoids, and pre-formed mediators, such as histamine from intracellular granules, thus causing bronchoconstriction. It has been suggested that cold air exerts its effect through its low water content, thus participating in the drying of the respiratory mucosa [12]. These mechanisms explain why EIA often occurs shortly after heavy exercise and not during maximum exercise intensity, a fact that is important in the diagnosis of EIA.

PARTICIPATION IN PHYSICAL ACTIVITY AND SPORTS AND TRAINING OF ASTHMATIC ADOLESCENTS

It has been reported that asthma limits the participation of children in physical activity, especially vigorous physical activity [14]. Therefore, optimal asthma treatment is important in order to control EIA and allow full participation in physical activity, play and sporting activities [15]. When asthmatic children participate in physical exercise activities and training, their quality of life improves as well as their physical fitness [15], although asthma severity and lung function are not influenced [16]. In addition to quality of life and fitness, the level of physical activity is also increased after participating in training, leading to a more active and, according to FLAPPER *et al.* [17], happier life.

With modern asthma care, asthmatic 13-yr-old children from the Oslo (Norway) birth cohort study were as fit as healthy children and as physically active [18]. Thus, asthmatic children and adolescents can be as physically active and fit as healthy children, and may participate in sports activities on an equal level with them, provided they receive optimal treatment for their asthma.

Several studies report that when participating in systematic physical training, the asthmatic adolescent or child improves their fitness, quality of life and exercise level [15, 17]. This was confirmed by RAM *et al.* [16], who performed a Cochrane Database-based meta-analysis of eight training studies including 226 asthmatics from 6 yrs of age. They concluded that physical training improved fitness as measured by increased maximum oxygen uptake. However, there was no change in lung function. After that study was published, COUNIL *et al.* [19] reported similar findings: improvement in aerobic and anaerobic fitness in their adolescent training group (mean age 13 yrs), but no improvement in lung function after 6 weeks of exercise training with bouts of high-intensity physical activity. FANELLI *et al.* [20] also reported improved quality of life and asthma control in their actively training group compared with a nontraining group. MOREIRA *et al.* [21] conducted a 3-month training programme in adolescents (mean age 13.4 yrs) and found no changes in asthma control; however, they reported a reduction in total and house dust mite-specific immunoglobulin E in their training group.

In addition, VAHLKVIST and co-workers [22, 23] reported that newly diagnosed asthmatic adolescents were less fit and had lower levels of vigorous physical activity than control subjects; starting anti-inflammatory treatment and obtaining better asthma control increased both fitness and level of vigorous activity after 1 yr of treatment. This also shows that level of physical activity in asthmatic children and adolescents may be seen as a marker of asthma control [23].

These later studies thus confirm the meta-analysis by RAM *et al.* [16], and all studies conclude that physical training is recommended in asthmatic children.

WHY DO HEALTHY ADOLESCENT ATHLETES DEVELOP ASTHMA AND BRONCHIAL HYPERRESPONSIVENESS?

Increased prevalence of asthma and bronchial hyperresponsiveness (BHR) has been reported among elite athletes, especially in endurance sports and most frequently in cross-country skiers and swimmers [24–26]. In Olympic athletes, a similar pattern was reported for the use of asthma drugs during the Olympic Games [27]. In cross-country skiers, the prevalence of asthma and BHR was found to increase with increasing age, and the prevalence was found to be higher the longer the athletes had competed [24, 28]. Among adolescent swimmers, we found a high prevalence of BHR from the age of 16 to 22 yrs, both measured by methacholine bronchial provocation and by eucapnic voluntary hyperpnoea [29]. Furthermore, a relationship between exposure to chlorinated swimming pools during childhood and adolescence, and prevalence of asthma and EIA suggests that exposure to chlorine and organic chlorine products during physical activity represents an environmental hazard [30, 31].

BOUGAULT and co-workers [32–34] reported several studies on swimmers and winter sport athletes. They reported that positive methacholine bronchial challenge tests were common

in competitive swimmers and winter sport athletes compared with healthy controls, being most common in swimmers [32]. Bronchial challenge testing with methacholine was found to be positive more often than tests for eucapnic voluntary hyperpnoea [33]. Of particular interest was the finding of increased neutrophil counts in induced sputum from both swimmers and winter sport athletes, and that the neutrophil counts correlated with the number of training hours per week in both groups [32]. Eosinophil counts were particularly increased in swimmers, as was the number of bronchial epithelial cells [32]. The relationship with training is of particular interest, and BOUGAULT *et al.* [34] found that in swimmers, 2 weeks of rest from training induced a reduction in bronchial responsiveness both to methacholine and eucapnic voluntary hyperpnoea. Eight out of 12 swimmers with BHR during training had no BHR during rest. In this particular study, no airway inflammation was detected, as measured by exhaled nitric oxide and induced sputum analyses, and no change in airway inflammation occurred from training to rest [34].

The first report that BHR increased after heavy exercise came from a study on adolescent swimmers (12–18 yrs of age) swimming 3,000 m. The increase in bronchial responsiveness from before to after the heavy exercise correlated with the level of exercise load (increase in blood lactate) in both asthmatics and healthy swimmers [35]. Later, SUE-CHU *et al.* [36] showed that during one competitive winter season, adolescent cross-country skiers developed signs of inflammation (lymphoid follicles and deposition of tenascin) in their bronchial biopsies whether they were asthmatic or not. These findings were later confirmed in animal studies: mice exercised by running developed signs of inflammation and epithelial damage in their lungs compared with sedentary mice [37]; Alaskan sledge dogs were examined by bronchoscopy and bronchoalveolar lavage before and after a sledge race across Alaska (USA) with similar findings [38]. These findings were recently replicated in humans, as increased levels of bronchial epithelial cells were found in induced sputum of amateur endurance runners after repeated half-marathon races, in addition to apoptosis of bronchial cells after the races. Increased serum levels of Clara cell protein 16 and increased interleukin-8 levels in induced sputum supernatants were also found, demonstrating increased airway inflammation caused by participation in the half-marathon races [39]. These studies agree with the findings in induced sputum from swimmers and cold weather athletes reported by BOUGAULT *et al.* [32]. Thus, training with a heavy exercise load will, through increased ventilation, cause a rise in airway wear and tear on the respiratory epithelium, with airway inflammation and epithelial damage as a result.

Water movement across cell membranes has been stated as an important part of the pathogenesis of EIA [12]. Aquaporins (AQPs) are channels for aqueous water transport driven by osmotic forces generated by sodium and chlorine ions, which are expressed in respiratory subepithelial glandular cells and alveolar type 1 cells in the lungs [40]. It has been shown that mice lacking the gene for AQP5 exhibit methacholine-induced bronchiolar hyperreactivity compared with normal mice [41]. PARK *et al.* [40] recently reported that in healthy athletes suspected of having EIA, a relationship was found between bronchial responsiveness to methacholine and diminished pilocarpine-induced sweat secretion, tearing rate and salivary flow rate. This indicates the possibility of a genetic factor making athletes more susceptible to developing EIA and BHR.

Intensive and regularly repeated training has been shown to influence autonomic regulation, with increased sympathetic and parasympathetic activity. FILIPE *et al.* [42] demonstrated increased parasympathetic activity in athletes by pupillometry, which was significantly higher in endurance runners. This is consistent with our practical experience that Norwegian competitive endurance athletes in our national teams respond particularly well to inhaled ipratropium bromide and with a higher reversibility to this drug than to inhaled β_2-agonists. This is also supported by the reports of KNOPFLI and co-workers [43–45] on cross-country skiers and training children.

THE ENVIRONMENTAL FACTOR

The exercising athlete will be more exposed to environmental agents in the surrounding air due to their increased ventilation during exercise. This is supported by the studies of children in swimming pools by BERNARD *et al.* [30], measurements of bronchial responsiveness in adolescent Norwegian competitive swimmers [29] and increased inflammatory markers in Finnish competitive swimmers [46]. Environmental exposures differ between sports; for example, cross-country skiers are repeatedly exposed to cold air [24], and athletes training and competing on ice rinks may be exposed to nitrogen oxides from the freezing machinery and ultrafine particles from polishing machines [47], corresponding to reports of high asthma prevalence among ice-hockey players [48] and figure skaters [49].

In a study conducted in southern California, USA, increased prevalence of asthma was found in children participating in sports in areas with high environmental ozone pollution. The study enrolled 3,535 children from 12 areas in southern California: six areas with a high level of pollution (ozone) and six with low pollution levels. After a follow-up period of 5 yrs, it was found that children actively participating in more than three types of sports in areas with high ozone levels had an increased relative risk of asthma of 3.3. Participation in sports in areas with low ozone levels gave no increased risk of asthma. Time spent outdoors in areas with high ozone levels was related to increased risk of asthma, but not in areas with low ozone levels [50]. Together with studies from Belgian swimming pools [30, 31], this demonstrates that it is necessary to have strict criteria for environmental exposures in sporting arenas where children and adolescents train and compete. Setting high standards for environmental conditions for sports may have important health consequences for the athletes.

There are two main phenotypes of asthma in endurance athletes. First, there are athletes who have asthma from early childhood, often accompanied by allergy. Secondly, there are those athletes who contract their asthmatic symptoms through repeated heavy training and competitions. The latter may not have the obvious asthmatic symptoms caused by acute episodes of bronchoconstriction, but rather experience cough and phlegm over prolonged periods of time, which may often be provoked by repeated competitions and viral infections. The latter phenotype is not unlike chronic persistent asthma.

Asthma in competitive athletes may occasionally be fatal. An American study identified nationwide deaths related to

competitive sports activities over a 7-yr period. Out of 263 sports-related deaths, 61 deaths were related to asthma provoked by competitive sports. Of these 61 deaths, most occurred in males <20 yrs of age; only one of the athletes who died used inhaled steroids and two used disodium cromoglycate, while the remainder used no controller asthma medication, underlining the necessity of optimal asthma care in asthmatic athletes [51].

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF EIA IN ADOLESCENT ATHLETES

Diagnosis of EIA may be made using a standardised exercise test. Standardisation of the test is very important for the outcome. The exercise load should be high, preferably ⩽95% of maximum exercise load for the adolescent, as assessed by heart rate [52]. The test should also be standardised with regard to environmental temperature and humidity [53]. The exercise test has high specificity for asthma, but lower sensitivity, especially when the adolescents are treated with inhaled steroids. Testing directly for bronchial responsiveness using bronchial challenge with methacholine has a higher sensitivity, but a lower specificity, for asthma [28, 54, 55]. The methacholine test represents a useful measurement of the athlete's respiratory problems.

It has been stated that sport-specific field exercise tests are much more sensitive in athletes [56], but this could not be verified in another study [28]. In addition, eucapnic voluntary hyperpnoea [57], although an indirect test of bronchial responsiveness, is a sensitive test in athletes. Performance of this test is physically demanding [29]. Inhalation of mannitol has also been suggested as a substitute measure of EIA [58]. The mannitol test may provide information about indirect bronchial responsiveness comparable to EIB, but will give none of the information about physical fitness, motor skills and motor development that is also obtained by a trained observer through an exercise test in children and adolescents.

EIA usually occurs shortly after heavy exercise. Typically, bronchial constriction reaches its maximum 6–10 min after exercise, and the dyspnoea will be expiratory. If respiratory distress occurs during maximum exercise and is inspiratory, exercise-induced vocal cord dysfunction (VCD) may be a more probable diagnosis. The condition was first described by REFSUM *et al.* [59], then later by MCFADDEN JR and ZAWADSKI [60], and in adolescents by LANDWEHR *et al.* [61]; among well-trained adolescent athletes, and particularly females, this diagnosis is as a frequent as EIA. This differential diagnosis is very important, as asthma treatment will have no effect on this condition. One should be aware that EIA may coexist with VCD [62]. There is no consensus definition of VCD, but the following has been proposed: an intermittent extrathoracic airway obstruction, mainly during inspiration, leading to dyspnoea of varying intensity [63]. Exercise-induced VCD should be suspected when respiratory stridor occurs during maximum exercise intensity and is inspiratory. A ratio of less than one for maximal inspiratory flow at 50% of forced vital capacity/maximal expiratory flow at 50% of forced vital capacity, after a methacholine bronchial provocation, has been taken as suggestive of VCD [63]. A maximum-intensity treadmill run, when audible inspiratory stridor occurs during maximum intensity, can confirm the diagnosis. The diagnosis is further verified by continuous laryngoscopic exercise testing [64, 65]. There are different treatment modalities for exercise-induced VCD, and

TABLE 1 Possible differential diagnoses to exercise-induced asthma (EIA)

Diagnosis	Clinical presentation in the athlete
EIA	Symptoms occur shortly after (sometimes during) physical exercise
	Expiratory dyspnoea
	Rhonchi and sibilating rhonchi observed by auscultation
	Respiratory retractions
	Gradual improvement occurs either spontaneously or after inhaled bronchodilator
Exercise-induced VCD	Symptoms occur during maximum exertion
	Symptoms disappear when exercise is stopped, unless the patient continues to hyperventilate
	Inspiratory dyspnoea
	Audible inspiratory sounds from the laryngeal area and no signs of bronchial obstruction
	No effect of pre-treatment with inhaled bronchodilator [65]
Exercise-induced hyperventilation	Hyperventilation with respiratory dyspnoea and increased end-tidal carbon dioxide [67]
EIAH	Occurs in well-trained athletes with high maximum oxygen uptake
	Primarily thought to be due to diffusion limitations and ventilation–perfusion inequality
	Incomplete diffusion in the healthy lung may be due to a rapid red blood cell transit time through pulmonary capillaries [68]
Swimming-induced pulmonary oedema	May occur after heavy swimming exercises with symptoms of haemoptysis, cough and respiratory distress
	Reduced FVC/FEV_1 for ⩽1 week afterwards [69]
Other chronic lung diseases	Reduced baseline lung function may reduce physical performance due to limitations in airflow and lung volumes [70]
Other general disease	Chronic heart diseases and other general disorders
Poor physical fitness including obesity	Related to expectations
	High heart rate after low exercise load

VCD: vocal cord dysfunction; EIAH: exercise-induced arterial hypoxaemia; FVC: forced vital capacity; FEV_1: forced expiratory volume in 1 s.

even surgery through endoscopic supraglottoplasty has been described as useful in selected patients [66]. Exercise-induced hyperventilation with increased end-tidal CO_2 at the end of exercise has also been described as a differential diagnosis to EIA in children and adolescents [67].

Other, not uncommon differential diagnoses to EIA are presented in table 1. Among these, unexpectedly poor physical fitness should be mentioned. This includes obesity, which may represent a differential diagnosis to EIA in the common asthmatic patient, but rarely in athletes.

TREATMENT OF EIA IN ADOLESCENT ATHLETES

Asthma in adolescent athletes should be treated in the same way and according to similar guidelines as asthma in other adolescents. However, special precautions must be taken related to the rules given by the doping authorities with respect to the use of inhaled β_2-agonists and inhaled steroids in sports. These rules often change, sometimes annually, even if the changes are minor. However, the physician caring for children and adolescents who are active in sports should know and be updated on these rules (tables 2 and 3).

According to the commonly used guidelines, mild asthma should be treated with inhaled β_2-agonists *p.r.n*. However, inhaled β_2-agonists were subject to restrictions for use in sports and mild asthmatics did not satisfy those restrictions. Then, from January 1, 2011, the World Anti-Doping Association (WADA) changed the rules. Inhaled salbutamol may now be used freely with a maximum daily dosage of $\leqslant$1,600 μg. This is also the case for inhaled salmeterol, without a specified maximum dosage. However, for the use of inhaled terbutaline and inhaled formoterol, the old rules still hold, and application for a therapeutic use exemption (TUE) is necessary for the use of these drugs. WADA has given no reasons for the change of rules and there is no scientific reason why inhaled β_2-agonists from one pharmaceutical company should be free, whereas drugs from other pharmaceutical companies should be restricted. From January 1, 2011, all inhaled steroids are free for use.

When bronchodilation is needed, inhaled ipratropium bromide may also be tried before exercise or competition, as this drug has no restrictions related to sports. Montelukast also has no restrictions and can be tried, both due to its bronchodilating and its partly anti-inflammatory effect. With the presence of BHR and respiratory symptoms, inhaled steroids should be tried. Inhaled steroids are among the drugs that can be used freely from January 1, 2011, leaving the decision of use to the physician. If the athlete satisfies the new rules for use of restricted inhaled β_2-agonists, treatment can be given according to the commonly used guidelines, but with the use of TUEs for certain drugs (table 3). The common guidelines for controlling and relieving medications should be followed. It should also be remembered that prescribing treatment is part of the diagnostic process. If treatment fails, the diagnosis should be reconsidered.

HOW TO HELP ATHLETIC ADOLESCENTS WITH ASTHMA AND BHR TO CONTROL THEIR ASTHMA WITHOUT CONFLICT WITH THE RULES OF SPORTS AND DOPING AUTHORITIES

The special rules for the use of asthma drugs in sports should be understood and taken into consideration. When treating an athlete for respiratory problems during physical activity, the restricted drugs should not be prescribed until a thorough examination has been made. This is very important if the athlete is to avoid problems with the doping authorities.

The physician should know the current rules for the use of asthma drugs in sport. Changes in the rules are often made at the start of the year. The valid rules can be found on the website of the WADA and on the websites of national anti-doping authorities. The new rules for 2011 are given in table 3.

TABLE 2 Overview of the asthma drugs for which the athlete must apply for the use through a therapeutic use exemption (TUE) from January 1, 2011, according to World Anti-Doping Agency regulations

Medication	Regulations
Inhaled β_2-agonists	
Salbutamol	Free use up to a maximum daily dose of 1600 μg
	Urinary salbutamol >1000 $ng \cdot mL^{-1}$ is considered an adverse analytical finding unless proven that this was due to a dose of inhaled salbutamol $\leqslant$1600 μg over 24 h
Salmeterol	Free use
Terbutaline	Prohibited
	TUE necessary
Formoterol	Prohibited
	TUE necessary
Other inhaled β_2-agonists	Prohibited
All inhaled steroids	Free use
Combination inhaled steroid + β_2-agonist	
Inhaled steroid + formoterol	Formoterol prohibited
	TUE necessary
Inhaled steroid + salmeterol	Free use
Inhaled ipratropium bromide	Free use
Montelukast	Free use

TABLE 3 Diagnostic procedures in the process of diagnosing asthma in sports according to present rules for obtaining a therapeutic use exemption (TUE) for certain inhaled β_2-agonists (terbutaline and formoterol), as required by the World Anti-Doping Agency and the International Olympic Committee Medical Commission (IOC-MC)

Diagnostic procedure	Results required
Clinical history of respiratory symptoms and clinical examination + one of the following objective tests	Positive clinical history and signs indicative of the presence of asthma
Lung function[#]	12% increase in FEV_1 after inhaled bronchodilator
Standardised exercise test for EIB or exercise field test	10% decrease in FEV_1 after exercise challenge
BHR to methacholine[¶]	$PC_{20} \leqslant 4$ mg·mL^{-1} or $PD_{20} \leqslant 2$ μmol in athletes without inhaled steroids
	$PC_{20} \leqslant 16$ mg·mL^{-1} or $PD_{20} \leqslant 8$ μmol in athletes on inhaled steroids for $\geqslant 1$ month
Mannitol inhalation test[+]	$\geqslant 15\%$ reduction in FEV_1
	Determination of mannitol PD_{15}
Eucapnic voluntary hyperventilation test	$\geqslant 10\%$ reduction in FEV_1
Inhalation of hyperosmolar solutions	$\geqslant 15\%$ reduction in FEV_1

EIB: exercise-induced bronchoconstriction; BHR: bronchial hyperresponsiveness; FEV_1: forced expiratory volume in 1 s; PC_{20}: provocative concentration causing a 20% decrease in FEV_1; PD_{20}: provocative dose causing a 20% decrease in FEV_1; PD_{15}: provocative dose causing a 15% decrease in FEV_1. [#]: spirometry with reversibility to inhaled bronchodilator; [¶]: histamine not currently permitted by IOC-MC; [+]: Aridol® test (Pharmaxis Ltd, Frenchs Forest, NSW, Australia).

Internationally competing athletes use a web-based system to inform the doping authorities where they will be staying at any time (called ADAMS). Within this system, there are specific forms to be completed for TUEs. Printouts of lung function examinations and measurements of BHR can be scanned and attached to these forms. For some asthma drugs, the athlete must apply through a TUE; other drugs are now completely free (inhaled steroids, salbutamol and salmeterol). Declarations of use were introduced in 2010 but have now been abolished. When submitting an application for TUE, the athlete will receive approval (or rejection) within a few weeks after submitting the form.

For the use of certain inhaled β_2-agonists, there are objective requirements to be met. It is the responsibility of the athletes themselves to know the rules and to abide by them. Systemic β_2-agonists are not allowed at all. To use the inhaled β_2-agonists terbutaline and formoterol, clinical symptoms of asthma and additional objective measurements are required, as shown in table 3. The rules for the different drugs are given in table 2.

Athletes on a national level do not have to apply for a TUE. However, they will have to satisfy the same rules to use the drugs. If the athlete is subjected to a doping test, the doping authorities will collect information from the relevant physician.

It is the responsibility of the athletes themselves to know the rules and to abide by them. However, the physician caring for adolescent athletes should know the rules in order to be able to offer care in accordance with the rules and thus avoid any problems for the athlete.

It is important to offer the correct treatment for the individual adolescent athlete, in accordance with their disease severity and the rules of the sport. The aim is to help the athlete with asthma to fulfil their potential in physical activity and sports in spite of their illness. We are now able to control asthma and help the patient to optimise their asthma control in such a way that the adolescent athlete with asthma may compete at an international level.

STATEMENT OF INTEREST

None declared.

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