INTRODUCTION

Regular physical exercise is recognised as an important component to attain optimal health and swimming is among the preferred activities in developed countries. Buoyancy properties of water and the warm humid atmosphere of indoor swimming pools are particularly attractive for infants, the older generation or people with various ailments. Although there are recognised benefits on health, there is evidence that people who regularly attend indoor chlorinated swimming pools are at risk of developing airway disorders, associated with repeated inhalation of chlorine byproducts.1–4

Chlorine, an inexpensive and easy-to-use product, is widely used in swimming pools worldwide as a chemical disinfectant, serving as the principal barrier to microbial contaminants.5 During chlorination process, chlorine reacts with organic matter brought by swimmers to form disinfection byproducts such as chloramines. The concentrations of these chlorine byproducts are regularly controlled in the pools’ water and regulated by the WHO.5 However, gases such as nitrogen trichloride (NCl3) are also formed during the reaction. NCl3 is responsible for the strong smell reported by swimmers in chlorinated swimming pools and is strongly irritating for the airways.5 According to guidelines from the WHO, the maximum cut-off for NCl3 in pools’ atmosphere is 0.5 mg/m3,5 but its levels are not routinely measured. Other more expensive disinfectants may be used to replace chlorine, but data are still limited on their effects on the respiratory tract.

As the ventilation rate during swimming determines the quantity of inhaled chlorine byproducts, elite swimmers, training many hours in the pools, are likely to be more affected by these products. Exposure to these products may also account for the high prevalence of upper and lower airway disorders reported in this group.5 In keeping with this possibility, is the recent finding of significant airway inflammation and remodelling on bronchial biopsies of swimmers similar to what is found in those with mild asthma.7

In this report, we discuss the prevalence of upper and lower airway disorders in swimmers, underlying mechanisms of development and persistence, their general management and the future research needed to help understand their clinical significance in order to prevent potential long-term damage to the airways.

ABSTRACT

Elite competitive swimmers are particularly affected by airway disorders that are probably related to regular and intense training sessions in a chlorinated environment. Upper and lower airway respiratory symptoms, rhinitis, airway hyper-responsiveness, and exercise-induced bronchoconstriction are highly prevalent in these athletes, but their influence on athletic performance is still unclear. The authors reviewed the main upper and lower respiratory ailments observed in competitive swimmers who train in indoor swimming pools, their pathophysiology, clinical significance and possible effects on performance. Issues regarding the screening of these disorders, their management and preventive measures are addressed.

ADVERSE RESPIRATORY HEALTH EFFECTS OF SWIMMING IN CHLORINATED SWIMMING POOLS

Airway symptoms are often reported by children, lifeguards, pool workers and swimmers, who are regularly exposed to a chlorinated swimming pool environment.1489

Upper airways

Prevalence and diagnosis

The most disturbing symptoms among swimmers seem to originate from the upper airways. Nasal obstruction, rhinorhoea, sneezing and nasal itching are reported by up to 74% of competitive elite swimmers, during the training season.9–11 The diagnosis of nasal disorders is often made through a physical examination and medical history. In the case of recurrent obstruction or rhinorhoea, nasal cytology, endoscopy and/or rhinomanometry may provide additional information on the nature of the disease.11

Mechanisms of rhinitis

Exercise itself may cause rhinitis in athletes independently of atopic status.12 There may be a decrease in nasal resistance after exercise,13 following the stimulation of the autonomic nervous system during physical activity. The latter may act as an active decongestant (sympathetic activation), although it may also induce exercise-induced rhinorhoea (parasympathetic influence).1314

In a recent study on nasal cytology, 44% of young competitive swimmers with rhinitis had a pre-existing allergic component. In those with non-allergic rhinitis, 63% had a predominant nasal neutrophilic inflammation.11 After the use of a nose-clip during a 30-day swimming period, nasal symptoms were significantly reduced, particularly in those with neutrophilic inflammation. This highlights the irritating role of chlorine on the nasal mucosa.11 The mechanisms and consequences of such an irritating effect are still unclear, but epithelial damage may occur, as observed in the lower airways.3

Nasal symptoms may also be due to an acute viral infection or a postinfectious state. There is a high incidence of viral infections in elite swimmers
To regularly measure the level of NCl₃ in the air.

Clinical consequences and possible effects on performance

Nasal symptoms experienced by competitive swimmers during the training season impair their quality of life and may worsen their performance. Symptoms due to allergic rhinoconjunctivitis, quality of life and performance may improve with appropriate medication and environmental measures.

Pyne et al failed to provide significant evidence for an association between upper airway disorders and competitive performance in elite swimmers. However, the authors judiciously emphasised that the mean differences observed in performance time between healthy and affected swimmers (ie, 0.1 to 0.5 s in 100 to 200 m events) were greater than differences between winning a medal or missing a medal altogether.

Lower airway diseases

Prevalence and diagnosis

Screening in competitive swimmers has demonstrated that up to 76% have symptomatic or asymptomatic airway hyperresponsiveness (AHR) and/or exercise-induced bronchoconstriction (EIB). Asthma is generally diagnosed clinically on the basis of symptoms such as wheezing, dyspnoea, chest tightness, phlegm production and cough, associated with objective evidence of variable airway obstruction or AHR. The prevalence of asthma before the beginning of the competitive career seems to be similar or slightly increased in swimmers, compared with other endurance athletes and healthy subjects. This suggests that swimmers do not engage in swimming due to their asthma, but rather develop respiratory disorders during their athletic career. Moreover, the development of AHR generally occurs in young adults rather than in adolescent competitive swimmers. Such AHR may be the result of the cumulative effects of years of intense swimming training, the repeated inhalation of large volumes of air containing chlorine byproducts possibly inducing airway inflammatory and structural changes.

Symptoms

Asthma-like symptoms are commonly reported by swimmers, children, lifeguards and bathers who regularly attend chlorinated swimming pools. Cough is the most common exercise-related symptom and a study reported that 18% of swimmers had to cease a training session at least once due to the severity of the symptoms associated with the strong odour of the pool environment.

Pulmonary/airway function changes

Many competitive swimmers demonstrate AHR to direct or indirect stimuli, independently of the presence or not...
of swimming-related symptoms. In accordance with the criteria used by the International Olympic Committee-Medical Commission’s (IOC-MC) Independent Asthma Panel, about 60% of young competitive swimmers had AHR to at least one bronchial provocation test. Little information is currently available on the comparison between synchronised swimmers, free-water swimmers, swimmers, divers and water-polo players. However, the very high training level encountered in Olympic swimmers and synchronised swimmers, training up to 40 h per week, may explain the high prevalence of AHR in these two swimming specialities. In swimmers, AHR is usually found in the presence of a normal resting flow-volume curve, emphasising the necessity to use a bronchial provocation or a reversibility test to assess airway function. Furthermore, in some athletes, provocation tests with direct stimuli such as methacholine may be negative while EIB can be confirmed by indirect tests such as eucapnic voluntary hyperpnoea (EVH) in these athletes.

**Mechanisms**

As for endurance athletes, increased ventilation sustained for many hours per day during many weeks of swimming training can probably affect the airway epithelium. Epithelial damage, in addition to the osmotic stress, may lead to the release of inflammatory mediators and possibly initiate a repair and rehydration process involving fibrogenic cytokines. Since an indoor swimming pool environment is warm and humid, dehydration of the airways should be less than for other sports, thus protecting against EIB and AHR. However, further evidence suggests that in chlorinated indoor swimming pools, repeated inhalation of chloramines formed during the reaction of chlorine with organic matter brought in the pool by swimmers can contribute over the years to allergic diseases. Elite swimmers, breathing large volumes of air containing chlorine derivatives, immediately above the water surface, are particularly at risk. In young children, the possibility that asthma develops following repeated attendance of chlorinated swimming pools remains controversial and needs further study to better assess the role of chlorine in the development of airway disorders.

**Epithelial damage and chlorine hypothesis**

Indoor chlorinated swimming pool attendance induces a rapid and transient disruption of the airway epithelium in recreational swimmers, children or passively exposed attendees, without concomitant development of asthma symptoms. Inhaled chlorine byproducts certainly have the potential to cause structural changes in the airway epithelium, thus permitting allergens easier access to antigen-presenting cells, possibly promoting allergen sensitisation. Allergies, AHR, asthma and other airway disorders may then possibly develop as a consequence of repeated exposure to these byproducts.

The mechanisms responsible for epithelial damage and AHR in swimmers remain incompletely understood. The increased oxidative stress observed in swimmers’ airways and reduced antioxidant capacity may promote the release of inflammatory mediators and sensitisation of airway smooth muscle, contributing to the development of AHR in swimmers.

**Inflammation and remodelling of the airways**

Similar inflammation and remodelling changes have been observed in the bronchial mucosa and/or sputum of competitive swimmers, when compared with nonathletes with mild asthma. Bronchial remodelling was observed in swimmers independently of AHR to methacholine or EVH. These changes could presumably constitute a normal adaptation process to intense training and be partly reversible after the end of the swimming career, although this remains to be confirmed. Despite the fact that clinical consequences of these changes remain uncertain, it seems advisable to propose preventive measures such as reduction of chloramines’ exposure in pool environments. We do not know, however, if preventive treatments should be offered to swimmers, in order to try to reduce airway inflammation and the possibility of structural changes.

Non-invasive measures of airway inflammation, such as with exhaled nitric oxide (eNO) or induced sputum analysis, may be useful to detect active airway disease, but these tests may not be sensitive enough to detect a more subtle inflammation of the airway mucosa. Neutrophil cell counts in induced sputum of competitive swimmers have been shown to correlate with the number of training hours per week. However, no significant difference in induced sputum inflammatory cell counts was observed between competitive swimmers and controls, when performed 12 h or more after the last training session. Furthermore, no change in eNO was observed after swimming training in competitive swimmers.

**Clinical consequences and possible effects on performance**

When treated, Olympic swimmers with asthma perform their sport as well or better than other athletes without asthma. The consequences of EIB on performance seem obvious, but for asymptomatic AHR, they remain unclear. In a certain proportion of the general population, asymptomatic AHR is associated with the subsequent development of asthma, particularly in subjects with atopy. In swimmers, AHR has been shown to be transient. The clinical consequences of AHR in this group are also unclear and need to be further studied.

**What do the swimmers report to the physician?**

It has been suggested that competitive swimmers, particularly the youngest, may consider their upper and lower airway symptoms as a ‘normal phenomenon’ due to the intensity of exercise or the presence of a strong chemical odour in the swimming pool rather than rhinitis or asthma. Consequently, many of them do not mention symptoms to their general physician, and do not get the opportunity to consult a respiratory physician.

**Should swimmers be screened for respiratory disorders?**

Whether we should screen competitive swimmers for rhinitis, allergy, and AHR or asthma is still debated, mainly due to the costs involved. Currently, questionnaires are available on allergies and respiratory symptoms of athletes, and may constitute an inexpensive prescreening assessment. Spirometry and a bronchodilator reversibility test are also easy to perform and may be ordered on a yearly basis in high-level swimmers, despite a high baseline forced expiratory volume in one second. In athletes reporting symptoms during swimming, the recommendations of the IOC-MC Independent Asthma Panel should be followed and a test should be performed to prove AHR or EIB, according to the described methodologies.

**GENERAL MANAGEMENT AND PREVENTION**

**Management**

Currently, rhinitis and asthma in swimmers are managed as in the general population. No data, however, are available on the benefits of treatment with nasal anticholinergic agents or corticosteroids for rhinitis in swimmers, a disorder that is...
mainly characterised by a neutrophilic rather than eosinophilic inflammation. The management of asthma is mostly based on the use of fast-acting bronchodilators for intermittent symptoms or for the prevention of EIB, in addition to inhaled corticosteroids (ICS). Inhaled long-acting β2-agonists or leucotriene receptor antagonists may be added if asthma control is not achieved by a low dose of ICS.43

**Prevention**
Considering its simplicity to use, efficiency and low cost, chlorine will probably continue to be used in the future. Reducing exposure to inhaled chlorine byproducts, however, could potentially help reduce the risk of airway disorder in swimmers. A summary of recommendations to enhance the quality of water and air in the indoor swimming pools is presented in table 1.

**Should we replace chlorine by other disinfectants?**
Alternative chemical disinfectants to chlorine such as ozone and chlorine dioxide are increasingly being used.5 Some studies reported that the use of a copper-silver disinfectant does not induce epithelial damage in swimmers or favour the development of asthma in school children.53,54 On the other hand, copper-silver disinfection cannot remove organic material and does not seem to ensure the total elimination of viral pathogens from water, even if its use is combined with low levels of chlorine.45 Each alternative disinfection measure produces its own set of byproducts and/or is partially effective as a means of disinfection.5 Therefore, there is a need to better understand the chemistry, toxicology and epidemiology of chemical disinfectants and their associated byproducts of disinfection. However, whatever the product uses, it should not compromise the microbiological quality of swimming pools’ water.

**A need to change bathers’ behaviour**
Because NCl3 levels in the air above swimming pool water is influenced by ambient ventilation and water chemistry, a proper ventilation of the pool environment and reduction of human protein matter in the water may reduce respiratory health problems of swimmers.5 Efficient ventilation in indoor swimming pools would increase the turnover of ambient air and remove concentrated chloramines.

An efficient measure to reduce chloramines production would also be a modification of swimmer’s behaviour (table 1). In addition, adhering to a maximal occupancy rate in the swimming pools, expressed as the number of bathers per unit of volume, would help to keep cleaner air and water of swimming pools.46 Altogether, these measures could significantly reduce the accumulation of chloramines in the swimming pools’ ambient air and contribute to a better respiratory health of the swimmers and bathers.

**CONCLUSION**
Particular attention should be paid to the respiratory health of competitive swimmers, its management and prevention. Simple measures could be taken to reduce airway dysfunction in this population with minimal financial costs. Further studies are needed to understand the combined effects of exercise and inhalation of chlorine byproducts and the direct effect of chloramines as potential inducers of epithelial damage and airway inflammation. The impact of such disorders on athletic performance and their effects on the health of swimmers during and after the end of their training career should be evaluated.

**What this study adds**
This review underlines the impact of swimming in a chlorinated environment on respiratory health of people regularly attending swimming pools. This review may help clinicians and trainers to better understand the mechanisms of airway disorders in swimmers and to become aware of the necessity for a proper management.

**Contributors** Both authors contributed equally to the redaction of this manuscript and the literature search. VB is the guarantor.

**Competing interests** None.

**Provenance and peer review** Commissioned; internally peer reviewed.

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