

Airways Disorders and the Swimming Pool

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KEYWORDS

• Swimming • Asthma • Chlorine • Exercise • Airways

KEY POINTS

- Physical activity has beneficial effects on the health of subjects with asthma. Among different sports swimming is the less asthmogenic.
- Although controversial, some authors suggested that regular chlorinated swimming pools attendance favors the development of allergic diseases and asthma in infants, young children, adolescents, and recreational swimmers.
- Lifeguards and competitive swimmers, who are frequently exposed to a chlorine environment, are particularly concerned by an increase in occupational asthma in the former, and airway disorders, especially airway hyperresponsiveness, rhinitis, and allergies.
- Intense swimming training in a chlorinated swimming pool on a recurrent basis may promote the development of a specific phenotype of asthmatic airways.
- Simple hygienic measures could help reduce the quantity of chlorine by-products released in the swimming pool environment, and possible airway disorders in swimmers.

INTRODUCTION

Swimming is a common physical activity worldwide for recreational and competitive purposes. In western countries, swimming is often part of schools sports curricula, especially for young children. Swimming is also recommended for those having a body-weight limitation to physical activity, elderly subjects, or those with disabilities or injury. The humid and warm atmosphere encountered in indoor pools is considered particularly beneficial for patients affected by respiratory diseases, such as asthma, and swimming is associated with less exercise-induced bronchoconstriction (EIB) than other types of sport.^{1,2}

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Physical activities performed in the water are predominantly performed in indoor swimming pools, partly for reasons related to the climate. Despite the high efficiency of chlorine and its derivatives to control nearly all viral and bacterial water contamination, there is controversy regarding the possible deleterious effects of chlorine disinfection on the development of asthma and allergic diseases.^{3,4} Indeed, increasing evidence suggests a relationship between regular attendance at chlorinated swimming pools and the development of airway injury, allergic diseases, and asthma.^{5–10} Although it is generally accepted that elite swimmers and lifeguards are the most likely to be affected by such exposure,^{11–21} the possibility that young children or recreational swimmers frequently exposed to such an environment will develop airway disorders remains controversial.^{3,4}

This article discusses the current benefits and risks of swimming in chlorine-disinfected swimming pools for subjects with and without asthma, and provides an overview of the current knowledge on the effects of indoor pool environments on the airways of swimmers.

BENEFITS OF SWIMMING IN SUBJECTS WITH ASTHMA OR EIB

EIB and exercise-induced asthma, which is evidenced by a fall in forced expiratory volume in 1 second (FEV₁) after exercises, can be observed in up to 90% of subjects with asthma.^{22,23} However, regular physical activity improves global health of these subjects, reduces exercise-induced asthma symptoms, and should be part of asthma management.²⁴ In subjects with asthma, swimming is associated with a lower frequency of EIB compared with running or cycling, when exercise is performed at same the level of intensity.^{1,25} For this reason, swimming is often recommended because of its lower asthmogenicity. The precise mechanism underlying this protective effect is still unclear but it is most likely related to the influence of high humidity and warm temperature of the air inhaled during exercise.^{1,25} Additional mechanisms have been proposed. For example the hydrostatic pressure shifts blood volume from the legs to the chest, possibly counteracting excessive respiratory heat and water loss.²⁶ Through chest compression, the immersion-related pressure may also help to reduce the expiratory effort.²⁶ Another mechanism is the bronchodilation induced by the CO₂ retention associated with hypoventilation secondary to a controlled breathing pattern during swimming.²⁷

Influence of Humid and Warm Environment on the Airways

The respiratory heat and water loss associated with high minute ventilation during exercise seems to be responsible for EIB in subjects who are hyperresponsive.^{1,25,28} Although a reduction of airway temperature may influence EIB,²² the osmotic consequences of respiratory water loss are thought to be most important.²⁹ The increase in airway osmolarity induces a movement of water across the airway epithelium to the airway surface, to replace the respiratory water loss. An increase in osmolarity in the local environment favors the release of inflammatory mediators, such as histamine, prostaglandins, and leukotrienes from inflammatory cells. It is these mediators that are thought to cause bronchoconstriction. Both ventilation and degree of humidification of air inhaled during exercise determine the degree of water loss from the airways and thus the chance of having EIB. Reducing water loss from the airways decreases the possibility of EIB. On average, air humidity of swimming pools is about 60% (and likely close to full saturation at the surface of the water), whereas mean air temperature is usually around 27°C.^{13,30} Inhaling such humid and warm air during swimming can protect against EIB or reduce its magnitude. Although this has been debated,³¹ swimming

remains less asthmogenic compared with running when minute ventilation and the water content of inspired air during exercise are similar.^{22,25} At the same ventilatory rate, when the air is dry and less than 10 mg/H₂O per liter, treadmill running but not swimming generally induces EIB in subjects with asthma.²⁵

Influence of Hydrostatic Pressure, Body Immersion, and Breathing Pattern

The hydrostatic pressure exerts compression on the chest, which facilitates expiration,³² and suppresses the dependence on gravity, facilitating the return of venous blood from the legs to the chest.³³ The shift of blood volume is thought to play a role in modulating heat exchange and EIB.²⁶ This was demonstrated by Gilbert and colleagues²⁶ who studied the influence of wearing antishock trousers (which cause blood to shift acutely from the legs to the thorax on the airway response) to 4-minute hyperpnea challenge. Whereas no change in FEV₁ was observed in control subjects after wearing or not wearing antishock trousers (mean posttest FEV₁ fall, $2 \pm 1\%$), in subjects with asthma, a significant reduction of bronchoconstriction was observed (the mean posttest FEV₁ fall was 21% without and 3% with antishock trousers). The authors suggested that the shift of blood from the legs to the thorax attenuated hyperpnea-induced bronchoconstriction because of a concomitant change in airway blood supply, which increases airstream temperature and disrupts thermal gradient that develops after hyperpnea. However, the recumbent position also increases the thoracic venous return yet it provides no protective effect against EIB.³⁴

A controlled breathing pattern caused by the different types of swimming, the mouth being frequently under the water surface except in backstroke, and characterized by a prolonged expiration, a short duration of inspiration, and hypoventilation may induce CO₂ retention.²⁷ The resulting hypercapnia can then induce a bronchodilation protecting from development of EIB.²⁷

Swimming Programs in Subjects with Asthma

Few studies have been conducted on the influence of physical activity programs in swimming pools on severity and frequency of asthma exacerbations. Generally, the authors recommend a 2-month physical retraining with 2- or 3-weekly sessions lasting 30 to 45 minutes, preferentially as interval training.^{35–42} The intensity of exercise varies from one study to another, but exercising at the load at which ventilation and carbon dioxide increase disproportionately to oxygen uptake (first ventilatory threshold) combined with high-intensity periods has been shown to be well-tolerated for rehabilitating people with respiratory diseases.^{37,39} Swimming programs in subjects with asthma lead to an improvement in aerobic capacity, with a concomitant increase in the workload required to provoke EIB, a decrease in the severity of asthma exacerbations,^{36,40} hospitalization rate or unscheduled physician visits,⁴² daily medication,^{36,38} school absenteeism,⁴⁰ and improved quality of life.^{39,40} Airway responsiveness may be slightly reduced after swimming programs⁴² or unchanged.^{36,38} The benefits are probably not caused by swimming itself, but rather by the effects of participation in an exercise program, as suggested by studies showing improvements with non-swimming-related training activities.⁴³ Moreover, there is a possible influence on the airways of an increased cholinergic tone caused by the immersion of the face in cold water (diving reflex).⁴⁴ A positive correlation has also been observed between the decrease in heart rate caused by the diving reflex and the dose of methacholine inducing a 45% fall in airway-specific conductance.⁴⁴ The increased cholinergic tone observed in asthma may play a role in the bronchoconstriction during face immersion in cold water.⁴⁵

Subjects with asthma who would like to perform swimming activities are often encouraged to do so in a warm and humid environment. Physical activities other

than swimming, however, may also be beneficial and could be selected according to subjects' preference. The potential problem of exposure to airborne irritants resulting from water chlorination has not yet been fully addressed and the long-term effects of swimming training programs need to be better documented.^{36,40,41} Subjects with asthma often report respiratory symptoms and may develop airway hyperresponsiveness (AHR) after attendance at a chlorinated pool,^{46–48} but swimming is less potent than other types of exercises in provoking bronchoconstriction. It should be stressed that most sports are well tolerated when medication is taken for prevention of EIB (although if asthma is well controlled, the athletes rarely need a short-term preventative medication, such as inhaled short-acting β_2 agonists).

EFFECTS OF CHLORINE-DISINFECTED SWIMMING POOL ENVIRONMENT IN CHILDREN AND RECREATIONAL SWIMMERS

The interaction of chlorine with organic material and ammonium introduced into the pool water by bathers, such as sweat, urine, soap residues, and cosmetics, produces a variety of disinfection by-products (DBPs).^{30,49} Some DBPs are irritating for the airways, whereas others are suspected of having carcinogenic potential with repeated exposure to high concentrations.⁴⁹ The agents most often described as potential inducers of respiratory disorders are gaseous trichloramines (NCl_3). The aerosols of these products, just above the pool water, may also be detrimental but there are few data available on the effects of these compounds.^{49–52} The levels of DBPs in the air depend on several parameters, such as the quantity of organic matter brought by bathers into the water, the efficiency of the ventilation, water renewal, and water characteristics.⁴⁹

Chronic Effects

The acute toxicity from inhaling high concentrations of chlorine and its by-products has been recognized for a long time.^{53–60} Evidence that low-dose exposure to chlorine products may chronically damage the respiratory tract of elite swimmers, lifeguards, and pool workers is also increasing.^{11–21} Whether these products affect recreational swimmers and children regularly attending chlorinated pools is, however, still controversial. A few recent cross-sectional studies involving child and adolescent recreational swimmers or adult swimmers have suggested a relationship between chlorinated swimming pool attendance and the development of asthma or allergic diseases.^{5,8–10,61–64} In those attending swimming pools during infancy, a higher risk of bronchiolitis, which increases the risk of asthma and allergic sensitization, has also been suggested,^{7,65} although not confirmed.⁶⁶ These findings, however, do not allow a conclusion to be made in favor of an association between attendance at swimming pools by recreational swimmers and the development of asthma or allergic diseases. To the contrary, a recent prospective longitudinal study performed in 5738 British children suggested that swimming did not increase the risk of asthma or allergic symptoms.⁶⁷ Furthermore, in this last study, swimming was associated with improved lung function and a lower risk of asthma symptoms, especially among children with preexisting respiratory conditions. These controversial data stress the need for further research on the role of swimming pool attendance in the development of allergic diseases and asthma. Importantly, studies should be comparable and use similar inclusion criteria, methodology, and statistical models to avoid inconsistencies. For example, hygiene measures for swimming pool management vary among the countries and can lead to different results.

The "Chlorine Asthma Hypothesis"

Carbonnelle and colleagues⁶ described a transient epithelial injury after a 1-hour swimming session in a chlorinated environment in recreational swimmers, children, or passively exposed attendees. As proposed by Bernard,⁵² such epithelial injury could allow allergens to easy access to antigen-presenting cells, and possibly promote allergen sensitization (Fig. 1). It is possible that the development of allergic diseases and asthma could be facilitated by repeated attendance at chlorinated swimming pools in association with allergen exposure. The time of recovery from the epithelium injury is short (12–24 hours)⁶ and it remains to be determined whether recreational swimmers training twice a week or more are at risk of developing allergic diseases or asthma. There is nevertheless a need to improve disinfection techniques and the hygiene and ventilation in indoor swimming pools to minimize chlorine by-product inhalation and allow bathers to swim without significant risk. Further studies should also consider swimming-induced rhinitis in a chlorinated environment, a frequent problem that remains insufficiently studied.

ELITE SWIMMERS' AIRWAYS

Swimmers, including synchronized swimmers, may train more than 30 hours per week, and, if so, they belong to the athletic population with the highest number of annual training hours. Consequently, they are particularly exposed to by-products of chlorine-disinfection, especially NCl_3 , which they probably inhale in high concentrations from the air just above the water. It has been suggested that the increased

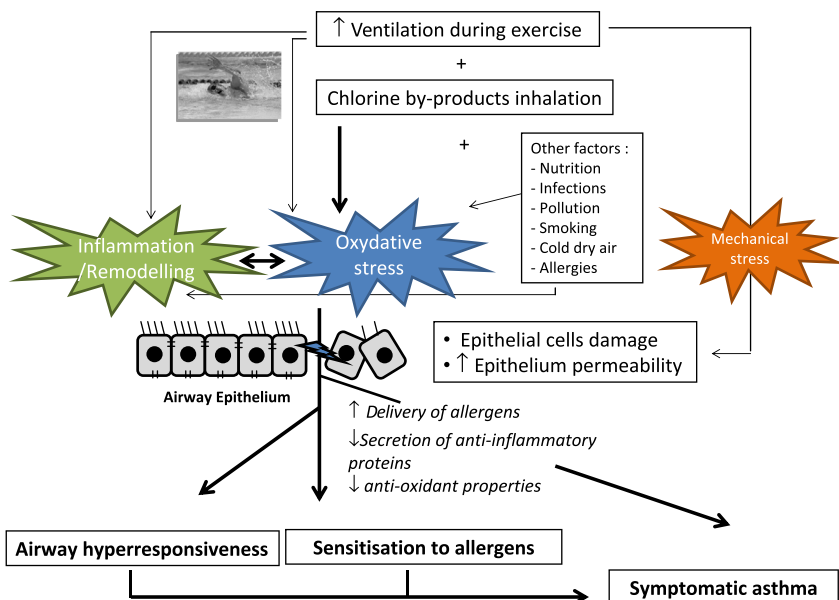


Fig. 1. Simplified hypothesis on the combined roles of inhalation of chlorine by-products and exercise in allergic sensitization, airway hyperresponsiveness, and asthma development. **Bold lines** represent the additional effect of chlorine by-products on exercise. (Data from Bernard A. Chlorination products: emerging links with allergic diseases. *Curr Med Chem* 2007;14:1689–99.)

prevalence of allergy, respiratory symptoms, and AHR in swimmers who compete is caused by the combined effects of DBP inhalation and the many hours per week of training.^{11,13,15}

Prevalence of Airway Disorders and EIB in Swimmers

Swimmers are affected by lower and upper respiratory symptoms. During the training season, 74% of competitive swimmers complain of nasal obstruction, rhinorrhea, sneezing, or itching.^{68,69} Screening of competitive swimmers has also demonstrated that up to 76% may have AHR or EIB when measured in the laboratory.^{11,12} The prevalence of asthma before starting a competitive career seems to be similar or slightly increased in swimmers, compared with athletes performing other sports or healthy subjects. This suggests that most commonly, swimmers do not begin swimming because of asthma, but rather develop respiratory disorders associated with asthma during their athletic career. It is the recreational swimmers or children, rather than the swimmers in training, who complain most often of respiratory symptoms attributed to a strong chemical odor of chlorine.^{13,70} Asymptomatic AHR or EIB seems to occur more frequently in swimmers compared with athletes performing other sports,¹⁵ which raises the question of symptom perception in competitive swimmers and the clinical significance of AHR or EIB as documented in a laboratory environment.

Significance of AHR and EIB

Several tests may be performed to diagnose EIB: exercise itself; eucapnic voluntary hyperpnea of dry air (EVH); and inhalation of hyperosmolar aerosols, or mannitol (discussed elsewhere in this issue). The choice of the bronchoprovocation test is often made according to availability, specificity, and sensitivity to identify potential for EIB. Castircum and colleagues⁷¹ reported that among 55% of swimmers who had a positive diagnosis for EIB in response to EVH, only 3% had EIB after swimming 8 minutes at 85% of maximal heart rate or more. The authors concluded that EVH challenge was a highly sensitive test for identifying potential for EIB in elite swimmers, in contrast to the swimming challenge test. The positive response to EVH observed in swimmers may be secondary to an incomplete recovery of the airway epithelium from the injury provoked by the cumulative effects of intense training in a chlorinated environment. Because swimmers are not likely to experience significant dehydration of the airways during swimming, a positive EVH test may indicate an intermediary state between health and asthma, possibly a preclinical stage of asthma.⁷² Further studies are needed to clarify the mechanisms and the significance of a positive bronchial provocation test in swimmers.

Mechanisms for Airway Disorders in Swimmers

Why swimmers have a high prevalence of AHR, as measured by various bronchoprovocation tests, is poorly understood. This AHR may develop after combined effects of inhaling by-products of chlorine and the mechanical stress caused by repeated high-level ventilation sustained during several hours of training per day.⁷³

AIRWAY INFLAMMATION AND EPITHELIAL DAMAGE: IS CHLORINE GUILTY?

The recent chlorine hypothesis suggests a link between exposure to by-products of chlorine and allergic diseases or asthma in industrialized countries.⁵² Inhaled by-products of chlorine have the potential to interact with airway epithelium resulting in oxidative stress and airway inflammation. When inhalation of these by-products

occurs repeatedly in elite swimmers it may result in an impairment of antioxidant activity in the airways,⁷⁴ and contribute to the development of allergic diseases,⁵² increased airway responsiveness,^{11–15} or symptomatic asthma.^{14,75,76} Inflammatory and remodeling processes have been observed in the bronchial mucosa of competitive swimmers when compared with nonathletes with mild asthma.⁷⁶ The structural changes observed in the bronchi of swimmers were independent of the documentation of AHR.⁷⁶ We consider from our observations and others that intense swim training in a chlorinated swimming pool on a recurrent basis may promote the development of a specific phenotype of asthmatic airways in elite swimmers.

Despite the evidence implicating inhalation of chlorine by-products in the development of airway disorders, the observed structural changes and hyperresponsiveness may simply reflect a normal adaptive response to intense training. Indeed, remodeling and inflammation of the airways have also been observed in bronchial biopsies of elite skiers.⁷⁷ In elite skiers repeated dry air hyperpnea may cause structural changes, and evidence favors a high minute ventilation for inducing epithelial damage as reflected by markers of increased airway permeability.^{6,78}

Upper and Lower Airways Interaction

Respiratory symptoms originating from the upper airways are very common in swimmers.^{79,80} In a recent study of nasal cytology, Gelardi and colleagues⁶⁸ observed nasal inflammation of a neutrophilic type in most symptomatic elite swimmers, and they attributed this to the irritant effect of chlorine. After 30-days using a nose-clip during swimming, nasal symptoms were significantly reduced, particularly in those with neutrophilic inflammation. This finding highlights the role played by the by-products of chlorine on the nasal mucosa, either by inhaled gases or by the chlorinated water directly entering the nose. It is likely that the effects of these by-products are similar on the upper and lower airway epithelium.⁸¹

Clinical Consequences and Possible Effects on Performance

Rhinitis has been shown to affect the quality of life of swimmers during an intense period of training, and may thus have an impact on performance.⁶⁹ Elite swimmers with a diagnosis of asthma perform as well as or even better than those without asthma during competitions.⁸² Although the potential for EIB, as documented by a positive response to EVH, is high among swimmers, most competitive swimmers do not report respiratory symptoms to their physician.¹⁵ It has been suggested that they consider symptoms a normal phenomenon caused by intense training or the presence of chlorine, and do not report them because they do not occur during swimming but rather at another time. A positive response to EVH reveals potential for EIB, and EIB may not necessarily occur after a swimming test or session. Some swimmers, however, may have EIB and be asymptomatic when exercising in the pool environment. Observations from a French study showed a significant reduction in airway responsiveness in eight competitive swimmers after the annual swimming pool cleaning compared with before the cleaning.⁸³ Moreover, in swimmers AHR/EIB and rhinitis can be transient and a partial remission has been observed after a period of rest as short as 2 weeks without swimming (Fig. 2).^{69,84} During a 5-year follow-up study, a significant attenuation of airway responsiveness was reported in 26 elite swimmers after cessation of training for at least 3 months, whereas no change was found in those swimmers who were still involved in competitive swimming.⁸⁵ Whether inflammation and remodeling of the airways observed during a swimmer's career may be reversible remains to be determined.

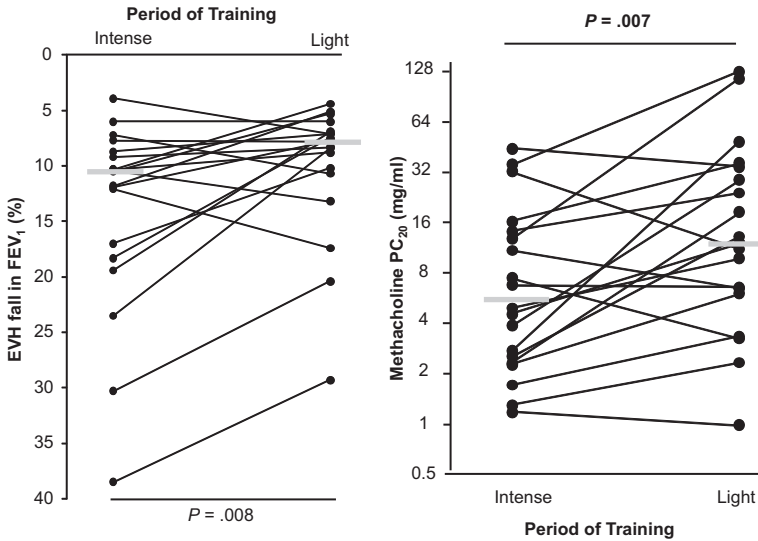


Fig. 2. Individual changes in percentage fall in FEV₁ after eucapnic hyperpnea challenge (left) and in methacholine PC₂₀ (right) according to the training period in 19 competitive swimmers. EVH, eucapnic voluntary hyperpnea of dry air. (Data from Bougault V, Turmel J, Boulet LP. Airway hyperresponsiveness in elite swimmers: Is it a transient phenomenon? *J Allergy Clin Immunol* 2011;127:892–8.)

SPECIFIC MANAGEMENT OF AIRWAY DISORDERS AND PREVENTION IN SWIMMERS

Management of asthma in athletes is mainly based on current guideline recommendations,^{86,87} and is the same for swimmers as for other athletes. However, whether one should treat asymptomatic swimmers with increased airway responsiveness to a bronchial provocation test as one would for a patient with asthma is still debated and currently difficult to answer. Obviously, the best measure to reduce the potential impact of swimming in chlorinated swimming pools on the airways should be to reduce the release of chlorine by-products into the ambient air of the pool. Because NCl₃ levels in the air above swimming pool water are influenced by the interaction between bathers' hygiene and water chlorine by-products, a reduction of human protein matter in the water may lessen respiratory health problems of swimmers. To do so, simple hygienic measures should be taken by the bathers, such as to take a shower and take off make-up and other cosmetics before entering the pool, to wear a swim cap, not to urinate in the water, and to use a swimsuit reserved exclusively for swimming.⁸⁸ To reduce exposure to volatile chemical contaminants, the concentration in water should be controlled and there should be adequate air flow across water surface (from forced or natural ventilation) and adequate amounts of fresh air.

One may ask if chlorine disinfection should be replaced by another method. Indeed, alternative chemical disinfectants, such as ozone, copper-silver, and bromine-based products, are being used increasingly. Bromine-based disinfectants are an alternative to chlorination,⁸⁹ whereas ozone and ultraviolet irradiation may be useful to eliminate chlorine by-products or reduce the quantity of chlorine required for disinfection.^{49,90–92} Copper-silver ionization is also used in some countries and seems to have less deleterious effects on the airways.^{9,93} Each disinfectant, however, produces a variety of by-products and has variable efficacy in eliminating microorganisms. The properties

of each disinfectant should therefore be assessed more thoroughly to ensure sufficient water disinfection, particularly in high-attendance pools, while avoiding untoward consequences on the airways.

WHAT IS NEXT?

Further studies should be conducted on the specific effects of DBPs on the airways of elite swimmers, and initiated in children and recreational swimmers. The impact of airway disorders on athletic performance and on the health of swimmers during and after their training career should be evaluated. The management of swimming pools can be improved to ensure proper disinfection of the water while also reducing the DBP released. Comparisons of the effects of chlorinated and nonchlorinated swimming pools on the airways and the effects of chlorinated pools with or without the use of strict rules of hygiene should also be promoted.

SUMMARY

Swimming is a healthy activity and is well tolerated by most subjects with asthma. This is likely because of the high humidity of the ambient air that results in less heat and water loss from the airways during exercise reducing the risk of experiencing EIB. For this reason swimming is frequently recommended to subjects with asthma to improve their aerobic capacity and quality of life. However, there is increasing evidence of an impact from exposure to by-products of chlorine on the development of airway disorders, including asthma, AHR, rhinitis, and allergic diseases. Elite swimmers are particularly concerned because the prevalence of airway disorders is the highest among this group compared with other athletes. Both the high ventilation of exercise and the inhalation of chlorine by-products are likely to contribute to the development of these disorders. Further studies are needed to characterize more specifically the role of by-products of chlorine on the airways and the development of the structural changes in the airways that are observed in swimmers.

REFERENCES

1. Bar-Yishay E, Gur I, Inbar O, et al. Difference between swimming and running as stimuli for exercise-induced asthma. *Eur J Appl Physiol* 1982;48:387–97.
2. Bar-Or O, Inbar O. Swimming and asthma. *Sports Med* 1992;16:397–405.
3. Piacentini GL, Baraldi E. Pro: swimming in chlorinated pools and risk of asthma: we can now carry on sending our children to swimming pools! *Am J Respir Crit Care Med* 2011;183:569–70.
4. Bernard A, Voisin C, Sardella A. Con: respiratory risks associated with chlorinated swimming pools: a complex pattern of exposure and effects. *Am J Respir Crit Care Med* 2011;183:570–2.
5. Bernard A, Nickmilder M, Voisin C, et al. Impact of chlorinated swimming pool attendance on the respiratory health of adolescents. *Pediatrics* 2009;124:1110–8.
6. Carbonnelle S, Francaux M, Doyle I, et al. Changes in serum pneumoproteins caused by short-term exposures to nitrogen trichloride in indoor chlorinated swimming pools. *Biomarkers* 2002;7:464–78.
7. Voisin C, Sardella A, Marcucci F, et al. Infant swimming in chlorinated pools and the risks of bronchiolitis, asthma and allergy. *Eur Respir J* 2010;36:41–7.
8. Bernard A, Nickmilder M, Voisin C. Outdoor swimming pools and the risks of asthma and allergies during adolescence. *Eur Respir J* 2008;32:979–88.

9. Bernard A, Carbonnelle S, Dumont X, et al. Infant swimming practice, pulmonary epithelium integrity, and the risk of allergic and respiratory diseases later in childhood. *Pediatrics* 2007;119:1095–103.
10. Chaumont A, Voisin C, Sardella A, et al. Interactions between domestic water hardness, infant swimming and atopy in the development of childhood eczema. *Environ Res* 2012;116:52–7.
11. Langdeau JB, Turcotte H, Bowie DM, et al. Airway hyperresponsiveness in elite athletes. *Am J Respir Crit Care Med* 2000;161:1479–84.
12. Zwick H, Popp W, Budik G, et al. Increased sensitization to aeroallergens in competitive swimmers. *Lung* 1990;168:111–5.
13. Potts JE. Adverse respiratory health effects of competitive swimming: the prevalence of symptoms; illness, and bronchial responsiveness to methacholine and exercise [dissertation]. Vancouver (British Columbia): University of British Columbia; 1994.
14. Helenius IJ, Ryttilä P, Metso T, et al. Respiratory symptoms, bronchial responsiveness, and cellular characteristics of induced sputum in elite swimmers. *Allergy* 1998;53:346–52.
15. Bougault V, Turmel J, Boulet LP. Bronchial challenges and respiratory symptoms in elite swimmers and winter sport athletes: airway hyperresponsiveness in asthma: its measurement and clinical significance. *Chest* 2010;138:31S–7S.
16. Massin N, Bohadana AB, Wild P, et al. Respiratory symptoms and bronchial responsiveness in lifeguards exposed to nitrogen trichloride in indoor swimming pools. *Occup Environ Med* 1998;55:258–63.
17. Jacobs JH, Spaan S, van Rooy GB, et al. Exposure to trichloramine and respiratory symptoms in indoor swimming pool workers. *Eur Respir J* 2007;29:690–8.
18. Nemery B, Hoet PH, Nowak D. Indoor swimming pools, water chlorination and respiratory health. *Eur Respir J* 2002;19:790–3.
19. Thickett KM, McCoach JS, Gerber JM, et al. Occupational asthma caused by chloramines in indoor swimming-pool air. *Eur Respir J* 2002;19:827–32.
20. Nordberg GF, Lundstrom NG, Forsberg B, et al. Lung function in volunteers before and after exposure to trichloramine in indoor pool environments and asthma in a cohort of pool workers. *BMJ Open* 2012;2(5). pii:e000973.
21. Fantuzzi G, Righi E, Predieri G, et al. Airborne trichloramine (NCl(3)) levels and self-reported health symptoms in indoor swimming pool workers: dose-response relationships. *J Expo Sci Environ Epidemiol* 2012. <http://dx.doi.org/10.1038/jes.2012.56>.
22. McFadden ER, Gilbert IA. Exercise-induced asthma. *N Engl J Med* 1994;330:1362–7.
23. Fitch K, Morton AR. Specificity of exercise in exercise-induced asthma. *Br Med J* 1971;4:577–81.
24. Ram FS, Robinson SM, Black PN, et al. Physical training for asthma. *Cochrane Database Syst Rev* 2005;(4):CD001116.
25. Inbar O, Dotan R, Dlin RA, et al. Breathing dry or humid air and exercise-induced asthma during swimming. *Eur J Appl Physiol* 1980;44:43–50.
26. Gilbert IA, Regnard J, Lenner KA, et al. Intrathoracic airstream temperatures during acute expansions of thoracic blood volume. *Clin Sci* 1991;81:655–61.
27. Donnelly PM. Exercise-induced asthma: the protective role of CO₂ during swimming. *Lancet* 1991;337:179–80.
28. Bar-Or O, Neuman I, Dotan R. Effects of dry and humid climates on exercise-induced asthma in children and preadolescents. *J Allergy Clin Immunol* 1977;60:163–8.

29. Anderson SD, Daviskas E. The mechanisms of exercise-induced asthma is.... *J Allergy Clin Immunol* 2000;106:453–9.
30. Drobnic F, Freixa A, Casan P, et al. Assessment of chlorine exposure in swimmers during training. *Med Sci Sports Exerc* 1996;28:271–4.
31. Bundgaard A, Schmidt A, Ingemann Hansen T, et al. Exercise-induced asthma after swimming and bicycle exercise. *Eur J Respir Dis* 1982;63:245–8.
32. Withers RT, Hamdorf PA. Effect of immersion on lung capacities and volumes: implications for the densitometric estimation of relative body fat. *J Sports Sci* 1989;7:21–30.
33. Park KS, Choi JK, Park YS. Cardiovascular regulation during water immersion. *Appl Human Sci* 1999;18:233–41.
34. Inbar O, Naiss S, Neuman E, et al. The effect of body posture on exercise- and hyperventilation- induced asthma. *Chest* 1991;100:1229–34.
35. Emtner M, Herala M, Stalenheim G. High intensity physical training in adults with asthma. *Chest* 1996;109:323–30.
36. Fitch KD, Morton AR, Blansky BA. Effects of swimming training on children with asthma. *Arch Dis Child* 1976;51:190–4.
37. Varray A, Mercier JG, Préfaut CG. Individualized training reduces excessive hyperventilation in asthmatics. *Int J Rehabil Res* 1995;18:297–312.
38. Weisgerber MC, Guill M, Weisgerber JM, et al. Benefits of swimming in asthma: effect of a session of swimming lessons on symptoms and PFTs with review of the literature. *J Asthma* 2003;40:453–64.
39. Weisgerber M, Webber K, Meurer J, et al. Moderate and vigorous exercise programs in children with asthma: safety, parental satisfaction, and asthma outcomes. *Pediatr Pulmonol* 2008;43:1175–82.
40. Wardell CP, Isbister C. A swimming program for children with asthma. Does it improve their quality of life? *Med J Aust* 2000;173:647–8.
41. Matsumoto I, Araki H, Tsuda K, et al. Effects of swimming training on aerobic capacity and exercise-induced bronchoconstriction in children with bronchial asthma. *Thorax* 1999;54:196–201.
42. Wicher IB, Ribeiro MA, Marmo DB, et al. Effects of swimming on spirometric parameters and bronchial hyperresponsiveness in children and adolescents with moderate persistent atopic asthma. *J Pediatr (Rio J)* 2010;86:384–90.
43. Chandrilleke MG, Carson KV, Picot J, et al. Physical training for asthma. *Cochrane Database Syst Rev* 2012;(5):CD001116.
44. Sturani C, Sturani A, Tosi I. Parasympathetic activity assessed by diving-reflex and by airway response to methacholine in bronchial asthma and rhinitis. *Respiration* 1985;48:321–8.
45. Kallenbach JM, Webster T, Dowdeswell R, et al. Reflex heart rate control in asthma. Evidence of parasympathetic overactivity. *Chest* 1985;85:644–8.
46. Penny PT. Swimming pool wheezing. *Br Med J (Clin Res Ed)* 1983;287:461–2.
47. Mustchin CP, Pickering CA. “Coughing water”: bronchial hyperreactivity induced by swimming in a chlorinated pool. *Thorax* 1979;34:682–3.
48. Stav D, Stav M. Asthma and whirlpool baths. *N Engl J Med* 2005;353:1635–6.
49. World Health Organization. International programme on chemical safety, environmental health criteria 216: disinfectants and disinfectant by-products. Geneva (Switzerland): WHO; 2000.
50. Keuten MG, Schets FM, Schijven JF, et al. Definition and quantification of initial anthropogenic pollutant release in swimming pools. *Water Res* 2012;46:3682–92.

51. Varraso R, Massin N, Hery M, et al. Not only training but also exposure to chlorinated compounds generates response to oxidative stimuli in swimmers. *Toxicol Ind Health* 2002;18:269–78.
52. Bernard A. Chlorination products: emerging links with allergic diseases. *Curr Med Chem* 2007;14:1689–99.
53. Agabiti N, Ancona C, Forastiere F, et al. Short term respiratory effects of acute exposure to chlorine due to a swimming pool accident. *Occup Environ Med* 2001;58:399–404.
54. Lemiere C, Malo JL, Boutet M. Reactive airways dysfunction syndrome due to chlorine: sequential bronchial biopsies and functional assessment. *Eur Respir J* 1997;10:241–4.
55. Parimon T, Kanne JP, Pierson DJ. Acute inhalation injury with evidence of diffuse bronchiolitis following chlorine gas exposure at a swimming-pool. *Respir Care* 2004;49:291–4.
56. Deschamps D, Soler P, Rosenberg N, et al. Persistent asthma after inhalation of a mixture of sodium hypochlorite and hypochloric acid. *Chest* 1994;105:1895–6.
57. Martinez TT, Long C. Explosion risk from swimming pool chlorinators and review of chlorine toxicity. *J Toxicol Clin Toxicol* 1995;33:349–54.
58. Bonetto G, Corradi M, Carraro S, et al. Longitudinal monitoring of lung injury in children after acute chlorine exposure in a swimming-pool. *Am J Respir Crit Care Med* 2006;174:545–9.
59. Ploysongsang Y, Beach BC, DiLisio RE. Pulmonary function changes after acute inhalation of chlorine gas. *South Med J* 1982;75:23–6.
60. D'Alessandro A, Kuschner W, Wong H, et al. Exaggerated responses to chlorine inhalation among persons with non-specific airway hyperreactivity. *Chest* 1996; 109:331–7.
61. Cotter A, Ryan CA. The pool chlorine hypothesis and asthma among boys. *Ir Med J* 2009;102:79–82.
62. Kohlhammer Y, Döring A, Schäfer T, et al. Swimming pool attendance and hay fever rates later in life. *Allergy* 2006;61:1305–9.
63. Font-Ribera L, Kogevinas M, Zock JP, et al. Swimming pool attendance and risk of asthma and allergic symptoms in children. *Eur Respir J* 2009;34:1304–10.
64. Ferrari M, Schenk K, Mantovani W, et al. Attendance at chlorinated indoor pools and risk of asthma in adult recreational swimmers. *J Sci Med Sport* 2011;14: 184–9.
65. Jacobs JH, Fuertes E, Krop EJ, et al. Swimming Pool attendance and respiratory symptoms and allergies among Dutch children. *Occup Environ Med* 2012;69: 823–30.
66. Schoefer Y, Zutavern A, Brockow I, et al. Health risks of early swimming pool attendance. *Int J Hyg Environ Health* 2008;211:367–73.
67. Font-Ribera L, Villanueva CM, Nieuwenhuijsen MJ, et al. Swimming pool attendance, asthma, allergies, and lung function in the Avon Longitudinal Study of Parents and Children cohort. *Am J Respir Crit Care Med* 2011; 183:582–8.
68. Gelardi M, Ventura MT, Fiorella R, et al. Allergic and non-allergic rhinitis in swimmers: clinical and cytological aspects. *Br J Sports Med* 2012;46:54–8.
69. Bougault V, Turmel J, Boulet LP. Effect of intense swimming training on rhinitis in high-level competitive swimmers. *Clin Exp Allergy* 2010;40:1238–46.
70. Levesque B, Duchesne JF, Gingras S, et al. The determinants of prevalence of health complaints among young competitive swimmers. *Int Arch Occup Environ Health* 2006;80:32–9.

71. Castricum A, Holzer K, Brukner P, et al. The role of the bronchial provocation challenge tests in the diagnosis of exercise-induced bronchoconstriction in elite swimmers. *Br J Sports Med* 2010;44:736–40.
72. Boulet LP. Asymptomatic airway hyperresponsiveness: a curiosity or an opportunity to prevent asthma? *Am J Respir Crit Care Med* 2003;167:371–8.
73. Tschumperlin DJ, Drazen JM. Chronic effects of mechanical force on airways. *Annu Rev Physiol* 2006;68:563–83.
74. Bougault V, Morissette M, Murray N, et al. Oxidative stress in swimmers' airways [abstract]. *Eur Respir J* 2010;36(Suppl 54):5582.
75. Bougault V, Turmel J, St Laurent J, et al. Asthma, airway inflammation and epithelial damage in swimmers and cold-air athletes. *Eur Respir J* 2009;33:740–6.
76. Bougault V, Loubaki L, Joubert P, et al. Airway remodeling and inflammation in competitive swimmers with and without airway hyperresponsiveness. *J Allergy Clin Immunol* 2012;129:351–8.
77. Karjalainen EM, Laitinen A, Sue-Chu M, et al. Evidence of airway inflammation and remodeling in ski athletes with and without bronchial hyperresponsiveness to methacholine. *Am J Respir Crit Care Med* 2000;161:2086–91.
78. Nanson CJ, Burgess JL, Robin M, et al. Exercise alters serum pneumoprotein concentrations. *Respir Physiol* 2001;127:259–65.
79. Deitmer T, Scheffler R. Nasal physiology in swimmers and swimmers' sinusitis. *Acta Otolaryngol* 1990;110:286–91.
80. Ondolo C, Aversa S, Passali F, et al. Nasal and lung function in competitive swimmers. *Acta Otorhinolaryngol Ital* 2009;29:137–43.
81. Clearie KL, Vaidyanathan S, Williamson PA, et al. Effects of chlorine and exercise on the unified airway in adolescent elite Scottish swimmers. *Allergy* 2010;65:269–73.
82. Fitch KD. An overview of asthma and airway hyper-responsiveness in Olympic athletes. *Br J Sports Med* 2012;46:413–6.
83. Simon-Rigaud ML, Eechout C, Bourdin H, et al. Hyperréactivité bronchique et natation. Influence de l'entraînement en atmosphère chlorée. *Sci Sports* 1997;12:142–7.
84. Bougault V, Turmel J, Boulet LP. Airway hyperresponsiveness in elite swimmers: Is it a transient phenomenon? *J Allergy Clin Immunol* 2011;127:892–8.
85. Helenius IJ, Ryttilä P, Sarna S, et al. Effect of continuing or finishing high-level sports on airway inflammation, bronchial hyperresponsiveness, and asthma. *J Allergy Clin Immunol* 2002;109:962–8.
86. Anderson SD, Kippelen P. Assessment and prevention of exercise-induced bronchoconstriction. *Br J Sports Med* 2012;46:391–6.
87. Bonini S, Bonini M, Bousquet J, et al. Rhinitis and asthma in athletes: an ARIA document in collaboration with GA2LEN. *Allergy* 2006;61:681–92.
88. Bougault V, Boulet LP. Airway dysfunction in swimmers. *Br J Sports Med* 2012;46:402–6.
89. Lourencetti C, Grimalt JO, Marco E, et al. Trihalomethanes in chlorine and bromine disinfected swimming pools: air-water distributions and human exposure. *Environ Int* 2012;15:59–67.
90. Lee J, Jun MJ, Lee MH, et al. Production of various disinfection byproducts in indoor swimming pool waters treated with different disinfection methods. *Int J Hyg Environ Health* 2010;213:465–74.
91. Wyatt TD, Wilson TS. A bacteriological investigation of two leisure centre swimming pools disinfected with ozone. *J Hyg (Lond)* 1979;82:425–41.

92. Weng S, Li J, Blatchley ER. Effects of UV 254 irradiation on residual chlorine and DBPs in chlorination of model organic-N precursors in swimming pools. *Water Res* 2012;46:2674–82.
93. Landeen LK, Yahya MT, Gerba CP. Efficacy of copper and silver ions and reduced levels of free chlorine in inactivation of legionella pneumophila. *Appl Environ Microbiol* 1989;55:3040–50.