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SWIMMING AGAINST THE CURRENT: ASTHMA AND UPPER AIRWAY DISORDERS IN COMPETITIVE SWIMMERS

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ABSTRACT

Purpose of the research: This review aims to assess the frequency of asthma and upper airway disorders in competitive swimmers, as well as identify potential environmental and training factors that contribute to the development of these conditions.
Materials and methods: The review of research literature was conducted through databases such as PubMed, Google Scholar, and Scopus, including studies from 2007 to 2025.
Results: Competitive swimmers have been shown to have a significantly higher incidence of exercise-induced asthma and chronic upper respiratory tract syndromes than in the general population. Particularly frequently observed cough, nasal congestion, wheezing, and bronchial hyperresponsiveness. There was also a correlation between the length of exposure to the swimming pool environment and the severity of the symptoms.
Conclusions: The environment of indoor swimming pools, especially exposure to chlorination byproducts, may contribute to the development of asthma and chronic upper respiratory tract diseases in swimmers. There is a need for further research and implementation of preventive measures, such as improving swimming pool ventilation and monitoring the health of athletes.

KEYWORDS

Asthma, Chlorine, Swimmer's Asthma, Disinfection Systems, Fungal Contamination, Allergens, Ventilation, Exercise-Induced Bronchoconstriction, EIB

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Introduction

Competitive swimming is a strenuous and endurance-based sport that demands not only outstanding physical condition but also precise movement coordination, a high level of breathing control, and long hours of intensive training.

In recent years, there has been a growing interest among scientists in the impact of the training environment on athletes' health. Swimmers spend a significant amount of time in the indoor pool environment, where they are regularly exposed to humidity and chemicals used to disinfect the water, such as chlorine and its derivatives. Despite the known benefits of swimming, this may cause health issues, especially in the respiratory system, including atopic asthma, allergic rhinitis, bronchial hyperresponsiveness [1], chronic airway inflammation [4], and other respiratory diseases.

This article aims to analyze the impact of the swimming pool environment on respiratory system diseases in competitive swimmers. The study aims to draw attention to health hazards resulting from long-term exposure to substances present in the pool environment and identify them. Understanding the risk factors is crucial for implementing disease prevention and health maintenance strategies among athletes.

Exercise-Induced Asthma

Asthma is a multifactorial condition characterized by chronic airway inflammation, reversible expiratory airflow limitation, and other respiratory symptoms, including wheezing, breathlessness, chest tightness, and cough. It affects approximately 339 million individuals worldwide. Sex differences in the prevalence, incidence, and severity of asthma have been well documented across life, with higher rates in males compared to females prior to puberty but opposite in adults. [25, 26]

Asthma is believed to impact 15–30% of Olympic athletes and a significant percentage of athletes in certain sports, including endurance athletes such as swimmers, distance runners, and cyclists. [25]

Exercise-induced bronchoconstriction (EIB) is a common issue for athletes, affecting both their performance and health. In sportspeople, especially at elite levels of competition, the prevalence of respiratory symptoms is surprisingly high, ranging from 20% to 70%, depending on the sport. The frequency rate also differs by sport, training area, and genetic background. [25,26,27]

Exercise is recognized as an indiscriminate stimulus for asthma, but ironically, it can also optimize lung function and reduce the severity of exercise-induced bronchoconstriction. In general, the physiological responses elicited by exercise differ between males and females, implying a relationship between sex, exercise, sex hormones, and atopic status in EIB pathophysiology. Participants exhibit individual-specific attributes, such as airway sensitivity, allergen and pollutant exposure, and temperature alterations. The pathogenesis of EIB involves intricate interactions between environmental and physiological factors. Drying and cooling of the airway are the critical mechanisms that contribute to osmotic and thermal hypotheses. Inflammation and hyper-responsiveness of the airway are shared factors.[25,26,27]

Top-level athletes can exhibit unique patterns of inflammation and increased airway sensitivity, which are influenced by the nature of the sport, training habits, and environmental factors. Swimming and other sports carry a high risk of EIB, with exposure to chlorine during swimming pool use as a significant risk factor. Inflammation, alteration in lung function, and interindividual variability play roles in EIB among athletes. [27]

Swimming elicits fewer respiratory symptoms than comparable endurance activities, such as running or cycling, at similar intensities. However, among professional swimmers, the frequency of respiratory symptoms, including wheezing, breathlessness, cough, and mucus production in the airways, is reportedly higher than in the general population. [25,26,27]

EIB pathophysiology

So far, however, the pathogenesis of EIB remains to be identified, but it is likely to be a consequence of physiological changes that occur in the lungs during exercise. Minute ventilation, that is, the amount of air that goes in or comes out of an individual's lungs each minute, increases as an individual exercises. [25,26] Water loss that occurs during the phases of increased ventilation is one of the key predisposing factors toward bronchoconstriction. Thus, the humidity of breathing air during exertion is also a significant factor in determining EIB. Intense exercise induces hyperosmolar conditions by introducing dry air into the airway, resulting in compensatory water loss that leads to osmotic movement of fluid across the airway surface. [26] This hyperosmolar condition is the cause of mast cell degranulation, resulting in the release of mediators, which include primarily leukotrienes, as well as histamine, tryptase, and prostaglandins. Evidence suggests that a mannitol challenge can induce mast cell activation and the release of mediators of bronchoconstriction in both asthmatics and non-asthmatics, indicating that the hyperosmolarity of airway fluid is a shared stimulus for mast cell activation. Eosinophils are also activated to release other mediators such as leukotrienes. It is due to this, therefore, that it results in airway inflammation and bronchoconstriction, as well as stimulation of sensory nerves, resulting in the release of neurokinins that stimulate the secretion of the gel-forming mucin MUC5AC. [26] The results are substantiated by evidence from experiments that exercise type is not the dominant variable; instead, ventilation requirements and the humidity of the air being inspired are the primary determinants of bronchoconstriction's presence or absence, as well as its intensity. Therefore, the primary cause of bronchoconstriction in a predisposed patient is either dehydration during times of increased ventilation or administration of an osmotically effective agent. Surprisingly, changes in airway temperature due to exercise or other thermal phenomena are believed to have little impact on the degree of bronchoconstriction that results. [26] Specific causes that may lead to increased prevalence and frequency of asthma among elite swimmers include chemical exposure, water droplet microaspiration, hyperventilation, and dysautonomia. [28]

Chlorine derivatives

Chlorine compounds, including sodium hypochlorite and chlorinated isocyanuric acids, are employed as primary disinfectants in pool treatment. They react with other chemicals in the water to produce trihalomethanes, primarily chloroform, and haloacetic acids, such as trichloroacetic acid. They react with swimmers' nitrogenous compounds, such as sweat, urine, and saliva, to produce chloramines. [28] Acute chloramine exposure has been considered frequent, but is not commonly reported in various instances. Respiratory distress, lung function impairment, and alterations in exhaled air (elevated leukotrienes (LT) and reduced FENO) following accidental exposure to chlorine among children have been documented. Little data exist regarding the long-term effects of chloramine exposure. Swimmers breathe air which lies directly over

the water surface, where the average concentration of chlorine is 0.42 mg/m3. Different studies reveal that at levels greater than 0.5 mg/m3, it produces severe eye and respiratory issues in swimmers. For a 2-hour training session, they can be exposed to levels of chlorine (4–6 g) higher than the exposure limits for an 8-hour day for a worker. [28] Chloramine concentrations in the air have been linked to upper respiratory symptoms and atopy in cross-sectional studies among lifeguards and pool workers. Predicted cumulative long-term chloramine concentrations had no association with airway hyperresponsiveness or lung function. A direct correlation has been reported between exposure to nitrogen trichloride by swimmers, whether through longer training sessions or higher concentrations, and biological indicators of response to oxidative stress. This result suggests that the generation of reactive oxygen species is not only associated with training but also with exposure to chlorinated chemicals. [28]

Chlorine is the most popular and effective substance for disinfecting swimming pool water effectively and quickly. However, the quantity of chlorine used for disinfection and its quality are important. To measure the chlorine content of a swimming pool, the total chlorine, free chlorine, and bound chlorine are assessed. It is considered most important to measure free chlorine, which is the chlorine that persists in the water and is capable of immediate disinfection by oxidising undesirable substances that enter the water from the air or through pool users with dead skin. If chlorine levels are too low, undesirable microorganisms can proliferate. Standards for free chlorine are 0.3-0.6mg/l in public pools; in private pools, it is estimated that the standard may be up to 1.2mg/l. Bound chlorine is chlorine combined with the nitrogen particles with which it has bound in the water, derived from contaminants that have entered the water. In the case of clean water, without impurities, this type of chlorine is sometimes close to zero, while a high amount of chlorine indicates significant pollution. The characteristic odour of chlorine in swimming pool facilities indicates the presence of chloramines, which are formed by the combination of chlorine with dust, sebum, and sweat, and are indicative of the disproportion of free chlorine to bound chlorine. Total chlorine is a combination of free and bound chlorine.

Chlorine solution has a destructive effect on bacteria and viruses by destroying both intracellular structures and preventing cell survival processes from occurring, as it affects intracellular enzymes and degrades the cell walls of microorganisms. Among other things, chlorine is broken down into hypochlorous acid and hypochlorite ion. Chlorine not only has an antimicrobial effect but also improves water clarity by blocking elements such as magnesium, iron, and copper. Chlorine works best at a PH of 7.0-7.4. It must neutralize all the substances that humans can bring into the water, i.e., urine, fecal matter, serum, sweat, dust, residues from cosmetics used, dead skin, and, above all, microorganisms - one person can bring in a billion bacteria. For microorganisms, pool water is an ideal environment for growth and, if they multiply, for their numbers to continue increasing. The most well-known pathogen that can multiply in water and cause numerous diseases in humans is Escherichia Coli. Failure to chlorinate the water is associated with the formation of a characteristic film on the water surface after a specific period.

Despite the harmfulness of using chlorine as a disinfectant, it is the most common due to its favourable price. Other disinfection methods, such as ozone, copper-silver, bromine, and UV disinfection, have the disadvantage of higher prices but do not achieve the same disinfection efficiency as chlorine. Consequently, even disinfection by other methods still needs to be supplemented by disinfection with chlorine or copper. However, promisingly, disinfection with chlorine in combination with ozone is less toxic than disinfection with chlorine alone.

Amateur swimmers and professional swimmers alike are exposed to the harmful effects of chlorine on the body not only through inhalation of volatile or aerosolised substances, but also through ingestion of water and absorption through the skin.

A study in Belgium found that regular use of swimming pools increases the incidence of asthma. [30] A survey conducted among pool workers revealed that they frequently experience lower respiratory infections and a runny nose, and some workers also reported having an asthmatic condition. [31] Case reports have been described indicating the induction of asthma by exposure to noxious substances such as chloramine in high doses, in individuals without previous asthma. [5] The harmful effects of chlorine on the respiratory tract are due to chlorine by-products, which have a strong oxidising potential. They can disrupt the integrity of tight junctions by disrupting barrier continuity. [32] It has also been shown that airway permeability, including increased permeability to chlorine oxidation products, can result from hyperventilation during intense swim training. [33] Other complaints frequently reported by pool users include visual problems, such as redness and itching. [31] The skin and epidermis are also exposed to the damaging effects of chlorine, not least through the loss of natural moisture. Even in individuals with no prior skin problems, the training effect of chlorine has been demonstrated; skin irritation is more frequently reported, along with increased dryness of the skin, erythema, or itching. Worryingly, allergic contact dermatitis and urticaria have also been linked to chlorinated water. The skin and epidermis are also exposed to the damaging effects of chlorine, not least through the loss

of natural moisture. Even in individuals with no prior skin problems, the training effect of chlorine has been demonstrated; skin irritation is more frequently reported, along with increased dryness of the skin, erythema, or itching. Worryingly, allergic contact dermatitis and urticaria have also been linked to chlorinated water. In addition, those who have the most contact with water and air in the pool, i.e., lifeguards and swimming instructors, have been shown to have a higher incidence of warts, ringworm, and eczema compared to pool workers who do not have such close contact with water or air inhalation. [31] The effect of other pool disinfectants on the occurrence of rashes was investigated, and, surprisingly, bromine and ozone disinfection did not show a significant reduction in rashes compared to chlorine disinfection. Disinfection based on electrolysis with low salt concentrations had the most effective result. A study in mice showed that rodents subjected to prolonged vapour inhalation of chlorine were more likely to have exacerbations of pneumonia, as there was a mobilisation of pro-inflammatory macrophages into the lungs. [34] High-performance and synchronised swimmers are second among sports with an increased prevalence of asthma. This is influenced not only by the aquatic environment and chlorine inhalation, but also by the fact that they are exposed to these substances for prolonged periods, and, in addition, airway remodelling occurs in the long term due to prolonged inflammation. [35] Increased airway swelling is not only due to the higher prevalence of asthma among swimmers, but also vascular leakage, which is another cause of airway inflammation among swimmers. An association was found between the vascular permeability index and increased neutrophils and eosinophils. [36] Impaired mucociliary transport, mediated by products of chlorine metabolism, has been reported in athletes with increased exposure to water. In addition, the content of nasal fluid was studied in professional athletes, who had a higher number of neutrophils compared to non-athletes. Interestingly, these changes quickly disappeared with the use of a nose clip or cessation of training. However, TNF alpha concentrations in professional swimmers were similar to those in non-trainees.

Hyperventilation

Competitive swimmers take enormous volumes of air that exist at the immediate surface level of water during 30 hours of swimming every week. Chronic hyperventilation may result in mild airway narrowing due to water evaporation during the rapid conditioning of large volumes of inhaled air. In animal models of exercise-induced hyperpnea, prolonged hyperventilation caused airway inflammation, obstruction, damage to and repair of the bronchiolar epithelium, hyperreactivity, and reduced β-agonist-induced relaxation. [28] In nonhabituated swimmers, airway neutrophilia has been reported, suggesting a potential link with endurance training or aerosol inhalation. Unlike running, acute swimming in a non-chlorinated pool does not substantially alter airway cells. Training augmented small airway inflammatory cells, but inflammatory activation was not increased. This could represent variation in the air inhaled during exercise, as the moist atmosphere of swimming may inhibit the cooling of airways and the evaporation of water. [28]

Autonomic nerve control

Autonomic nerves provide for the contraction and relaxation of airway smooth muscle. Cholinergic-parasympathetic neurons provide contractions, and adrenergic-sympathetic and/or noncholinergic parasympathetic neurons provide relaxations. Extensive training in swimming may influence autonomic control and cardiac function, leading to enhanced vagal dominance. [28] Enhanced parasympathetic activity may serve as a counterbalancing response to the sympathetic stimulation induced by both ordinary and rigorous training. This might lead not only to resting bradycardia but also to heightened bronchomotor tone, thereby increasing the potential for asthma development. [28]

Inflammation characteristics

Earlier research on induced sputum from non-asthmatic elite swimmers revealed a higher percentage of eosinophils and neutrophils compared to healthy controls. Asthmatic swimmers revealed higher eosinophils and lymphocytes than healthy subjects, and a higher neutrophil count than other asthmatic subjects. Airway responsiveness was significantly elevated in asthmatic swimmers compared with healthy swimmers and asthmatic subjects. [28] This suggests that components of asthma in competitive swimmers have a dual etiology: allergic inflammation induces sputum eosinophilia and elevated FENO, whereas sputum neutrophils are associated with frequent exposure to the pool environment. Elevated bronchial reactivity and lymphocyte count are likely to be associated with both etiologies, as they occurred identically in athletes and asthma patients. [28]

Mold and Allergen Risks in Enclosed Swimming Facilities

Indoor swimming pools create a distinctive microenvironment characterized by persistently high humidity levels and often inadequate ventilation, both of which promote the growth and spread of molds and other airborne allergens. Poolside surfaces, ceilings, locker rooms, and hidden structural areas provide ideal conditions for fungi such as Alternaria, Aspergillus, and Cladosporium to proliferate. [20,21] Additionally, house dust mites can persist in damp materials, such as towels, carpeting, and ventilation systems, contributing to the allergenic burden within these unique microclimates. [23] The condition for mold growth is the presence of sufficient moisture in materials or on surfaces. Thus, high humidity, poor ventilation, and cold surfaces prone to condensation all promote fungal colonization. Structural deficiencies, leaks, rising damp, or accidental water damage can further elevate moisture levels, turning pool surfaces, walls, and inaccessible spaces into reservoirs for mold spores. Mold can grow on a wide range of indoor materials, including wood, paper, plastics, rubber, and within a broad temperature range, making it highly resilient. Critically, mold growth indoors is often a result of moisture issues; unless the facility managers address this root cause, long-term mold exposure is inevitable. Mold spores can also survive dry conditions that temporarily inhibit growth, ensuring persistence within the environment. [22]

Studies of indoor pools have consistently found key allergenic molds present, with Aspergillus, Alternaria, and Cladosporium among the most common genera identified in water samples and on damp surfaces. Other fungi, such as Acremonium, Fusarium, Penicillium, and Chaetomium, have also been isolated; however, their presence is typically sporadic and depends on the efficacy of water treatment and facility maintenance. [20,21] The table below presents the results of a study published in [Rezaei et al., 2022 [21]], which found that a total of 34 out of 88 collected water samples were contaminated with fungal elements. The research was conducted on water samples collected from 11 swimming pools in Zahedan city, of which seven were old and crowded. The prevalence of fungal contamination was 38.7 %. [21]

Table 1. [Rezaei et al., 2022 [21]] Frequency of fungal contamination in eleven pools.

	Type of Fungi			
Valid	Frequency	Percent	Valid Percent	Cumulative Percent
	54	61.3	61.3	61.3
Alternaria spp	2	2.28	2.28	63.58
Aspergillus Flavus	7	7.96	7.96	71.54
Aspergillus Fumigatus	4	4.55	4.55	76.09
Aspergillus Niger	1	1.14	1.14	77.23
Cladosporium spp	4	4.55	4.55	81.78
Geotrichum	2	2.28	2.28	84.06
Mucor spp	1	1.14	1.14	85.2
Rhizopus spp	3	3.41	3.41	88.61
Phialophora spp	3	3.41	3.41	92.02
Trichophyton spp	1	1.14	1.14	93.16
Sterile mycelium	3	3.41	3.41	96.57
Penicillium spp	1	1.14	1.14	97.71
Unknown	2	2.28	2.28	100.0
Total	88	100.0	100.0	

While effective chlorination can reduce waterborne fungal contamination, surfaces, air, and hidden structures often remain significant reservoirs for mold proliferation. [20] Chronic exposure to these airborne allergens has clear health implications. Repeated inhalation of mold spores and other allergens can increase the risk of developing allergic rhinitis, atopic asthma, and other respiratory conditions. Competitive swimmers,

who spend extended periods training in these environments, are particularly vulnerable due to both the duration and intensity of exposure. [18] Infants and children attending chlorinated pools also face heightened risks; research indicates a dose-dependent relationship between early-life swimming in chlorinated pools and the later development of asthma and other respiratory allergies. [19]

Moreover, the interaction between organic allergens and chlorine disinfection by-products, such as chloramines, can exacerbate airway inflammation. [17,18] While chlorination remains the most common method for maintaining water hygiene, by-products released into the air, especially in poorly ventilated indoor pools, can irritate the respiratory tract. This irritation may extend from the upper airways to the bronchioles and alveoli, leading to long-term changes in lung function and increasing the prevalence of airway disorders among regular swimmers. [24] Given these risks, physicians, coaches, and facility operators must recognize and mitigate these environmental hazards. Strategies should include optimizing ventilation, controlling humidity, enforcing proper hygiene measures, and monitoring air quality to limit the accumulation of chlorine by-products and airborne allergens. While alternative disinfection methods, such as ozone or ultraviolet radiation, show promise, each has its limitations and associated costs, making careful implementation and ongoing evaluation necessary.

The evidence suggesting a role for chlorine by-products and persistent allergens in the development of "asthmatic" airways in swimmers highlights the importance of preventive measures in protecting indoor air quality. [17,18] This issue extends beyond elite athletes and includes infants, lifeguards, pool staff, and individuals with pre-existing respiratory conditions who may choose swimming for its low-impact benefits. Recognizing and addressing this hidden respiratory burden is a crucial step toward protecting airway health in aquatic environments, setting the stage for broader discussions on the management and prevention of airway disorders among swimmers. [18]

Practical implications and prophylaxis

Effective prevention of respiratory conditions in competitive swimmers requires an integrative approach combining environmental engineering, medical supervision, and athlete education. These elements must converge to address the fundamental challenge of maintaining high indoor air and water quality.

Chlorine-based disinfectants (CBDs) are indispensable for microbial safety in pool water. However, they react rapidly with organic and inorganic contaminants (urea, sweat, dandruff, skin flakes, and residual pharmaceuticals or personal care products) to form chlorinated oxidants and disinfection by-products (DBPs) such as monochloramine (NH_2Cl), dichloramine ($NHCl_2$), trichloramine (NCl_3), trihalomethanes (THMs), and haloacetic acids (HAAs). [1, 2] Under conditions of poor ventilation, volatile and particle-bound disinfection by-products tend to accumulate within the breathing zone, contributing not only to acute mucosal and cutaneous irritation but also to the development of chronic airway inflammation and bronchial hyperresponsiveness. [1, 3] This risk is further amplified by the multiple routes of exposure as these compounds can be inhaled, accidentally ingested with pool water, or absorbed through the skin, cumulatively increasing the chemical exposure load on swimmers and exacerbating respiratory symptoms. [1, 2, 4, 5]

Environmental engineering measures

Efficient ventilation and advanced water-treatment protocols form the technical backbone of exposure mitigation. In practice, this means maintaining higher air-change rates and intelligent air-supply control to dilute volatile DBPs. For example, recent computational and field studies show that even American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) compliant ventilation (≈approximately 4–6 air changes per hour with moderate fresh intake) often fails to prevent the accumulation of chloramine (NCl_3) in the swimmer's breathing zone. In contrast, increasing outside-air fraction can dramatically reduce NCl_3 levels – one experiment found a 55% drop in breathing-zone trichloramine when switching from recirculated to 100% outdoor air. [6, 7]

Likewise, continuous CO_2 monitoring can serve as a real-time indicator of swimmer occupancy. Correlations have been observed between occupancy-driven CO_2 levels and concentrations of chlorine-based disinfectants (CBDs) and disinfection by-products (DBPs), supporting the use of CO_2-based ventilation control systems. Such systems adjust fresh-air supply according to user load, effectively reducing indoor peaks of harmful chemical by-products and improving air quality. [8]

Alongside ventilation improvements, water treatment strategies aim to eliminate precursor compounds. For instance, ultraviolet (UV) irradiation efficiently destroys combined chlorine residuals; however, it can also increase the formation of chloro-DBPs (such as THMs) on its own. [9] To prevent the unintended increase in disinfection by-products from UV treatment alone, ultraviolet irradiation is often followed by ozonation. This sequential approach enhances the reactivity of the water, allowing ozone to rapidly oxidize organic precursors and thereby suppress the formation of additional harmful by-products. [9] Similarly, advanced oxidation processes (AOPs) boost contaminant removal while generating fewer DBPs. Combining ultraviolet irradiation with hydrogen peroxide has been shown to effectively eliminate trace pharmaceuticals while generating significantly fewer halogenated disinfection by-products compared to UV paired with chlorine. The UV/H_2O_2 process yields notably lower concentrations of trihalomethanes, haloacetonitriles, and related compounds compared to the UV/chlorine alternative. [10]

Minimizing the chlorine dose itself is also critical. Keeping free chlorine at the lowest effective level for disinfection significantly reduces DBP formation (since DBPs scale with chlorine exposure). Complementary or alternative sanitizers can further cut DBP load. Systems such as ozone, UV-C, bromine, or saltwater electrolysis reduce chlorine demand and thus total DBPs. [11] However, these non-chlorine methods must still be validated for by-product toxicity and consistency with microbial safety standards.

In summary, pooled data support a combined approach of enhanced ventilation and layered disinfection. In practice, this means exceeding the minimum ACH and utilizing CO_2 sensors to adjust the fresh air supply according to occupancy levels. [6, 8] Advanced oxidation (e.g., UV plus ozone or H_2O_2) can break down organics and micropollutants in the water, cutting secondary DBPs. [9, 10] These methods also enable partial or complete substitution of chlorine (e.g., with ozone or UV-C) depending on feasibility, while still ensuring disinfection. Altogether, an integrated strategy of high air exchange and sophisticated water treatment markedly reduces swimmers' exposure to chlorine and its irritant by-products, and should therefore help mitigate pool-related airway symptoms. [6, 9, 10]

Medical surveillance and training-load management

Prevention requires medical oversight. Swimmers should have regular respiratory screenings, including spirometry and exhaled NO testing, to detect lung abnormalities before symptoms occur. If exercise-induced bronchoconstriction (EIB) is suspected, formal provocation testing should be considered. Swimmers respond better to eucapnic voluntary hyperpnea (EVH) testing, which replicates exercise-induced hyperventilation, than methacholine or mannitol. [12]

Training should adapt to swimmers' respiratory health. 12 of 19 competitive swimmers developed airway hyperresponsiveness (AHR) during hard training. After ≥15 days of rest or light exercise, 67% of swimmers returned to normal airway responsiveness. [11, 13]

Long-term follow-up studies showed that swimmers who stopped high-level training had a lower incidence of asthma. However, those who kept training saw AHR and asthma diagnoses rise. [14] These findings suggest that intentional "microcycles" of reduced volume or off-season pauses can help swimmers and their lungs recover without compromising long-term training.

Education and hygiene for athletes

Athlete behavior serves as an additional layer in the successful prevention of respiratory conditions in competitive swimmers. Effective pre-swim hygiene significantly reduces the formation of disinfection by-product precursors. Mandatory 1-minute soap showers can remove most sweat, lotion, and urea from the skin. One study indicated that pre-swim bathing greatly reduced pool anthropogenic contaminants. [15] Enforcing "no urination" in water and following shower-and-soap protocols can reduce urea/ammonia levels, preventing the excessive synthesis of volatile DBPs, such as NCl_3.

Young athletes deserve special attention. Early exposure to indoor chlorine pools significantly increases the risk of asthma. A study found that preschoolers exposed to trichloramine concentrations of 0.15 mg/m³ had nearly double the incidence of early-onset asthma compared to those who were not exposed. [16] Thus, youth programs should prioritize well-ventilated pool conditions, limit training sessions, and educate parents and coaches to recognize "sentinel" signs, such as overnight coughing or exercise-induced wheezing, that may indicate early bronchial sensitization. Self-monitoring by logging symptoms and peak flows allows swimmers to adjust their training or seek help.

Recommendations for coaches and medical personnel

Coaches, trainers, and team physicians should create multi-part respiratory health regimens. Regular respiratory screening is beneficial. Health evaluations can track pulmonary function, training load, and performance with periodic spirometry and FeNO tests. If symptoms suggest exercise-induced bronchoconstriction, exercise or exercise ventilation challenge testing may be necessary. Also vital is continuous monitoring of interior air and pool chemistry. Collaborating with facility engineers to monitor CO_2 and airborne disinfection by-product concentrations, especially trichloramine, can guide early responses. If trichloramine levels reach the WHO standard of 0.5 mg/m³, consider improving ventilation, airflow dispersion, or limiting pool occupancy.

Individual asthma action plans can be beneficial for swimmers. Symptom records or peak-flow readings may be linked to staged reactions, such as changes in training intensity, warm-up tactics, or medication adjustments, to ensure a proactive approach. Please note that precise documentation is essential for Therapeutic Use Exemption (TUE) compliance.

Hygiene can also help. Encourage the use of soapy pre-swim showers, swim caps, and timed restroom breaks to minimize organic contamination and DBP development.

Finally, technological innovation collaboration may benefit. Teams can test air quality and athlete respiratory problems by piloting UV-C and ozonation water-treatment systems with low-dose disinfectants. These interventions can be evaluated for health outcomes and operational costs to inform long-term implementation decisions.

Summary

Indoor swimming conditions, especially those with poor ventilation and high chlorine levels, can lead to respiratory disorders in competitive swimmers, including asthma. Long-term exposure to trichloramine, mold, and airborne allergens is associated with increased airway inflammation and exercise-induced bronchoconstriction. Additional longitudinal research and focused preventive measures, including enhanced air exchange systems, advanced water purification, and respiratory health monitoring, are needed. Maintaining optimal indoor pool air quality is crucial to athlete health.

Disclosure

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