

BACKGROUND

- When chlorine is used in pools to kill germs, it binds to the nitrogen in body waste (sweat and urine) and can form chemicals called chloramines. (5).
- Chloramines in the water irritate the respiratory tract when they are released as gas from the water and into the air (5).
- Chloramines are heavier than normal air, so they settle on top of the water's surface where swimmers breathe them in during exercise (5).

CHLORINE CHEMISTRY

Total = Free chlorine + Combined chlorine

• Free chlorine:
hypochlorous acid + hypochlorite ion
 $\text{HOCl} \leftrightarrow \text{H}^+ + \text{OCl}^-$ [pH dependent reaction]

• Combined chlorine (chloramines):
• $\text{HOCl} + \text{NH}_3 \leftrightarrow \text{NH}_2\text{Cl}$ (monochloramine) + H_2O
• $\text{HOCl} + \text{NH}_2\text{Cl} \leftrightarrow \text{NHCl}_2$ (dichloramine) + H_2O
• $\text{HOCl} + \text{NHCl}_2 \leftrightarrow \text{NCl}_3$ (trichloramine) + H_2O

Chlorine combined with ammonia or organic nitrogen (causes chlorine taste and odor)



Image 1 (1)

PURPOSE

To evaluate the potential harming effects that chloramines may have on the respiratory function of elite swimmers.

METHODS

- A literature review that underlines the impact of competitive swimming in a chlorinated environment on the athlete's respiratory health. Ailments examined in the upper airways include nasal obstruction, rhinorrhoea, sneezing, rhinitis, and nasal itching. Within the lower airways symptomatic and asymptomatic airway hyper-responsiveness (AHR), exercise-induced bronchoconstriction (EIB), and asthma were explored (2).
- 23 elite swimmers were tested during the off-season (as a control measure) as well as 10 age-matched healthy nonallergic subjects and 10 mild asthmatics. All subjects had exhaled nitric oxide measurement, methacholine test, eucapnic voluntary hyperpnea challenge, allergy skin prick tests, and bronchoscopy with bronchial biopsies (4).
- 26 competitive swimmers were recruited, as well as 13 indoor (non-swimmer) athletes and 15 controls, none of whom had no history of asthma. Lung function was measured before, immediately after, and 24 hours after a 90-min intensive exercise protocol. Sputum induction was performed at baseline and again 24 hours after exercise. Exercise-induced bronchoconstriction (EIB) was also assessed by the eucapnic voluntary hyperventilation test (6).
- Study was completed at a single pool in Nancy, France. 22 male swimmers between the ages of 15-25 who swam in this pool exclusively were sampled. Swimmers were examined twice the week after the draining of the swimming pool and twice 24 weeks later, before the draining of the swimming pool. A new technique of water treatment with new pumps were used, resulting in a decrease in the concentration of chlorinated compounds. Exposure to disinfection by-products was evaluated by the chloramine concentration in the atmosphere of the swimming pool and by the presence of trihalomethanes (THM) in the blood of the swimmers (7).

RESULTS

- Up to 76% of competitive swimmers have airway hyper-responsiveness (AHR) and/or exercise-induced bronchoconstriction (EIB) (2).
- The Swimmers had higher airway mucosa eosinophil and mast cell counts than did the controls, as well as more goblet cell hyperplasia and a higher mucin expression than that of the healthy or asthmatic subjects (4).
- 23% of swimmers had a positive eucapnic hyperventilation (EVH) test compared with none of the indoor athletes and one of the controls. A selected panel of cytokines and serum biomarkers known to be involved in asthma and endothelial damage was also analyzed showing that baseline sputum eosinophil and neutrophil counts were significantly higher in swimmers compared with controls (6).
- Erythrocyte $\text{Cu}^{++}/\text{Zn}^{++}$ SOD and GSH-Px activities were found to be significantly associated with exposure of chlorinated compounds and training. The difference in the level of erythrocyte GSH-Px activity depended on the week of and on the number of hours of swimming. Irritant eye symptoms, nasal symptoms, throat irritations, and asthma were reported. When compared with a group of 256 male lifeguards, the prevalence of these symptoms were significantly higher in swimmers and the prevalence tended to increase with increasing exposure to nitrogen trichloride (NCl_3) (7).

CONCLUSION

- The release of chloramines is considered increasingly responsible for the occurrence of respiratory disorders in swimmers (3).
- The prevalence of asthma before beginning competitive swimming is similar to that of other sports. This suggests that swimmers do not engage in swimming due to their asthma, but rather develop respiratory disorders during their athletic career (2).
- There is not significant differences in performance for those swimmers with upper airway disorders. However, the differences in time were greater than the differences between medal placements (2).
- Chloramines are known to cause the disruption of epithelial tight junctions which can then facilitate the penetration of allergens/pollutants and the migration of inflammatory cells across the epithelial barrier (3).
- The best way to reduce the impact on swimmers' airways is to reduce the release of chloramines by focusing on proper hygiene, providing adequate ventilation, or using a different kind of disinfectant (3).

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