INTRODUCTION

Swimming has been recommended to people to keep healthy and fit since, at least, the Roman Empire. Nobody questions today the beneficial effects of physical exercise in water, so that swimming is recommended to prevent and treat some pathologies of the locomotive system—especially of the spine [27], circulatory problems [7] and it is even prescribed as the most appropriate sport for asthmatic children [59].

However, in recent years numerous papers have been published warning about health risks of swimming pool—and similar settings, e.g. spas—attendance [16,79]. Such risks are related to the use of chemical agents for disinfection, particularly chlorine and its disinfection by-products (DBPs). Therefore, the aim of this article was to review the available publications in the peer-reviewed scientific literature to provide insight into how chlorinated swimming pool attendance could affect the health of swimmers and associated staff. After describing the substances resulting from water chlorination and different ways of exposure, this article will discuss the effects of chlorine derivatives on the populations exposed to chlorine and its DBPs: recreational and competition swimmers, technical staff and operators. Finally, some recommendations are presented to preserve the benefits of swimming and to reduce potential harming effects as much as possible.

Swimming pool disinfection

The large number of people going to the pools causes water to have a great deal of organic (hair, sweat, epithelial cells, urine, etc.) and inorganic substances (dirt in nails and skin folds, solid waste from different sources, etc.), making the maintenance of physical and chemical properties within certain values necessary. Batjer et al. [9] pointed out that, even under strict hygienic conditions, each swimmer contributes a few grams of organic matter into the pool.

Water in swimming pools is not free from potentially dangerous substances. Already in the 1970s, studies conducted by Rook [66-68] showed that treated water for human consumption contained certain chemical substances that could be dangerous. The reaction of a number of polluting elements existing in water with chemicals used in disinfection results in numerous toxic substances generically grouped as disinfection by-products (DBPs).
Swimmers are exposed to these products in three ways [29,59]:

1) Water ingestion: all swimmers swallow water to a greater or lesser extent depending on variables such as age, sex, technical skills, swimming style, intensity and duration. The only study that has experimentally measured water ingestion is that of Evans et al. [32], reporting an average ingestion of 26.5 ml of water per session, although considerable differences were found depending on age (37 ml in children versus 16 ml in adults) and gender (45 ml in boys, 30 ml in girls, 22 ml in men and 12 ml in women).

2) Inhalation: many of the DBPs are volatile and as such they are inhaled by swimmers. This is actually the way most DBPs enter the body, 66% of the total. The amount of air ventilated by swimmers depends on their swimming pace, this way in intense swimming up to 100 l/min can be ventilated while in a more relaxed activity 5 l/min are ventilated. Drobnic et al. [26] estimated chlorine inhalation in a group of swimmers training in an indoor pool. In a two-hour training period, swimmers could be exposed to a chlorine amount over the recommended level for a worker after 8 hours exposure (from 4 to 6 g.). On the other hand, the concentration of volatile substances varies largely depending on the treatment and the type of pool (indoor or outdoor). For instance, in indoor pools trihalomethane concentration is 100 µg/l while in outdoor pools it does not exceed 10 µg/l [22,33,69].

3) Dermal absorption: the irritating effect of chemicals in water is well-known by swimmers, especially in the eyes and mucosa, but these substances can penetrate the skin too. Raykar et al. [64] have pointed out that some lipophilic and lipophobic products can go through the stratum corneum of the epidermis and spread into the body. This is considered to be the least important pathway but nobody has yet measured the amount of products that penetrate the organism in this way.

The substances swimmers are exposed to depend on the chemical treatment used. In the disinfection of swimming pool water, chlorine is the most commonly used product. But, apart from that, it is also the substance causing the most problems as a result of the DBPs it generates, as will be explained next.

Water chlorination

Since chlorine (Cl-) was discovered in 1774 by the German chemist Carl Wilhelm Scheele, its compounds have been used in different applications, i.e. explosives, pesticides, for whitening, refrigerating and of course for water disinfection. Several products derived from chlorine are used in water disinfection, grouped as follows [79]:

- a) products derived from non-stabilised chlorine like chlorine gas, sodium hypochlorite or calcium hypochlorite and
- b) products derived from stabilised chlorine, mainly isocyanides (sodium dichloroisocyanurate and trichloroisocyanuric acid).

From all such products, sodium hypochlorite (NaOCl) and dichloroisocyanide are the most widely used [22,46]. Regardless of the product used, the free or residual chlorine portion is responsible for disinfection. The amount of free chlorine allowed by the regulations varies across countries but it should not exceed 3 mg/l (up to 5 mg/l in drinking water) in public swimming pools [79]. Yet, many facilities use regular “shock doses” consisting of 20 mg/l as a preventative measure.

Due to its large oxidising capacity, apart from disinfecting, free chlorine reacts with nitrogenated organic compounds present in sweat and urine. All these compounds react to hypochlorite forming chloramines [42,46,71]. It must be noted that, even when chlorine and chlorinated compounds are used correctly, reactions leading to DBPs formation are inevitable [3]. The DBPs known at present are shown below [21,57,80]:

1) Trihalomethanes (THMs). These are the DBPs produced in larger quantities. Chloroform (CHCl₃) is the most representative and it is used to measure THM exposure in indoor swimming pools [74]. It is classified in group 2B of the IARC (International Agency for Research on Cancer) as a carcinogenic agent [41]. Nevertheless, very few studies on the effects on human health caused by environmental exposure over long periods have been conducted [2].

Due to their volatile nature, THMs are released into the air from the water surface and are therefore inhaled not only by swimmers but also by any other users or technical staff [33]. From all the variables affecting the THM level in indoor pools, the most relevant is the number of swimmers, existing a linear correlation between their number and THM concentration [22]. As for their concentration, it is not uniform: greater concentration has been measured on the surface, with a gradual decrease taking place as the distance from the water sheet grows [50].

Analyses performed in indoor pools in the United States [69], Italy [33] and England [22], indicate that THM concentrations in water are about 100 µg/l, ranging from 17.8 µg/l to 313 µg/l. In outdoor swimming pools, amounts range between 3.8 µg/l and 6.4 µg/l, which stresses the importance of having adequate ventilation.
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On the other hand, Fantuzzi et al. [33] have shown that THMs spread all around the premises in a swimming pool, reporting concentrations of 26.1±24.3 µg/m³ even in the reception desk.

With regard to concentrations in users, Aggazzotti et al. [3] pointed out that, after swimming for one hour, THM absorption is seven times higher than values while resting. They also noticed a negative correlation between chloriform values and age, which suggests that younger individuals absorb chloriform faster. The elimination of the absorbed chloroform is quick and complete after 10 hours. Levesque et al. [53] studied chloroform exposure in water in indoor swimming pools and found that 1 hour swimming meant a chloroform dose of 65 µg/kg/day, 141 times the dose of a 10 minute shower and 93 times the exposure by drinking water ingestion; they also found out that, when chloroform levels in water are kept between 150 and 300 ppb, the body concentration after 35 minutes swimming is equivalent to 55 minutes, reaching a balance between absorption and elimination. However, at levels over 500 ppb a balance is no longer possible and the chloroform load rises with longer swimming periods, which evidences the saturation of biotransformation mechanisms. Recently, Caro and Gallego [21] performed a study on workers' exposure to THMs, analyzing urine samples and measuring the concentration of chloroform and bromodichloromethane as an index of exposure to these compounds. Their results showed that THM uptake in swimmers was higher after 1 hour of exercise than that of workers after 4 hours of work.

2) Chloramines. These DBPs are responsible for the typical “swimming pool smell”, eye and mucosa soreness and skin dryness [58]. These inorganic compounds are volatile and highly reactive [40]. The most abundant chloramines are monochloramine (NH₂Cl), dichloramine (NHCl₂) and trichloramine (nitrogen trichloride, NCl₃). The latter is the most irritating and generates a stronger odour [40], noticeable at concentrations as low as 0.02 mg/l [48]. Hery et al. [39] reported that discomfort caused by chloramines starts with concentrations of just 0.3 mg/m³, while the levels recorded in swimming pools were 0.84 mg/m³.

In outdoor pools chloramines and THM tend to dissipate into the air. However, in indoor pools, a constant production of chloramines may result in high concentrations that can be dangerous [58]. Mean values recorded in France, Belgium and Germany swimming pools shifted between 0.2 and 0.9 mg/m³ [40,60], chloramines thus being one of the most concentrated air pollutants (the rest of pollutants the population is exposed to hardly ever exceed 0.3 mg/m³) [60]. Chloramine concentration grows with the number of users, water temperature and the turbulence caused by swimmers. That explains why in leisure centers chloramine concentration is higher than in public swimming pools [58].

Recently, Jacobs et al. [43] measured the presence of chloramines in 38 pools, finding mean concentrations of 0.65 mg/m³ and maximum of 1.34 mg/m³. A regression analysis of the data proved that the number of users in the water was significantly associated to the chloramine level, in such a way that an increase of 50 additional swimmers meant a 0.40 mg/m³ rise. They were also significantly associated (p<0.01) to the levels of trichloramine, the free chlorine in water and the ceiling height.

3) Haloacetic acids (HAAs). Among them, trichloroacetic acid (CCl₃COOH) is a strong carboxylic acid. Clemens and Scholer [23] analysed 15 indoor pools in Germany, reporting mean concentrations of 119.9 µg/l. In turn, Kim and Weisel [47] studied three indoor swimming pools in the US, reporting much higher concentrations, 419 µg/l on average. On the contrary, with outdoor swimming pools, concentrations hardly ever exceed 10 µg/l [23]. Once more, these findings underline the importance of adequate ventilation in pools to avoid dangerous concentrations.

4) Other substances. This category includes known DBPs which there is little information about, substances like haloketones, trichloroaldehydes, trichloronitromethane and cyanogen chloride.

The “chlorine hypothesis”

The consequences of accidental chlorine and DBPs exposure are well-known [1,17,24], but the harmful effects of chronic DBPs exposure in swimming pool at typical concentrations have only recently been determined. The link between swimming and respiratory health has been first investigated in Belgium with a study conducted with school children [13]. Surprisingly, their findings showed that the increase in the permeability of the pulmonary epithelium caused by DBPs can make children more prone to asthma, which is rather paradoxical if we bear in mind that swimming has traditionally been recommended to asthmatic subjects due to the warm, humid atmosphere of swimming pools.

Disorders in the function of clara cells -whose main mission is to protect the pulmonary epithelium- have also been reported. Lagerkvist et al. [49] used the plasma concentration of the protein secreted by these cells (CC16) to examine the response produced by exercising in environmental air and in the atmosphere of chlorinated swimming pools. The results proved that there were no significant changes in the mean CC16 concentrations before and after open-air exercising; yet, children regularly going to chlorinated pools had significantly lower CC16 levels. Therefore, regular exposure to DBPs in the air in indoor swimming pools could exert negative effects on the proper function of clara cells in children.

Regarding air concentrations of NCL₃ and pulmonary damage, Carbonelle et al. [20] observed that air concentrations of NCL₃ between 355 and 490 µg/m³ induced changes in the levels of CC16 and surfactant protein A (SP-A) and B (SP-B) in trained and recreational swimmers, showing epithelial cell injury. On the other hand, a recent research conducted by Carbonelle et al. [19] has shown that exercise performed in indoor swimming-pool containing less than 300 µg/m³ of NCL₃ in air does not induce short term changes in the serum level of SP-A, SP-B, CC16 and Krebs von den Lunge-6 (KL-6), another marker of pulmonary epithelial cell injury, showing the importance of an adequate control of NCL₃ concentration.
Recent studies reported on the relationship between swimming in indoor [11,60] and outdoor [14] chlorinated pools and the prevalence of panting, asthma, hay fever, rhinitis and atopic eczema, giving way to the so-called “chlorine hypothesis”. This hypothesis postulates that childhood asthma rise in industrialized countries would be caused, at least partly, by the increasing and largely uncontrolled exposure of young children to chlorination by-products containing the air of indoor swimming pools [13], mainly trichloramine [11]. However, this is a controversial hypothesis and some researchers have questioned the findings of Bernard et al. They argue several methodological [6,28,65] and political [65] aspects, among which the limitations of an ecological study design stand out. Therefore, until a clear cause-effect relationship between chlorinated swimming pool attendance and asthma development has been established using prospective longitudinal studies, this hypothesis should be considered with caution. In addition, it is difficult to evaluate how the pool chlorine hypothesis could explain the variation of asthma prevalence because there is insufficient information to distinguish the effects associated with DBPs exposure and their contribution from other variables such as swim exercise itself or other environmental and societal risk factors such as dust and air pollution [43], obesity [4], smoking [36], microbial infections [44], nutritional status [30] and others.

If this hypothesis is taken into account, special attention must be paid to baby swimming. In these early ages the body is in full development, being very sensitive to interactions with chemicals. In the last years, this has been a matter of great concern and some studies have been carried out to evaluate the effects of early swimming in the respiratory health of children.

Some researchers found that baby swimming increases the risk of infection in the respiratory tract and the middle ear, particularly in genetically predisposed children [62]. Evidence presented by Nystad et al. [61] suggests that some children could have an increased risk of wheeze from 6 to 18 months if they take part in baby swimming during the first 6 months of life; on the other hand, the risk of lower respiratory tract infections or otitis media was not increased. Finally, in Germany, Schoefer et al. [70] found an association between swimming pool attendance and higher rates of diarrhoea, but they did not find any connection between attendance of chlorinated swimming pools and allergic outcomes. However, the results of this study were conditioned by the different recommended levels of chlorine between Belgium where most of the studies have been performed) and Germany, with chlorine levels 10 times lower.

The results obtained by Bernard and Nickmilder [15], Bernard et al. [11] warned that the use of indoor chlorinated pools, especially by children, can interact with the state of atopy that in the respiratory system produces rhinitis and asthma. The authors reported that the risk is greater in children who swim in indoor chlorinated pools regularly before they are 6-7 years old. Children of these ages seem to be more sensitive to DBPs, an aspect that is enhanced by the fact that they use more the “splash pools”, kept at a higher temperature and subsequently with larger amounts of DBPs. Besides, when children play or learn to swim they inhale and swallow more gases and water. Bernard et al. [12] evaluated the impact of infant swimming on the allergic status and respiratory health of 341 schoolchildren aged from 10 to 13 years, measuring aeroallergen-specific immunoglobulin E (IgE), exhaled nitric oxide (eNO) and the serum concentration of CC16. They reported that children who swam as infants showed clara cell damage and permeability changes of the lung epithelial barrier, effects that are associated with higher risk of asthma and recurrent bronchitis. On the other hand, they did not find differences in the levels of IgE and eNO. Recently, a study conducted by Voisin et al. has shown that swimming in indoor and outdoor pools during infancy is associated with a dose-dependent increase in the risk of bronchiolitis. This effect independent of other risk factors as exposure to tobacco or smoke, day-care attendance or antecedents of atopic diseases and could increase the risk of asthma and respiratory allergies later during childhood [76].

**Effects on competition swimmers, technical staff and operators of swimming facilities**

In addition to the effects described earlier, abundant research has focused on other populations exposed to chlorine and its DBPs, not only swimmers who spend hours exercising in water but also technical staff and operators in contact with the environment of the swimming pool.

Regarding the incidence of asthma and bronchial hyperreactivity in athletes, studies conducted on US top level athletes [77,78] noticed a higher incidence of asthma in endurance disciplines such as cycling, swimming, rowing or long-distance skiing. These studies concluded that the higher prevalence could be attributed to the prolonged hyperventilation and higher exposure to allergenic and irritating agents while training, underlining that the environment where the activity is performed can be an important factor influencing the appearance of asthma or airway problems. Along these lines, Langdeau et al. [51] found that elite swimmers and subjects who practised winter sports had a higher airway hyperreactivity prevalence than sedentary individuals of the same age, being the incidence of asthma and airway hyper-responsiveness more frequent or more marked in swimmers than in cold air athletes.

Taking into account the aforementioned, environmental conditions of swimming pools could be an important factor in the development of such airway lesions. For this purpose, several researches have studied bronchial hyperreactivity and airway inflammation in swimmers. Helenius et al. [38] found out that bronchial hyperreactivity was more present in swimmers (48%) than in non swimmers (16%) and, after analysing the characteristics of the subjects’ sputum noticed that the amount of eosinophil and neutrophil leukocytes was significantly higher in swimmers. Similarly, swimmers with bronchial hyperreactivity had a greater amount of eosinophils (responsible for
pro-inflammatory reactions like allergies) than those without symptoms, as well as a significantly higher bronchial hyperreactivity to histamine. In contrast, Belda et al. [10] found that the inflammation could be mediated by neutrophils, rather than eosinophils, and could be related to the duration of training, perhaps due to exposure to chlorine derivatives.

Later studies carried out in competition swimmers showed a high asthma incidence and other respiratory symptoms induced by exercising, asthma and hay fever occurring more frequently in the group of international swimmers (19% and 21% respectively) [37]. These authors concluded that the most important risk factors for asthma were age and the amount of training done in the pool.

Likewise, an important proportion of swimmers often refer to irritating symptoms on eyes and throat, and headaches when swimming in indoor pools. In this respect, Levesque et al. [55] compared the prevalence of health problems in young swimmers and footballers and observed that the presence of respiratory symptoms as coughing, sore throat, sore eyes and outer ear otitis was greater in swimmers than in indoor soccer players. They concluded that the incidence of these symptoms could potentially be reduced by limiting exposure to chlorination by-products.

In the case of coaches, instructors, lifeguards or maintenance staff, DBPs inhalation is not negligible and after an hour’s exposure in a typical 100 µg/m³ atmosphere, a THM concentration of 30 µg is found in alveolar air [2]. Massin et al. [58] studied the occurrence of eye and respiratory symptoms in lifeguards working in indoor swimming pools to investigate the relationship with trichloramine concentrations.

According to the results, there was a high prevalence of irritation in eyes and nose, regardless of sex, which significantly increased with greater trichloramine concentrations. They also found a direct relationship between trichloramine concentration and temporary bronchial hyperreactivity, which could indicate that lifeguards can develop asthma as an occupational disease. Along the same lines, a few years later Thickett et al. [72] identified the first cases of occupational asthma in lifeguards. The results of their study evidenced a relationship between trichloramine exposure and asthma development in swimming pool workers. This is a very relevant finding, as it was previously thought that it only caused transient symptoms and discomfort.

Recently Jacobs et al. [43] estimated trihalomethane exposure at work in 624 indoor pool workers. The results showed that these individuals were more likely to develop conditions related to the upper airways (sinusitis, chronic colds, and a sore throat).

Those employees who, apart from working in the pools, also used them as swimmers had a greater occurrence of respiratory symptoms associated with rhinitis, nasal congestion, sneezing, and sore and tearful eyes. In addition, when the temperature and humidity were too high, the ventilation was not adequate and the workers reported discomfort caused by the presence of chemicals, the prevalence of respiratory and allergic symptoms was 1.4 to 4.6 times greater.

**Other effects**

Apart from the described effects of chlorine and its DBPs on the respiratory system, several studies have analysed the effects on other parts of the body.

Escartín et al. [31] studied the incidence of dental stains in competition swimmers. Prevalence was higher in the swimmers group (60.2%) than in controls (12.9%) and strong correlation was found with competition swimming so that the more hours of training per week in the pool, the greater the incidence of dental stains; this relationship was not linear, as a brusque increase was found in training periods longer than 6 hours a week.

Swimmers usually suffer from dry skin or xerosis [34]. Atopic [45,56,73] and contact [52,63] dermatitis have occasionally been identified. Less frequent is an increased production of grease, known as “swimmer’s shine” [8], caused by prolonged skin hyperhydration.

As far as the effects on the nose and nose physiology are regarded, Deitmer and Scheffler [25] found that swimmers had greater obstruction, itching, nasal discharge, sinusitis and allergies. Besides, swimmers often referred to an “allergy to chlorinated water”. This term was subsequently used by Bonini et al. [18] to describe a clinical case characterised by nasal obstruction, liquid nasal discharge and sneezing.

The relationship between chronic exposure and chlorinated compounds has also been explored using oxidative stress indicators in swimmers. Varraso et al. [74] evaluated DBPs exposure measuring chloramine concentration in the pool’s water and THMs in the swimmers’ blood. Cu^{2+}, Zn^{2+}, superoxide dismutase (SOD), and glutathione peroxidase (GSH-Px) were the biological markers of the oxidative stress response. The results confirmed that the oxidative stress found was not exclusively due to exercising and part of it was caused by the toxicity of chlorine and its DBPs. In this case, chloriform seemed to be the main factor.

Other research lines have attempted to assess potential health risks of chloroform exposure in swimmers. The highest concentration they found in competition swimmers was 10,000 times smaller than that required for tumours to appear in animals and, for that reason, the authors concluded that swimmers have a large safety margin [54]. Nonetheless, ascertaining what actually occurs in repetitive and long-term exposure to this product is still pending.

Aiking et al. [5] studied the hepatotoxic and nephrotoxic effects of chlorinated compounds in 18 competition swimmers, finding out that B2 microglobulin, and indicator of kidney damage, was significantly higher in the urine samples of younger swimmers.

Recently, Villanueva et al. [75] tried to determine whether the risk of developing bladder cancer was related to THMs exposure while showering, bathing or using indoor swimming pools, since studies conducted in drinking water had so pointed out. Their findings support experimental and epidemiological evidences that THMs, and possibly other DBPs, are associated with a higher risk of bladder cancer, not only through drinking water but also through ingestion, inhalation and dermal absorption in closed environments like swimming pools.
Chlorine has also been attributed the whitish or greenish colour of the hair of some swimmers. This pigmentation change is reversible and is only worrying from an aesthetic point of view, being the consequence of prolonged exposure to swimming pool water and sunlight. A whitish pigmentation is indeed caused by the decolouring power of chlorine but the greenish colour is caused by copper ions in water [8].

CONCLUSIONS
The review of the literature conducted shows the effects of chlorination used in indoor swimming pools and similar settings for different functional systems of the human body. From all the possible harmful effects reported, those affecting the respiratory system are the most noticeable, to the extent that they have given way to the "chlorine hypothesis" which postulates that swimming in a chlorinated pool from an early age could be an important environmental factor for asthma in children. Likewise, in 2002 the first documented cases of occupational asthma were reported in lifeguards. However, there are some dissenting opinions making this a topic which needs further investigation, since it is well established that there are other environmental and social factors that could contribute to the rise of respiratory disorders.

REFERENCES


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