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Review

Disinfection byproducts in swimming pool: Occurrences, implications and future needs

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ABSTRACT

Disinfection of swimming pool water is essential to deactivate pathogenic microorganisms. Many swimming pools apply chlorine or bromine based disinfectants to prevent microbial growth. The chlorinated swimming pool water contains higher chlorine residual and is maintained at a higher temperature than a typical drinking water distribution system. It constitutes environments with high levels of disinfection by-products (DBPs) in water and air as a consequence of continuous disinfection and constant organic loading from the bathers. Exposure to those DBPs is inevitable for any bather or trainer, while such exposures can have elevated risks to human health. To date, over 70 peer-reviewed publications have reported various aspects of swimming pool, including types and quantities of DBPs, organic loads from bathers, factors affecting DBPs formation in swimming pool, human exposure and their potential risks. This paper aims to review the state of research on swimming pool including with the focus of DBPs in swimming pools, understand their types and variability, possible health effects and analyze the factors responsible for the formation of various DBPs in a swimming pool. The study identifies the current challenges and future research needs to minimize DBPs formation in a swimming pool and their consequent negative effects to bathers and trainers.

Contents

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1. Introduction

Chlorination as a disinfection approach for water was first introduced in 1902 in Middlekerke, Belgium (MWH, 2005). Since then, chlorination has been known as an efficient disinfection approach for municipal water (Chlorine Chemistry Council, 2003; Health Canada, 2009; USEPA, 2006; WHO, 2011). Other disinfectants, such as, chloramine, chlorine dioxide, ozone and UV irradiation are also applied for water disinfection (AWWA, 2000; MOE, 2007). Disinfection of swimming pool water is essential to prevent outbreaks of infectious illnesses (Geldreich, 1989; USCDC, 1997; MOE, 2002; MWH, 2005; CDC, 2007; HaitiLibre, 2010). While disinfectants inactivate pathogens in swimming pools, unintended reactions between disinfectants (e.g., chlorine, chloramines, ozone, or chlorine dioxide) and natural organic matter (NOM), bromide/iodide and human inputs (e.g., constituents of sweat and urine, skin particles, hair, microorganisms, cosmetics, and other personal care products) form disinfection by-products (DBPs) (Weisel et al., 2009). The types and concentrations of DBPs depend on several factors, including the type and amount of disinfectant used, characteristics of the swimming pool and pool water and users' hygiene (Zwiener et al., 2007). In swimming pools, chlorine is the most commonly used disinfectant. The types of chlorine generally used are sodium hypochlorite (liquid bleach), calcium hypochlorite, or chlorine gas for the indoor pools (Ford Red, 2007), and stabilized chlorine products (e.g., stabilized chlorine granules, chlorinated isocyanurates, chlorine tablets) are typically used for outdoor swimming pools. The swimming pool waters generally have higher temperature leading to the higher rates of chlorine decay. To compensate for the chlorine demand, swimming pools use relatively higher doses of chlorine to ensure free residuals in the pool water (Richardson et al., 2010; Weisel et al., 2009). Higher free residual chlorine (FRC), higher temperature, constant organic loads, contact of water surface with air and water recirculation can affect DBPs formation in swimming pool. More than 600 DBPs have been identified in chlorinated waters, and many of them are mutagenic or carcinogenic (Richardson et al., 2007).

Swimming pools constitute environments with high levels of DBPs in water and air due to continuous disinfection and constant organic load from bathers (Kim et al., 2002; LaKind et al., 2010). To date, the DBPs identified in the swimming pool are trihalomethanes (THMs), haloacetic acids (HAAs), haloacids, halodiacids, iodo-THMs, haloaldehydes, halonitriles, haloketones (HKs), halonitromethanes, bromate, haloamides, haloalcohols, nitrosamines, combined available chlorine, and 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) and MX homologues, etc. (Richardson et al., 2010). The most prevalent DBPs in swimming pool are chloramines, THMs and HAAs, (Chu and Nieuwenhuijsen, 2002; Kim et al., 2002). Chloramines are also an important group of disinfectants, which may also form some DBPs in swimming pools (e.g., iodinated DBPs, N-Nitrosodimethylamine [NDMA]). Given the high nitrogen content of organic matter from bathers, nitrogenous DBPs such as, haloacetonitriles, nitrosamines (e.g., NDMA) can also be formed. In addition, elevated levels of ammonia in urine react with chlorine and lead to formation of chloramines, which are also found at high concentrations in swimming pools (Richardson et al., 2010; Zwiener et al., 2007; Walse and Mitch, 2008). Ammonia is also produced during ammonification of urea in biofilms. Past studies have reported the presence of biofilms in both chlorinated and unchlorinated swimming pools (Keuten et al., 2009; Schets et al., 2011; Casanovas-Massana and Blanch, 2013). The bacteria in the biofilms utilize urea as a nutrient and release ammonia as a residual. In addition, the dead cells separated from the biofilms add organic substances into the pool water. The addition of organics from the dead cells is nearly constant at a relatively low concentration. The biofilms and the separation of dead cells from the biofilms can play an important role in the formation and distribution of DBPs in swimming pools. Many DBPs, especially nitrogenous ones, are more cyto- and genotoxic than the regulated DBPs (i.e., THM and HAA) (Richardson et al., 2010). Therefore, it is not surprising that pool water is found to be more genotoxic than the source tap water, and that the type of disinfectant and illumination conditions altered the genotoxicity (Liviac et al., 2010). The genotoxicity study of swimming pool water reported that the pool water induced DNA damage in Hep-G2 cells (comet assay), and that most of the genotoxicity was associated with lower-molecular-weight (<200 g/mole) DBPs (Glauner et al., 2005). It is to be noted that the major routes of uptakes of DBPs from swimming pools are inhalation and dermal contact, while contribution from accidental ingestion may be minimal. In contrast, DBPs uptakes from drinking water