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Bioanalytical and chemical evaluation of disinfection by-products in swimming pool water

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ABSTRACT

Pool water disinfection is vital to prevent microbial pathogens. However, potentially hazardous disinfection by-products (DBP) are formed from the reaction between disinfectants and organic/inorganic precursors. The aim of this study was to evaluate the presence of DBPs in various swimming pool types in Brisbane, Australia, including outdoor, indoor and baby pools, and the dynamics after a complete water renewal. Chemical analysis of 36 regulated and commonly found DBPs and total adsorbable organic halogens as well as *in vitro* bioassays targeting cytotoxicity, oxidative stress and genotoxicity were used to evaluate swimming pool water quality. Dichloroacetic acid and trichloroacetic acid dominated in the pool water samples with higher levels (up to 2600 μg/L) than the health guideline values set by the Australian Drinking Water Guidelines (100 μg/L). Chlorinated DBPs occurred at higher concentrations compared to tap water, while brominated DBPs decreased gradually with increasing pool water age. Biological effects were expressed as chloroacetic acid equivalent concentrations and compared to predicted effects from chemical analysis and biological characterisation of haloacetic acids. The quantified haloacetic acids explained 35–118% of the absorbable organic halogens but less than 4% of the observed non-specific toxicity (cytotoxicity), and less than 1% of the observed oxidative stress response and genotoxicity. While the DBP concentrations in Australian pools found in this study are not likely to cause any adverse health effect, they are higher than in other countries and could be reduced by better hygiene of pool users, such as thorough showering prior to entering the pool and avoiding urination during swimming.

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1. Introduction

Chemical disinfectants minimise microbial pathogen growth in swimming pools and thus prevent potential adverse health effects in human. However, chemical disinfectants can interact with natural organic matter and organic micropollutants brought in by swimmers to form potentially hazardous compounds known as disinfection by-products (DBPs) (Richardson et al., 2010). More than 600 DBPs have been identified in drinking water (Richardson et al., 2007) and many DBPs were also found in swimming pool water (Chowdhury et al., 2014). Identified DBPs (Plewa et al., 2008) as well as swimming pool water samples (Glauner et al., 2005; Liviac et al., 2010; Plewa et al., 2011) were shown to be carcinogenic or mutagenic.

Epidemiological studies on chronic exposure to DBPs in drinking water suggested an association between bladder cancer and exposure to chlorinated drinking water (Villanueva et al., 2007; Cantor et al., 2010). Increased trihalomethane (THM) concentrations and some positive biomarkers of genotoxicity were observed in swimmers after a regular training session (Kogevinas et al., 2010).

The considerable research that has been done on DBPs in chlorinated drinking water has led to the definition of health-based guideline values (GV) for several DBPs in drinking water by the World Health Organisation (WHO, 2011), the Australian National Health and Medical Research Council (NHMRC, 2011), the United States Environmental Protection Agency (U.S. EPA, 2011), and the European Union (European Parliament and European Council, 2009). There are few GVs for DBPs in swimming pool water apart from THMs, e.g., the German Norm (DIN, 19643-1, 2011), but the WHO recommended reading across drinking water GVs while considering differences in exposure route and amount of ingested water (WHO, 2006).

Although there are currently guidelines for managing risks in recreational water in Australia they do not include DBPs in swimming pools (NHMRC, 2008). The Australian Drinking Water Guidelines (ADWG) have only set GV in drinking water for 10 out of 23 of the recognised DBPs due to the limited knowledge on occurrence and toxicity (Appendix A, Table S1 (NHMRC, 2011)).

Drinking water commonly serves as source water in pools, and it contains organic and inorganic (e.g., bromide and iodide) precursors for DBP formation as well as previously formed DBPs during disinfection at water treatment plants. Pool users further introduce anthropogenic organic micropollutants (e.g., cosmetic products such as sunscreen, deodorant and lotions) and natural organic matter from bodily excretions such as saliva, urine and sweat. Other factors such as filling water quality, pool type (i.e., outdoors versus indoors), intensity of usage, temperature, pH, disinfectant used, disinfection process and contact time can all contribute to the overall complicated chemistry of swimming pool water (WHO, 2006).

Although more than 600 DBPs have been identified, there are still many unknown DBPs. Hua and Reckhow (2007) showed that about 45% of the halogenated DBPs (measured as adsorbable organic halogens (AOX)) attributed to known DBPs during chlorination, indicating that 55% were still unknown. This highlights the need to have a multidisciplinary approach to investigate the complex mixture effect of swimming pool water.

The goal of this study was to identify and quantify relevant DBPs by chemical analysis of various swimming pool waters across Brisbane, Australia. Precursors were measured in the form of total nitrogen (TN), total organic nitrogen (TON) and total organic carbon (TOC). TN is a measure of both organic and inorganic nitrogen while TON measures organic compounds including proteins, amino acids, and urea. TOC is a measure of the organic carbon in water and is mainly composed of humic substances that contribute to the formation of DBPs. As representative DBPs, 27 volatile DBPs and 8 haloacetic acids (HAA) were quantified.

Cell-based *in vitro* bioassays are useful in water quality assessment and complement chemical analysis of DBPs as they can capture the effects of heterogeneous mixtures of known and unknown compounds and give information on specific endpoints relevant for human or environmental health (Escher and Leusch, 2012). The reactive properties of known DBPs lead to a focus of biological assessment on genotoxicity and carcinogenicity in previous studies (Richardson et al., 2007; Plewa et al., 2012).

Liviac et al. (2010) evaluated the genotoxicity of pool water extracts and found highest effects when the water was treated with bromochlorodimethylhydantoin, followed by chlorination and a combination of free chlorine and UV produced the samples with lowest genotoxicity, albeit still higher than tap water. Plewa et al. (2011) investigated the cytotoxicity of pool water extracts and found higher toxic effects towards mammalian cells than for chlorinated tap water. We demonstrated the applicability of bioanalytical tools for the investigation of DBP formation and the toxicity of DBPs formed in a full-scale drinking water treatment plant (Neale et al., 2012) and in lab-based experiments on the formation potential of DBPs from different organic matter precursor fractions (Farré et al., 2013).

Not only genotoxicity but also the non-specific cytotoxicity and the oxidative stress response were found to be good indicators of the formation of DBPs (Neale et al., 2012; Farré et al., 2013). We assessed non-specific toxicity with the bioluminescence inhibition assay with *Vibrio fischeri* (Tang et al., 2013b) and the induction of oxidative stress response using the AREc32 assay (Wang et al., 2006). With respect to genotoxicity, the bacterial assay umuC was applied to address the SOS response, an early indicator of DNA damage (Oda et al., 1985), and the CellSensor™ p53RE-*bla* HCT-116 assay (Knight et al., 2009) to measure the p53 activation in mammalian cells, which is an adaptive stress response to DNA damage that triggers repair, cell cycle arrest and apoptosis (Bieging and Attardi, 2012).

By complementing chemical analysis (precursor analysis, DBP analysis and halogen-specific AOX analysis) with

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