Health risk of swimming pool disinfection by-products: a regulatory perspective

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ABSTRACT

While disinfection of swimming pools is indispensable for microbiological safety, it may lead to the formation of disinfection by-products. Most studies agree that inhalation exposure is the predominant pathway of the associated health risks, but assumptions are based on concentrations measured in water and evaporation models. Pool water and air were sampled in 19 swimming pools. Trihalomethanes were detected in all sites; chloroform being the most abundant species. Concentrations ranged between 12.8–71.2 μg/L and 11.1–102.2 μg/m^3 in pool water and air, respectively. The individual lifetime carcinogenic risk associated with chloroform in swimming pools exceeded 10^{-6} in all age groups for recreational swimmers and 10^{-5} for elite swimmers and staff, even if the pool complied with the national standards. Inhalation exposure was estimated and found to be the most relevant, however, different mass transfer models from water measurements significantly under- or overestimated the health burden compared to direct calculation from the concentration in air. The observed health risks call for defining regulatory values and monitoring requirement of indoor air quality in swimming pools.

Key words | disinfection by-products, health risk assessment, indoor air quality, regulation, swimming pool, trihalomethanes

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INTRODUCTION

Disinfection of swimming pool water is important to protect bathers against water transmitted microbial pathogens. Chlorine based disinfectants are the most commonly applied products due to the versatility, effectiveness, low cost and residual disinfecting power of chlorine. However, it is also known that the reactions between free chlorine and organic matter introduced by the bathers in the pool water lead to the formation of disinfection by-products (DBPs) (Manasfi *et al.* 2017). Though over 100 different DBPs have been identified so far, the most commonly detected DBPs in chlorinated pool water are trihalomethanes (THMs): chloroform ($CHCl_3$), bromodichloromethane (BDCM), dibromo-chloromethane (DBCM) and bromoform ($CHBr_3$); mono-, di- and trichloramine (NCl_3), halonitriles (HANs) and haloacetic acids (HAAs) (Richardson *et al.* 2010; Lourencetti *et al.* 2012). The concentration and the composition of DBPs in water were shown to correlate with a number of factors including pool water quality (concentration of organic matter and nitrogen-forms), method of disinfection (hypochlorite > chlorine gas > chloro isocyanurates > chlorodioxide), good operational practice (bather load, efficiency of filtration, adequate replenishment of fresh water, etc.). DBPs are also present in the vapour layer above the pool water. Due to their volatility, THMs and NCl_3 are the most likely to evaporate and persist in the air, especially indoors, in the absence of proper ventilation.

Several DBPs were shown to have adverse health effects, though evidence is more plentiful for drinking water than for

doi: 10.2166/wh.2018.178

pool water. Long-term exposure to THMs was associated with bladder cancer (Villanueva *et al.* 2015) and colon cancer (Hamidin *et al.* 2008). Chloroform was categorized as a Group 2B carcinogen by the International Agency for Research on Cancer, and it is listed as a reproductive toxicant. Evidence is less readily available for other DBPs, such as chloroamines. Though high concentrations of chloroamines, especially NCl_3, were linked to various symptoms (such as eye irritation), there are no consensus data on the carcinogenicity, genotoxicity, mutagenicity, or reproductive toxicity of the most volatile trichloramine (Florentin *et al.* 2011). Toxic effects of DBPs were recently confirmed also on a metabolomic level (van Veldhoven *et al.* 2018).

Detrimental effects on respiratory function (such as asthma or chronic bronchitis) are also related to the inhalation of DBPs (NCl_3 and THMs). Elite swimmers and staff members (i.e. trainers and life security guards) experience the longest exposure times, and are accordingly considered a potential occupational hazard risk group from DBPs. Children are more vulnerable to health risks associated with swimming, though the outcomes of epidemiological studies on the relationship of swimming and childhood asthma are inconclusive (Villanueva *et al.* 2015). The general conclusion (WHO 2006; CDC 2016) – supported by a current meta-analysis – is that the beneficial effect of physical exercise outweighs the chemical health risk (Valeriani *et al.* 2017).

Despite this positive message, from a regulatory perspective it is necessary to identify and, where possible, mitigate preventable health risks. It is a long-standing challenge to find the correct balance between microbiological safety and the chemical hazards associated with swimming pools. Risk assessment methods provide the tool for quantifying health risks, and there are a number of available models for this purpose (van Hemmen 1999). SWIMODEL is specifically aimed at assessing exposure from water through ingestion, inhalation and dermal (including buccal/sublingual, orbital/nasal and aural surfaces) pathways. The ConsExpo model was originally developed for risk assessment of consumer products, but it is sufficiently flexible in adapting different exposure and uptake scenarios to be used for DBP exposure in swimming pools.

Previous risk assessment studies indicated inhalation to be the most significant exposure route (Chen *et al.* 2011). Most assessment tools use water concentration values and transfer models for the characterization of risk from inhalation exposures, which may lead to widely different risk values based on the underlying algorithm. SWIMODEL allows for direct assessment of inhalation risk from concentrations measured in air, but also uses two different volatilization algorithms, based on Henry's and Raoult's laws. According to the latter, in ideal solutions the vapour pressure of components is in linear correlation with their mole fraction in the solution: $p_i = p_i^0 * x_i$, where p_i is the partial vapour pressure of compound i in the gas phase, p_i^0 is the vapour pressure of the pure compound i and x_i is the mole fraction of compound i in the solution. In ideally weak solutions, p_i^0 is substituted by the compound specific Henry constant (K_H). The applicability of the two algorithms depends on the concentration of the components in water. The algorithm used in ConsExpo is closer to the Raoult's law approach, though it also takes other factors into account (Prud'homme de Lodder *et al.* 2006). The evaporation of a substance from pool water surface is described by the following equation:

$$\frac{dA_{air}}{dt} = K \cdot S \cdot \frac{M}{RT} \cdot (P_{eq} - P_{air}) - Q \cdot V_{room} \cdot C_{air}$$

where C_{air} = concentration of substance in the room air (kg/m^3), S = surface of the pool area (m^2), K = mass transfer coefficient (m/s), M = molecular weight of the substance (kg/mole), R = universal gas constant (J/mol/K), T = temperature (K), P_{eq} = equilibrium vapour pressure (Pa), P_{air} = actual vapour pressure (Pa), V_{room} = room volume (m^3), Q = room ventilation rate (number of air changes per time, L/s).

The above equation describes the amount of substance evaporating from the pool into the air and the subsequent removal of the substance by ventilation of the room air. The evaporation is driven by the difference between the vapour pressure in the room air (P_{air}) and the equilibrium vapour pressure of the substance in the product (P_{eq}). In dilute solutions, the evaporation is approximated by Raoult's law.

The aim of the current study was to: (i) investigate THM formation in 19 Hungarian swimming pools; (ii) calculate and compare the health risk of exposure to DBPs during

swimming using different exposure models; and (iii) cross-check the results of on-site air quality measurements and mass transfer models. Chloroform was used as a model compound for this assessment due to its abundance, the known health effects, and the sparse available toxicological and epidemiological data for the other DBPs. The results are discussed in the light of regulatory implications.

METHODS

Sampling and analysis

Nineteen indoor swimming pools were investigated in the metropolitan area of Budapest, Hungary. Air and water samples were collected. Operational parameters (water treatment and disinfection method, ventilation, bather load, pool size, recirculation) were recorded during sampling.

Air samples were collected by active samplers (SKC Sidekick, SKC Ltd, Dorset, UK). The inlet of the air samplers was placed at a distance of 0.5 m from the edge of the pool, at a height of 0.4 and 1.5 m above the water level, corresponding to the breathing zone of the swimmers and the staff, respectively. For the collection of THMs, air was pumped through Anasorb® CSC adsorbent tubes (SKC Inc., Eighty Four, PA, USA) at a flow rate of 0.16 L/min for 60 min (total of 9.6 L). One sample was collected at each height in the case of each pool. For validation purposes, three samples were collected simultaneously at different spots of the pool in the case of a randomly selected swimming pool.

THMs were extracted from the activated charcoal absorber by 10 min ultrasonicaton in 2 mL carbon-disulphide. For the quantitative analysis of THMs, a gas chromatograph (HP 6890, Hewlett Packard, Palo Alto, CA, USA) coupled with mass spectrometer (HP 5973A, Hewlett Packard, Palo Alto, CA, USA) was used.

Water samples were collected from the pools parallel to the air sampling. Temperature, pH and conductivity were measured using a portable probe (Multi 3430 SET F, WTW, Germany). Total organic carbon (TOC) was measured using a high TOC-II analyser (Elementar, Germany). Absorbable organic halogens (AOX) were measured using a AOX-200 analyser (Mitsubitshi, Japan). Free and combined chlorine levels were determined using DPD colorimetric titration according to the ISO 7393-1:1985 standard. The THMs in water samples were measured with a gas chromatograph (HP 6890, Hewlett Packard, Palo Alto, CA, USA) with a head-space (HP 7694, Hewlett Packard, Palo Alto, CA, USA) and a mass spectrometer (HP 5973A, Hewlett Packard, Palo Alto, CA, USA).

Statistical analysis

Pearson's linear correlation coefficients (r) with a two-tailed test of significance (p) were determined to show relationships among pool chemical parameters and the concentration of chlorination by-products measured in the water and air samples by using the software package of IBM SPSS Statistics for Windows, version 21 (IBM Corp., Armonk, NY, USA).

Risk assessment

Chloroform was used as a model compound for comparing different exposure models. SWIMODEL (US EPA 2003) and ConsExpo (Delmaar & Schuur 2018) were used for the risk assessment.

Human health risk (carcinogenic risk and health risk index) was calculated for adults, 7–10 and 11–14 year olds, elite and recreational swimmers. The median concentration of chloroform measured in the pools was used. The following exposure pathways were estimated: (i) water ingestion during swimming, (ii) absorption through the skin, (iii) inhalation, (iv) absorption through buccal, nasal, ocular and aural surfaces.

Receptor and exposure parameters were used from the corresponding survey of the American Chemistry Council and the US EPA Exposure Factors Handbook (US EPA 2011), adapted to scenarios which are applicable in Hungary (Table 1). Pool parameters (where necessary) were set to 25×15 m surface area, 1.5 m depth and 27.5 °C.

Oral intake was calculated as:

$$LADD_{oral} = \frac{C_W \cdot IR \cdot ET \cdot F \cdot ED \cdot CF}{BW \cdot AT}$$

Table 1 | Receptor and exposure scenario parameters used in exposure assessment

		Recreational swimmer			Elite swimmer		
Receptor and exposure parameters	**Unit**	**Child 7–10 years**	**Child 11–14 years**	**Adult (male)**	**Child 7–10 years**	**Child 11–14 years**	**Adult (male)**
Body weight	kg	30.18	48.17	78.1	30.18	48.17	78.1
Body surface area	cm^2	10,400	15,900	19,400	10,400	14,200	19,400
Exposure duration	year	4	4	30	4	4	22
Exposure frequency	event/year	60	60	60	65	189	238
Inhalation rate	m^3/h	1	1	1	1.9	1.9	3.2
Water ingestion rate	ml/h	50	50	25	25	25	12.5
Exposure time	h/event	1	1	1.3	1	1	1.83

Values are derived from the American Chemistry Council and the US EPA Exposure Factors Handbook (US EPA 2011), adapted to Hungarian scenarios, where necessary. The same parameters were used in SWIMODEL and ConsExpo.

where $LADD_{oral}$ = lifetime average daily dose (oral), C_W = chemical concentration in water (mg/L), IR = intake rate, ET = exposure time (h/event), F = swimming frequency (events/year), ED = exposure duration (year), CF = mass conversion factor from μg to mg (0.001), BW = body weight (kg), AT = averaging time (days) (70 × 365 days = 25,550 days).

Absorption through the skin and the inner dose within the body depends on the ratio of body surface and weight. The same absorption rate was used for all age-groups, in the following formula:

$$LADD_{dermal} = \frac{C_W \cdot K_p \cdot SA \cdot ET \cdot F \cdot ED \cdot CF}{BW \cdot AT}$$

where $LADD_{dermal}$ = lifetime average daily dose (dermal), C_W = chemical concentration in water (mg/L), SA = surface area (m^2), K_p = permeability coefficient (cm/h).

Exposure through inhalation depends on the breathing rate and ventilation rate. Chloroform uptake was estimated from the concentration measured in water by evaporation algorithms (based on Henry's law and Raoult's law). Inhalation exposure was also determined directly from the air measurements:

$$LADD_{inhalation} = \frac{C_a \cdot R \cdot ET \cdot F \cdot ED \cdot CF}{BW \cdot AT}$$

where $LADD_{inhalation}$ = lifetime average daily dose (inhalation), C_a = chemical concentration in air (μg/m^3), R = breathing rate (m^3/hour).

Absorption through the orbital, nasal, buccal, sublingual and aural surfaces may also contribute to the chloroform uptake, as a function of water intake and the absorption factor:

$$LADD_{buccal,sublingual} = \frac{C_W \cdot ET \cdot WI \cdot F \cdot ED \cdot AR \cdot CF}{BW \cdot AT}$$

$$LADD_{orbital,nasal} = \frac{C_W \cdot ET \cdot WI \cdot F \cdot ED \cdot AR \cdot CF}{BW \cdot AT}$$

where $LADD_{buccal,sublingual}$ = lifetime average daily dose (buccal, sublingual), $LADD_{orbital,nasal}$ = lifetime average daily dose (orbital, nasal), WI = water intake (L/h), F = swimming frequency (events/year):

$$LADD_{aural} = \frac{C_W \cdot K_{ow} \cdot K_p \cdot SA \cdot ET \cdot F \cdot ED \cdot CF}{BW \cdot AT}$$

where $LADD_{aural}$ = lifetime average daily dose (aural), K_{ow} = octanol/water partition coefficient (unitless), K_p = permeability coefficient (cm/h), SA = surface area of ears (cm^2).

Individual excess lifetime cancer risk was estimated as:

$$IELCR = SF \cdot LADD$$

where SF is the slope factor ($SF_{oral/dermal}$ is $SF_{inhalation}$).

The other applied model was ConsExpo (Delmaar & Schuur 2018), which was originally designed for exposure analysis of household chemicals. Pool disinfectants are

covered by this category, and ConsExpo contains the appropriate scenarios to be used for the risk assessment of pool disinfectants. Disinfectant Products Fact Sheet of the ConsExpo Web software was used, based on the guidelines of RIVM (Prud'homme de Lodder 2006). Constant rate mode was used for the estimation of inhalation exposure:

$$C_{air} = \frac{A_0 \cdot (w_f / t_r)}{q \cdot V} \cdot (1 - e^{-qt})$$

where C_{air} = concentration of substance in the room air (kg/m^3), t_r = emission duration (s), A_0 = amount of product used (in this case, the mass of chloroform in m^3 pool water) (kg), w_f = weight fraction of the substance in the product (in this case = 1), V = room volume (m^3), q = ventilation rate of the room (number of air changes per time) (L/s).

Based on the information collected from the pool operators, the ventilation rate was set to twice per hour. Mass transfer coefficient was set to 1.0 m/h.

Dermal absorption was estimated using the Diffusion Through Skin module of ConsExpo using the following equation:

$$A_{\text{abs}} = A_{skin} \cdot \left(1 - \exp \cdot \left(-\frac{P \cdot S}{V} \cdot t\right)\right)$$

where A_{abs} = dermal absorption of the substance, A_{skin} = amount of substance on the skin (in this case, the mass of chloroform in 1 cm thick layer of water close around the total skin surface) (kg), V = volume of the substance on the skin (follows from the concentration and the amount on the skin) (m^3), P = permeability of the skin (m/s), S = exposed skin area (m^2), t = exposure time (s).

RESULTS

Swimming pools selected for the study were similar in their main operational characteristics. All pools were operating indoors, and were used primarily for swimming; spa pools and feature pools were excluded from the study. Conventional pool water treatment was used, flocculation with aluminum and filtration on sand or diatoma filters. Sodium hypochlorite was used for disinfection, secondary disinfection (UV or ozone) was not used.

The disinfection regime was effective: all pools were microbiologically compliant (data not shown). THMs were detected in all pools, both in pool water and the air of the pool area. Median concentration of total THMs was 31.0 μg/L (mean 36.4 μg/L, range 13.9–72.4 μg/L), 40.6 μg/m^3 (mean 56.3 μg/m^3, range 13.1–131.5 μg/m^3) and 44.6 μg/m^3 (mean 55.0 μg/m^3, range 11.6–105.3 μg/m^3) in pool water and air at 40 and 150 cm height, respectively. The THM concentration exceeded the Hungarian limit value for pool water (50 μg/L) in four pools (Hungarian Ministerial Decree 1996). Chloroform was the most abundant THM, comprising more than 80% in pool water and air in all but one of the investigated swimming pools (Figure 1). Median chloroform concentration was 28.9 μg/L (mean 31.9 μg/L, range 12.8–71.2 μg/L), 40.6 μg/m^3 (mean 48.2 μg/m^3, range 13.1–102.2 μg/m^3), 35.1 μg/m^3 (mean 48.9 μg/m^3, range 11.6–96.6 μg/m^3) in the pool water, air at 40 and 150 cm, respectively.

Chlorine concentration in the pools was relatively low, free and combined chlorine were 0.61 ± 0.25 and 0.55 ± 0.22 mg/L, respectively. Free chlorine exceeded the Hungarian legislative limit value (1 mg/L) in one pool (Hungarian Ministerial Decree 1996), while the combined chlorine was above the limit (0.5 mg/L) in more than half of the pools (10/19). Total organic carbon concentration was 3.01 ± 1.16 mg/L. Water temperature (28.0 ± 2.8 °C) and pH (8.29 ± 0.47) were both in the range which is favourable for the formation of THMs. Conductivity varied in a wide range (760–3,160 μS). The concentration of adsorbable organic halogens (AOX) was very high (377 ± 168 μg/L); THMs explained only 3–19% of the measured AOX concentration.

The median chloroform concentration in water (28.9 μg/L) was used for risk assessment. The air concentration for the assessment was selected by choosing the pool with median chloroform concentration taking the corresponding concentration measured at 40 and 150 cm height (39.2 and 44.6 μg/m^3, respectively). The former value was used for estimating the exposure of swimmers, while the latter was for staff. The values were close to the median chloroform concentrations in air, but this approach was selected to reflect a realistic exposure scenario.

Both models (SWIMODEL and ConsExpo) confirmed the long-term health risk of chloroform. The individual excess lifetime cancer risk (IELCR) in all age groups

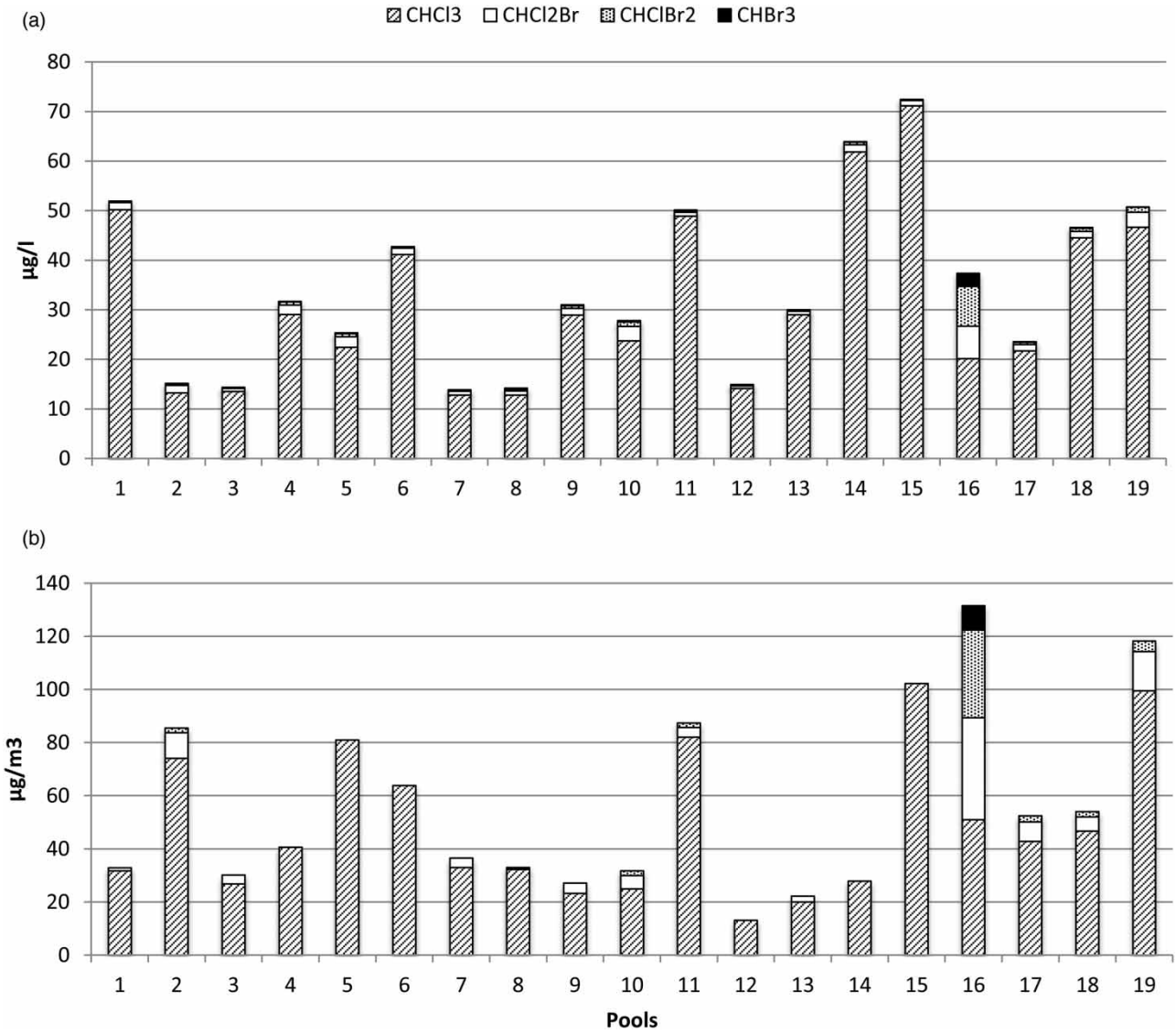

Figure 1 | Concentration of THMs in the investigated indoor swimming pools, in (a) pool water and (b) pool air at 40 cm. $CHCl_3$ – chloroform, $CHBrCl_2$ – bromodichloromethane.

exceeded the accepted level of 10^{-6} for recreational swimmers based on the result of SWIMODEL calculated directly from air measurement value (Table 2). In a scenario of similar lifetime swimming habits, the total IELCR was 7.75×10^{-6}. The calculated risk for elite swimmers was even higher for adults and the total IELCR (5.97×10^{-5}) exceeded even the acceptable occupational risk level (10^{-5}). Inhalation was responsible by far for the highest proportion of exposure, 93–94% in recreational and 97–98% in elite swimmers. Dermal exposure made up the rest of the total intake, while oral, buccal, nasal and aural exposure was negligible. For elite swimmers, lifetime adjusted risk through dermal exposure was in itself close to the acceptable level (8.91×10^{-7}).

SWIMODEL also allows for estimating inhalation exposure from concentrations measured in water by a Henry's and Raoult's law based model. The calculated concentration in air was 4,356 µg/m^3 by the Henry model, and 5.5 µg/m^3 by the Raoult model; the former being two orders of magnitude higher, the latter one order of magnitude lower than the actual measured concentration. The associated inhalation risk showed the same difference, as its correlation

Table 2 | IELCR of chloroform from swimming exposure of recreational and elite swimmers in different age groups by exposure route

	Recreational swimmer				Elite swimmer			
Age (years)	**7–10**	**11–14**	**Adult**	**Total**	**7–10**	**11–14**	**Adult**	**Total**
SWIMODEL								
Oral	3.22×10^{-8}	1.49×10^{-8}	2.64×10^{-8}	7.35×10^{-8}	1.52×10^{-8}	2.28×10^{-8}	5.39×10^{-8}	8.44×10^{-8}
Dermal	5.95×10^{-8}	3.78×10^{-8}	1.82×10^{-7}	2.80×10^{-7}	2.81×10^{-8}	1.15×10^{-7}	7.47×10^{-7}	8.91×10^{-7}
Inhalation	2.33×10^{-6}	1.08×10^{-6}	3.83×10^{-6}	7.25×10^{-6}	2.09×10^{-6}	6.29×10^{-6}	5.02×10^{-5}	5.85×10^{-5}
Buccal	3.22×10^{-8}	1.49×10^{-8}	2.64×10^{-8}	7.35×10^{-8}	7.60×10^{-9}	2.28×10^{-8}	5.39×10^{-8}	8.44×10^{-8}
Nasal	3.22×10^{-8}	1.49×10^{-8}	2.64×10^{-8}	7.35×10^{-8}	3.78×10^{-9}	1.14×10^{-8}	2.70×10^{-8}	4.22×10^{-8}
Aural	2.14×10^{-9}	9.92×10^{-10}	3.50×10^{-9}	6.63×10^{-9}	1.01×10^{-9}	3.03×10^{-9}	1.44×10^{-8}	1.84×10^{-8}
All	2.49×10^{-6}	1.16×10^{-6}	4.10×10^{-6}	7.75×10^{-6}	2.15×10^{-6}	6.46×10^{-6}	5.10×10^{-5}	5.97×10^{-5}
ConsExpo								
Oral	3.21×10^{-8}	1.49×10^{-8}	2.63×10^{-8}	7.32×10^{-8}	1.52×10^{-8}	2.30×10^{-8}	5.41×10^{-8}	9.23×10^{-8}
Dermal	5.84×10^{-8}	3.79×10^{-8}	1.79×10^{-7}	2.76×10^{-7}	2.78×10^{-8}	1.10×10^{-7}	7.00×10^{-7}	8.38×10^{-7}
Inhalation	1.89×10^{-6}	1.14×10^{-6}	3.18×10^{-6}	6.21×10^{-6}	2.87×10^{-6}	6.44×10^{-6}	3.31×10^{-5}	4.24×10^{-5}
Buccal	NA	NA	NA	NA	NA	NA	NA	NA
Nasal	NA	NA	NA	NA	NA	NA	NA	NA
Aural	NA	NA	NA	NA	NA	NA	NA	NA
All	1.98×10^{-6}	1.19×10^{-6}	3.39×10^{-6}	6.56×10^{-6}	2.92×10^{-6}	6.58×10^{-6}	3.38×10^{-5}	4.33×10^{-5}

IELCR was calculated from the mean chloroform concentration measured in pool air at 40 cm height by SWIMODEL (upper) and the mean chloroform concentration measured in pool water by ConsExpo (lower).

with the chloroform concentration in this range is linear (Table 3). The total lifetime risk was close to the accepable level for both recreational and elite swimmers even with the more realistic Raoult's model (9.83×10^{-7} and 7.98×10^{-6}, respectively), while Henry's model resulted in extreme values (7.78×10^{-4} and 6.29×10^{-3}, respectively).

ConsExpo only calculates from water concentration, and considers oral, dermal and inhalation exposure (buccal, nasal and aural pathways are not included). Nevertheless, ConsExpo yielded very similar values to those calculated by SWIMODEL from air concentration (Table 2).

The risk for children was always lower than for adults due to the shorter exposure times (Table 2). However, greater sensitivity resulted in higher risk values in younger children (7–10 vs. 11–14).

Pool staff members (e.g. life-guards or trainers) represent a different scenario. In their case, only inhalation exposure

Table 3 | IELCR of chloroform from inhalation exposure during in recreational and elite swimmers in different age groups

	Recreational swimmer				Elite swimmer			
Age (years)	**7–10**	**11–14**	**Adult**	**Total**	**7–10**	**11–14**	**Adult**	**Total**
SwimodelA	2.33×10^{-6}	1.08×10^{-6}	3.83×10^{-6}	7.25×10^{-6}	2.09×10^{-6}	6.29×10^{-6}	5.02×10^{-5}	5.85×10^{-5}
SwimodelH	2.51×10^{-4}	1.16×10^{-4}	4.12×10^{-4}	7.78×10^{-4}	2.25×10^{-4}	6.75×10^{-4}	5.39×10^{-3}	6.29×10^{-3}
SwimodelR	3.17×10^{-7}	1.46×10^{-7}	5.19×10^{-7}	9.83×10^{-7}	3.20×10^{-7}	8.53×10^{-7}	6.80×10^{-6}	7.98×10^{-6}
ConsExpo	1.89×10^{-6}	1.14×10^{-6}	3.18×10^{-6}	6.21×10^{-6}	2.87×10^{-6}	6.44×10^{-6}	3.31×10^{-5}	4.24×10^{-5}

IELCR is calculated using the following models: SWIMODEL, using the mean chloroform concentration measured in pool air at 40 cm height (SwimodelA); SWIMODEL, using the mean chloroform concentration measured in pool water and the mass transfer model based on Henry's low (SwimodelH); SWIMODEL, using the mean chloroform concentration measured in pool water and the mass transfer model based on Raoult's low (SwimodelR); ConsExpo, using the mean chloroform concentration measured in pool water and the mass transfer calculation (ConsExpo).

is relevant, but exposure is longer (the usual receptor value is 8 h/event, inhalation rate 0.8 m^3/h, 250 times a year for 30 years), resulting in high IELCR values (6.64×10^{-5}), well above the acceptable level of occupational risk.

DISCUSSION

The adverse long-term health effect of THMs, and especially chloroform, is undebated by most studies, however, the estimated levels of excess risk are very diverse (Table 4). All published studies agree that it exceeds the acceptable level of 10^{-6}, but IELRC values up to 10^{-3} were reported previously (Lee *et al.* 2009), although the concentrations measured in the pool water were similar or lower than the present study. The wide range of IELCR estimates is partly due to differences in the exposure scenarios (e.g. number and duration of swim events per year), but predominantly derived from the the mass transfer estimates between water and air. In our assessment, the SWIMODEL calculation from air concentration and the ConsExpo model reached approximately the same conclusion, with the estimated excess risk in the range of $1.16–4.10 \times 10^{-6}$ for all age groups of recreational swimmers and up to 5.10×10^{-5} for elite swimmers (adults) (Table 2).

SWIMODEL (developed by US EPA in 2003) is the most widely used model for risk assessment of indoor pools. It offers a screening type assessment for chemicals and their degradation products in pool water, and a choice of different exposure models, for example conservative or worst-case exposure calculation. The model extends beyond the classic oral, dermal, and inhalation pathways, covering buccal/sublingual, orbital/nasal and aural uptake as well, which may also be relevant during swimming. Exposure input parameters of recreational and elite swimmers are derived from peer-reviewed publications.

ConsExpo is the most recent tool (the new web version released in February 2018) to assess the risk of exposure to consumer products, including swimming pool disinfectants. The scenarios offered by ConsExpo adequately describe the inhalation, dermal and oral exposure during a pool visit. It does not address exposure through the mucosa, but this did not lead to a significant difference between the risk estimates by the two models for chloroform. ConsExpo is the model recommended for chemical safety assessment by the European Chemicals Agency. In the present study, it gave the best estimate for chloroform in air calculated from the concentration measured in water.

Using mass transfer models from water to air in SWIMODEL based on Henry's and Raoult's laws yielded four orders of magnitude difference both in air concentration estimates and the corresponding IELCR values, the former over-, while the latter underestimating the results of air measurements (Table 3). Previous studies using SWIMODEL relying on the Henry mass transfer model also arrived at much higher IELCR values (Table 4, Lee *et al.* 2009), similar to those obtained in the present study by the same method ($10^{-3}–10^{-4}$ excess risk). Lourencetti *et al.* (2012) also found that the actual DBP concentrations measured in air amounted only to 6–11% of the concentration expected by Henry's law. Other models were suggested to give a closer estimate of exposure, such as the fugacity model (Catto *et al.* 2012a), physiologically based toxicokinetic modeling (Catto *et al.* 2012b), multi-pathway

Table 4 | Different IELCR estimates of chloroform from swimming exposure in previous studies

Chloroform concentration (water, μg/L)	Chloroform concentration (air, μg/m^3)	Inhalation slope factor	IELCR from inhalation	Reference
40.7	nd	8.10×10^{-2}	1.15×10^{-3}	Lee *et al.* (2009)
9.81	13.97	8.05×10^{-2}	2.80×10^{-4}	Chen *et al.* (2011)
3.00	5.34	Nd	1.05×10^{-5}[a]	Chowdhury (2015)
40–55[a]	nd	8.10×10^{-2}	5.16×10^{-4}	Wang *et al.* (2014)
20.32	Nd	6.1×10^{-3}[b]	4.34×10^{-5}	Panyakapo *et al.* (2008)

Where results were differentiated by gender and age groups, values for adult males are used for comparison.
[a]All THMs.
[b]Oral slope factor.

exposure modeling (Chen *et al.* 2011) or purpose-designed exposure models (Hsu *et al.* 2009), but the outcome risk values still vary between 10^{-6} and 10^{-4} (Table 4). The extent of the difference derived from the different models applied, and its implications for the final risk evaluation, need to be considered in future for other DBPs as well.

Until recently, it was a common agreement that inhalation is the main uptake pathway of chloroform, comprising up to 99% (Chen *et al.* 2011), and this was also confirmed by the present results. Some studies, however, suggest comparable or even higher risk of dermal exposure (Aprea *et al.* 2010; Chowdhury 2015). The difference mainly lies in the permeability coefficient used by the various assessments (0.0089 vs. 0.002 m/h). Analysis of different exposure scenario-based biomonitoring (exhaled breath concentration) indicated the comparable importance of dermal exposure to inhalation (Aprea *et al.* 2010).

The identified health hazards and the difficulties of its accurate estimation call for the overview of regulations to identify whether they provide an adequate level of protection for the pool visitors and workers. Most regulations regarding disinfection and DBPs (where applicable) relate to levels of free and combined chlorine. Hungarian legislation sets the limit value for free chlorine (1 mg/L maximum, without a minimum) below and for combined chlorine (0.5 mg/L) above the action levels defined in most internationally recognised guidelines (Hungarian Ministerial Decree 1996). According to WHO Guidelines on Recreational Water Quality, free chlorine up to 5 mg/L does not result in adverse health effects or bather complaints, while 0.2 mg/L combined chlorine (if mainly in the form of NCl_3) is an irritant and may lead to an unpleasant odour. The CDC Model Aquatic Code recommends 1–3 mg/L free chlorine (below 1 mg/L, the inactivation of *Cryptosporidium* oocysts is compromised), and sets the action level on combined chlorine to 0.4 mg/L as a technically feasible solution, while agreeing that 0.2 mg/L is optimal for health protection (CDC 2016). The UK Pool Water Technical Advisory Group suggests free chlorine between 0.5 and 1 mg/L as an optimum, and up to 3 mg/L in special cases (e.g. spas) (PWTAG 2016). Combined chlorine cannot exceed 1 mg/L and half of the concentration of free chlorine.

In the present study, the free chlorine levels were well within (or even below) the nationally or internationally accepted range, and the combined chlorine levels indicated overchlorination in more than half of the investigated pools.

Most countries or organisations regulate THMs in drinking water: the European Drinking Water Directive parametric value is 100 μg/L, the USEPA recommendation is 80 μg/L cumulatively for the four chlorinated and brominated compounds. The WHO Guidelines on Drinking Water Quality set separate guide values for each compound: 300 μg/L for chloroform, 100 μg/L for bromoform and DBCM, and 60 μg/L for BDCM (WHO 2011). However, there are only a limited number of examples for the regulation of THMs in swimming pools. For most parameters, where oral exposure is the relevant pathway, swimming pools comprise lower hazard due to the difference in the usual intake volume (2 L vs. 30–50 mL), and health-related limit values are higher in pool regulation. THMs are different in this regard, since the largest proportion of the uptake is through inhalation. Hungarian legislation defines the same, internationally strict regulatory limit value, 50 μg/L for drinking water and pool water. In the present study, only four of the 19 investigated pools did not comply with this value, but 17 were over the even more stringent German standard of 20 μg/L.

Physico-chemical characteristics of the pool water were shown previously to influence the formation of DBPs (Manasfi *et al.* 2017). Organic matter and free chlorine are usually the key determinants. However, in the current study only AOX correlated to the TOC concentration, while chloroform and THMs to free chlorine (Table 5). Combined chlorine, on the other hand, showed strong ($p < 0.01$) correlation to all three. Increase in conductivity – just like high TOC – usually indicates bather overload or insufficient water replenishment. This was observed in seven of the 19 pools. Conductivity also correlated only to AOX (Table 5).

The pH of most pools (13/18) was above the usually recommended range (6.5–7.8 in the Hungarian regulation), but there was no correlation between the pH and any of the investigated DBPs (Table 5). According to previous studies, THM formation usually increases at higher pH, however, these conditions were shown to produce lower genotoxicity than other DBP, such as HANs, which are generated at lower (<7.0) pH (Hansen *et al.* 2012).

The indoor air quality of a swimming pool is generally not regulated. If there are guidance or regulatory values

Table 5 | Correlation of chloroform, THMs and AOX concentration measured in pool water to physico–chemical characteristics of water by Pearson correlation (two-tailed significance)

Water quality parameter	Chloroform	Total THMs	AOX
TOC	0.364 (0.125)	0.410 (0.81)	0.801 (0.000)[a]
pH	0.285 (0.104)	0.402 (0.088)	0.258 (0.287)
Conductivity	0.066 (0.788)	0.170 (0.486)	0.718 (0.001)[a]
Free chlorine	0.595 (0.007)[a]	0.526 (0.021)[b]	0.296 (0.218)
Combined chlorine	0.648 (0.003)[a]	0.704 (0.001)[a]	0.855 (0.000)[a]

[a]Correlation is significant at the 0.01 level (two-tailed).
[b]Correlation is significant at the 0.05 level (two-tailed).

available, they usually focus on chloroamines: an NCl_3 action level of 500 μg/m^3 is recommended by several organizations (WHO 2006; CDC 2016). The action level recommendation is based on a limited number of epidemiological studies (Villanueva *et al.* 2015), however, there are strong limitations for the use of NCl_3 for regulatory purposes: there is no available consensus method for its routine monitoring, and the toxicology background is also weak. THMs on the other hand are more readily monitored, and their omnibus presence in the air of chlorinated pools deems them suitable as candidate markers of excess DBP formation and/or the absence of proper ventilation. The present results underline that water measurements cannot substitute for air quality monitoring. The concentrations measured in air at two different heights showed strong correlation (two-tailed Pearson correlation 0.772, $p < 0.01$), and between water and the results at 150 cm (0.599, $p < 0.01$), but the relationship observed between the concentration in water and air at 40 cm was weak (0.441, $p = 0.058$).

The risk assessment indicated that even if the water is compliant with the limit values (which are health based), the IELCR still exceeds the generally accepted level of 10^{-6}. The wide variation of results by different evaporation models also suggest a necessity to perform air measurements.

CONCLUSIONS

The health risk associated with DBP exposure in swimming pools has a growing body of evidence, but its estimate depends largely on the risk assessment models. We have observed weak correlation between concentrations measured in water and air, suggesting the need for air quality monitoring as a regulatory tool, since inhalation is still assumed to be the most relevant pathway. When relying solely on water measurements, most studies overestimate the risk due to the applied mass transfer models, but in the current assessment the ConsExpo model gave good estimates on the evaporation of chloroform.

Regulation (either in a legal from or as best practice guidance or standards) is an important tool for health protection. Based on the current results, not even the most stringent German limit value for THMs in pool water (20 μg/L) provides a sufficient level of protection, and in air, chloroform above 5 μg/m^3 already leads to IELCR above 10^{-6}. Better management practices of both water and air are necessary to reduce the risk to bathers' health.

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First received 16 July 2018; accepted in revised form 3 September 2018. Available online 19 October 2018