

Article

## Occurrence and Spatial and Temporal Variations of Disinfection By-Products in the Water and Air of Two Indoor Swimming Pools

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**Abstract:** In order to improve disinfection by-product (DBP) exposure assessment, this study was designed to document both water and air levels of these chemical contaminants in two indoor swimming pools and to analyze their within-day and day-to-day variations in both of them. Intensive sampling was carried out during two one-week campaigns to measure trihalomethanes (THMs) and chloramines (CAMs) in water and air, and haloacetic acids (HAAs) in water several times daily. Water samples were systematically collected at three locations in each pool and air samples were collected at various heights around the pool and in other rooms (e.g., changing room) in the buildings. In addition, the ability of various models to predict air concentrations from water was tested using this database. No clear trends, but actual variations of contamination levels, appeared for both water and air according to the sampling locations and times. Likewise, the available models resulted in realistic but imprecise estimates of air contamination levels from water. This study supports the recommendation that suitable minimal air and water sampling should be carried out in swimming pools to assess exposure to DBPs.

**Keywords:** disinfection by-products; swimming pool; exposure assessment; water and air monitoring; spatial and temporal variations; volatilization model

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## 1. Introduction

It is well known that the disinfection of swimming pool water generates by-products (DBPs) as a result of chemical interactions between chlorine and nitrogenous or organic matters that come from swimmers or are naturally present in water [1]. Indeed, both the high quantities of disinfectant required to ensure protection of bathers against microbiological risks and the continuous pool loading with organic and nitrogenous precursors (e.g., body fluids, skin particles, hair, and cosmetics) from bathers contribute to the formation of high quantities of these DBPs. Whereas water recirculation tends to concentrate the non-volatile DBPs in the pool, the turbulence generated by swimmers promotes the diffusion of volatile compounds from water into the ambient air [2]. Moreover, in indoor swimming pools in particular, ventilation conditions may not necessarily be sufficient enough to efficiently remove DBPs in the air.

Among the numerous DBPs ( $n > 600$ ) and apart from the emerging ones newly discovered thanks to analytical progress [3,4], three main classes are traditionally identified: trihalomethanes (THMs)—including chloroform (TCM), dichlorobromomethane (DCBM), chlorodibromomethane (CDBM) and bromoform (TBM), haloacetic acids (HAAs) and chloramines (CAMs)—including monochloramine (MCAM), dichloramine (DCAM) and trichloramine (TCAM). THMs are known to volatilize easily from water into ambient air, contrary to HAAs. As for CAMs, MCAM and TCAM are usually the main compounds encountered in water and air, respectively [5].

THMs and HAAs are suspected to have various health effects, mainly regarding carcinogenic risk (e.g., bladder cancer) or adverse reproductive outcomes (e.g., intra-uterine growth retardation) [6,7]. However, these issues have been investigated primarily for exposure involving household drinking water use activities (e.g., consumption, showering) without (or seldom) accounting for exposure resulting from swimming pool attendance. Irritation (respiratory and ocular) associated with exposure to CAMs (particularly TCAM) among swimming pool attendees or workers are well acknowledged [8–15]. Likewise, currently growing interest concerns potential allergic and asthmatic impacts of these contaminants, especially on the young population (e.g., baby swimmers) [16–22]. In this context, various actions, such as the use of dechloramination devices, are currently considered to reduce CAM exposure which is of prime interest and worth to be evaluated first as short-term health effects could be produced. Nevertheless, some reports suggest that this technology could promote the formation of other DBPs, especially THMs [23,24], and so the long-term health effects relative to a potential carcinogenic risk.

More recently, the potential mutagenicity and genotoxicity of swimming pool water, possibly linked to DBPs, have been considered [3,4,25–27] and there is growing international interest in assessing DBP exposure in swimming pools and related risks [20,28–36].

In the Province of Quebec (Canada), few studies have documented the occurrence of DBPs in swimming pools. To our knowledge, only one study by Lévesque *et al.* [37], comparing the occurrence

of health complaints between two groups of swimmers and soccer players, reported levels of CAM in the water (450  $\mu\text{g/L}$ –1,030  $\mu\text{g/L}$ ) and air (260  $\mu\text{g/m}^3$ –410  $\mu\text{g/m}^3$ ) of seven swimming pools. This study showed a link between irritation symptoms, more frequently reported among swimmers, and CAM concentrations in the air; it also showed that more respiratory complaints were experienced at levels above 370  $\mu\text{g/m}^3$ . This value, under the reference limit of 500  $\mu\text{g/m}^3$  suggested by a French study [38], is close to, but still above, the value of 300  $\mu\text{g/m}^3$  proposed by Parrat [39] and also above the toxicity reference value of 0.4  $\mu\text{g/m}^3$  proposed by Bonvallot *et al.* [40]. In another previous study, Lévesque *et al.*, documented the water and air concentrations of TCM in eight various indoor swimming pools located in the Quebec City area while they were assessing associated exposure and risk among competitive and leisure swimmers [41]. Reported mean TCM water and air concentrations ranged from 18  $\mu\text{g/L}$  to 80  $\mu\text{g/L}$  and from 78  $\mu\text{g/m}^3$  to 329  $\mu\text{g/m}^3$ , respectively. More recently, Simard reported the monthly evolution (during 12 months) of DBPs levels in water samples from 15 indoor and 39 outdoor swimming pools in Quebec City [5]. The THM levels ranged between 17.5  $\mu\text{g/L}$  and 113.5  $\mu\text{g/L}$  (mean = 44  $\mu\text{g/L}$ ) in indoor swimming pools and reached up to 300  $\mu\text{g/L}$  in outdoor pools. These levels exceed the regulatory standard adopted in Germany that requires THM pool water concentration under 20  $\mu\text{g/L}$ . Simard reported CAMs ranging between 300 and 1,700  $\mu\text{g/L}$ , and between 10 and 800  $\mu\text{g/L}$  for indoor and outdoor pools, respectively [5]. These authors also observed an important accumulation of HAAs with levels up to 1,100  $\mu\text{g/L}$  and above 2,200  $\mu\text{g/L}$  in indoor and outdoor pools, respectively. However, their study was limited to water contamination only. Indeed, only a limited number of studies have explored the relationship between air and water contamination by DBPs [29,41–45]. The study of Hamel is of particular interest, as the authors examined the evolution of THM and CAM levels in the water and air of four swimming pools in France [42].

How DBPs are distributed into and between various media (*i.e.*, water and air) of a swimming pool and the extent to which contamination fluctuates in time are issues that continue to require investigation in order to improve DBP exposure assessment through suitable environmental monitoring and/or predictive modeling strategies. HAAs, more particularly, should be investigated, since little data regarding these DBPs are currently available [5,46,47]. The modeling of THM volatilization and resulting levels in water and air, influenced by numerous factors (e.g., number of swimmers, ventilation, and water turbulence), is another challenging concern. Hsu *et al.* [48] and Dyck *et al.* [49] have proposed two interesting approaches which focus on TCM and whose reliability should be further explored. Hsu *et al.* have developed a robust mathematical model accounting for environmental conditions and occupant activities and using computational fluid dynamics to predict TCM concentrations into the indoor swimming-pool air. However, this model requires numerous assumptions, particularly concerning the description of the indoor airflow patterns, which make its use difficult. The works of Dyck *et al.* resulted in an easier equation as described in a following section.

In this context, the present study aimed at documenting the variability of the occurrence of the main DBPs in water (THMs, HAAs and CAMs) and in the ambient air (THMs, CAMs) of two indoor swimming pools in Quebec City through intensive monitoring campaigns. The study examined the spatial variations of DBPs (*i.e.*, in the pool water, in the air around the pool and in premises) as well as within-day and day-to-day variations of DBPs in both the water and the air. The database developed was then used to test various THM volatilization models. We also sought to establish the extent to

which frequent or occasional water and air samplings might be required in order to properly assess DBP exposure in pools and/or for risk assessment and potential regulatory purposes.

## 2. Methodology

### 2.1. Study Sites

For this investigation, two public indoor swimming pools ([A] and [B]) in Quebec City (Canada) were selected among those previously investigated by Simard [5]. Sites with a basic configuration consisting of a single pool were preferred. Technical information relative to each swimming pool is presented in Table 1.

**Table 1.** Technical information on configuration and water treatment in each studied swimming pool.

Parameters	Pool [A]	Pool [B]
Dimensions ([m] × [m])	25 × 14.4 (360 m <sup>2</sup> )	25 × 12 (300 m <sup>2</sup> )
Pool volume (L)	682,000	860,000
Water Disinfectant	Sodium hypochlorite (automated injection)	
Indicative DBP concentrations in water (µg/L) reported by Simard <i>et al.</i> [5]		
THM	26.1	28.6
HAA	267.0	388.9
CAM	574.9	493.1

### 2.2. Sampling Program for Air and Water

Two consecutive sampling sessions were carried out during the first week of June (S1) and that of July (S2) 2010, respectively. The same sampling programs were carried out at the same time in both swimming pools.

#### 2.2.1. Air Sampling (THMs and TCAM)

Basically, the program of each session consisted of four 95 min-sampling periods/day during five consecutive days (from Monday to Friday, between 9:30 am and 4:30 pm approximately). For each period, 95 min-integrated sampling was used to estimate THM levels in the air. Samples were collected at 30 cm and 150 cm above the surface water on the pool edge in the middle of the swimming pool. Depending on the day, other air samples were also collected near the breathing zone (150 cm) in various rooms, including men's and women's changing rooms, lifeguard's office, administrative office or operational room. The air sampling strategy differed slightly for TCAM, whose concentrations in the air were measured only two times/day. For this parameter, 120-min integrated air samples were collected along the pool edge in the middle of the swimming pool 150 cm above the water surface once in the morning (through the first and second period) and once in the afternoon (through the third and fourth period).

### 2.2.2. Water Sampling (THMs, HAAs and CAMs)

For water, duplicate spot samples were collected at midway through each period in three different locations around the pool: the shallow end, the middle of the pool and the deep end. All water samples were taken at approximately 30 cm under the water surface and were analyzed for THMs and HAAs. Only samples in the middle of the pool were collected for CAM analysis (as well as for other physicochemical parameters: pH, temperature, free residual chlorine and total chlorine).

### 2.3. Analytical Procedures

#### 2.3.1. Air Samples

Air samples for THM measurements were collected using a pump (AirLite Sampler Model 110-100, SKC Inc., Eighty Four, PA, USA) at 165 mL/min flow rate for 95 min through adsorption into activated carbon tubes (ORBO™ 32 Small Activated Coconut Charcoal (20/40), 100/50 mg; Sigma-Aldrich, #cat 20267-U). Tubes were sealed and stored on ice for analysis within three days of sampling. A solution of carbon disulfide (1 mL) was used for desorption (carbon disulfide, ACS reagent, ≥99.9%; Sigma-Aldrich, #cat. 180173). After ultrasound heating during 30 min (Branson Bransonic 1200 Ultrasonic Cleaner Heated Water Bath), 1 µL was injected into a gas chromatograph combined with electron capture detector (CP-3800, Varian. He: 1.0 mL/min, column VF5ms 30 m [L] × 0.25 mm [ID] × 0.25 µm [Film thickness]). The limits of detection (LOD) were 0.69, 0.102, 0.095 and 0.112 µg/m<sup>3</sup> for TCM, DCBM, CDBM and TBM, respectively. TCAM in air was analyzed according to the reference method developed by Héry *et al.* [38]. Requisite sampling cassettes were prepared and analyzed by the Laboratoire d'études et de recherche en environnement et santé of the École des hautes études en santé publique de Rennes (LERES, EHESP, France) following NF ISO 10304-1 procedure. The LOD was 50 µg/m<sup>3</sup>. Air samples for TCAM measurements were pumped at a 1 L/min flow rate for 120 min through the cartridges. All pumps were calibrated each morning prior to sampling.

#### 2.3.2. Water Samples

For THM and HAA analyses, 40-mL glass vials with screw caps and polytetrafluoroethylene-lined silicone septa were prepared beforehand with a chlorine-quenching agent (166 µL of ammonium chloride at 30 g/L) to prevent further chlorinated DBP formation. After careful collection to prevent bubble formation, the samples were kept in an icebox and then stored at 4 °C in the laboratory until analysis. THMs in water were extracted by solid phase microextraction SPME technic with PDMS 100 µm fiber (Supelco, #cat. 57341) and determined using a gas chromatograph (Varian GC model 3900; column Factor Four VF-5ms 30 m [L] × 0.25 mm [ID] × 0.25 µm [Film thickness]) with ion-trap mass spectroscopy detection (Varian MS model 2100T). HAAs were measured according to EPA method 552.2 [50] using gas chromatograph (Perkin Elmer Autosystem XL included column Zebron 1701: 30 m [L] × 0.32 mm [ID] × 0,25 µm [Film thickness]) with electron capture detector (GC-ECD) (methane-argon gas with purity of 99.99%). HAAs were extracted using methyl-tert-butyl-ether (Fisher Scientific HPLC grade, #cat. E127-4), using 2-bromopropionic acid as extraction standard “surrogate” (Supelco, #cat. 47645). For quality assurance, field blanks, duplicate samples and

internal standards (1,2,3-Trichloropropane, Supelco #cat. 47669-U) in each sample were conducted. The limits of detection (LOD) ranged between 0.6 and 1.1 µg/L for THMs and between 0.1 and 1.6 µg/L for HAAs. For CAMs, the LOD was 10 µg/L-Cl<sub>2</sub>.

Water samples for physicochemical measurements were collected in 250 mL plastic bottles (Nalgene). Inorganic CAMs in water were estimated by spectrophotometry (HACH DR 5000, 515 nm-reading, 1 cm-cell) according to 4500-Cl-G DPD method [51]. Solid DPD (DPD free chlorine reagent HACH, #cat. 21055-28) was used instead of liquid DPD prescribed in the 4500-Cl-G method. Apart from CAMs, other physicochemical measurements carried out included pH (Denver Instruments Model-AP 15), temperature (alcohol thermometer), and, according to 4500-Cl-F method, free residual chlorine (HACH DR 890-MTH 8021) and total chlorine (HACH DR 890-MTH 8167).

#### 2.4. Volatilization Models

Two tools were compared for the predictions of TCM air concentrations from water levels: (i) the volatilization model (VTM) integrated into the physiologically based toxicokinetic modeling developed by Haddad *et al.* [52] and based on the work of McKone [53,54] and (ii) the level III fugacity model (FUG) currently proposed by Dyck *et al.* [49]. The VTM model assumes, under steady-state conditions, that the concentration in the air of a room depends primarily on the ventilation rate of the room and the chemical input into the room, the latter depending on the concentration of the chemical in the water, the volume of water used and the duration of water use. An efficiency factor is used to quantify the water-to-air transfer. First, we used the by-default parameterization of the model as originally assumed by Haddad *et al.*, and then we adjusted the following parameters to better fit its predictions on the empirical data: flow = 7.42 L/min; ventilation = 0.66 m<sup>3</sup>/min; efficiency factor = 0.390 (unitless). The FUG was developed on the basis of Mackay's work [55] and is based on the concept of fugacity. It accounts for interactions between several multimedia environments (*i.e.*, water, air and also human organism), including flow and non-equilibrium conditions. From this process, a linear equation was reported by Dyck *et al.*, to predict TCM air concentration (TCMa in µg/m<sup>3</sup>) from TCM water concentrations (TCM in µg/L):  $TCMa = -0.039 + 4.229 \times TCM$ .

#### 2.5. Statistical Analysis

Student t-tests (with Satterthwaite correction for unequal variances) were used to compare DBP levels between [A] and [B] as well between S1 and S2. First, relationships between the various DBP concentrations were studied using scatter plots and secondly, by calculating the Pearson correlation coefficients. A mixed analysis of variance (ANOVA) model was adjusted to these concentrations separately for each pool. The fixed factors are the sampling place, time and day. The random factor is the session. The MIXED procedure of the SAS program was used for the analyses [56]. Variance was modeled with the GROUP statement of the function REPEATED to ensure that homogeneity of variance and degrees of freedom were adjusted accordingly. We selected the best model of variance using the Akaike Information Criteria (AIC). In some cases, the normality assumption was not verified, due to some outliers. However the analysis without the outliers led to similar results and the same conclusion. The significance levels used for these analyses were 0.01 for the ANOVAs and 0.05

otherwise. Data under the LOD were substituted by  $\text{LOD}/(2^{0.5})$ . The comparisons between the volatilization models were based on the calculation of least square residual means.

### 3. Results

Table 2 provides an overview of the concentrations of DBPs measured in pools [A] and [B] during the two sampling sessions. Table 3 presents the values of the physicochemical parameters and cumulative number of bathers during those sessions.

**Table 2.** DBP concentrations in pool water and air of [A] and [B] (all samples).

	<i>n</i> <sup>a</sup>	Pool [A]		<i>n</i> <sup>a</sup>	Pool [B]	
		Mean ( $\pm$ SD)	[Min–Max]		Mean ( $\pm$ SD)	[Min–Max]
TTHMs <sup>b</sup> in water ( $\mu\text{g/L}$ )	119	28.8 ( $\pm$ 5.8)	[13.3–46.0]	116	24.3 ( $\pm$ 5.5)	[10.4–38.1]
TCM <sup>b</sup>		28.8 ( $\pm$ 5.8)	[13.3–46.0]		24.3 ( $\pm$ 5.5)	[10.4–38.1]
HAA9 <sup>b</sup> in water ( $\mu\text{g/L}$ )	120	217.6 ( $\pm$ 46.5)	[111.3–390.4]	120	257.8 ( $\pm$ 38.6)	[138.6–365.0]
DCAA <sup>c</sup>		93.3 ( $\pm$ 28.6)	[48.0–191.5]		112.1 ( $\pm$ 21.8)	[69.1–163.2]
TCAA <sup>b</sup>		107.5 ( $\pm$ 23.0)	[54.0–190.7]		128.9 ( $\pm$ 22.2)	[59.2–201.0]
BCAA		1.81 ( $\pm$ 0.80)	[0.6–3.0]		1.8 ( $\pm$ 0.9)	[0.4–2.9]
BDCAA		15.0 ( $\pm$ 6.7)	[<LOD–23.6]		15.1 ( $\pm$ 6.2)	[6.1–23.5]
CAMs <sup>c</sup> in water ( $\mu\text{g/L Cl}_2$ )	39	689 ( $\pm$ 166)	[376–981]	40	526.9 ( $\pm$ 113)	[268–802]
MCAM <sup>c</sup>		323 ( $\pm$ 55)	[188–434]		284 ( $\pm$ 81)	[<LOD–450]
DCAM		25 ( $\pm$ 97)	[<LOD–593]		11 ( $\pm$ 21)	[<LOD–70]
TCAM <sup>b</sup>		341 ( $\pm$ 183)	[<LOD–650]		232 ( $\pm$ 146)	[<LOD–557]
TTHMs <sup>c</sup> in air ( $\mu\text{g/m}^3$ )	78	130.3 ( $\pm$ 49.1)	[47–311]	76	90.2 ( $\pm$ 33.1)	[33.7–180.3]
TCM <sup>c</sup>		128.7 ( $\pm$ 48.5.2)	[46.4–306.7]		89.1 ( $\pm$ 32.8)	[33.6–177.7]
DCBM <sup>c</sup>		1.55 ( $\pm$ 0.7)	[<LOD–4.3]		1.1 ( $\pm$ 0.5)	[<LOD–2.6]
TCAM <sup>b</sup> in air ( $\mu\text{g/m}^3$ )	19	220 ( $\pm$ 68)	[110–350]	18	139 ( $\pm$ 42)	[80–210]

<sup>a</sup> number of samples; <sup>b</sup> statistically significant for T-test (equal variances) ( $p < 0.05$ ); <sup>c</sup> statistically significant for T-test (unequal variances) ( $p < 0.05$ ).

**Table 3.** Mean physicochemical parameter values and cumulative number of bathers in [A] and [B] during S1 and S2.

	Pool [A]		Pool [B]	
	S1	S2	S1	S2
Temperature ( $^{\circ}\text{C}$ )	27.8	28.5	27.9	27.6
pH	7.2	7.5	7.2	7.4
Free Chlorine (mg/L)	1.32	1.32	1.19	0.89
Total Chlorine (mg/L)	1.88	1.88	1.68	1.43
Cumulative number of bathers	239	862	122	530

#### 3.1. DBP Levels in Water

##### 3.1.1. Occurrence and Speciation

As shown in Table 2, TCM was the only THM found in water samples. No brominated THMs were detected. In the previous study by Simard [5], TCM was the most abundant THM (approximately 97% of all measured THMs). Among the nine HAAs usually measured (HAA9), mostly dichloroacetic acid (DCAA) and trichloroacetic acid (TCAA) were present in high concentrations. Only bromochloroacetic

acid (BCAA) and bromodichloroacetic acid (BDCAA) were also detected at very low concentrations. MCAM and TCAM were the main species of CAMs found in water while DCAM was measured in very low levels and only in approximately 25% of samples.

### 3.1.2. Spatial Variations

The levels of TCM and CAMs were significantly higher in [A] than in [B] ( $p < 0.0001$  in each case). Conversely, HAA9 concentrations (especially TCAA and DCAA) were significantly lower in [A] (Table 2). Figure 1 illustrates the DBP concentrations measured at the three different sites where the samples were collected (*i.e.*, deep, medium or shallow end of the pool).

**Figure 1.** Mean DBP water concentrations ( $\mu\text{g/L}$ ) in the swimming pool [A] during the four sampling periods of each day of the campaign S1 (Time = 0 min (9:00 am)–350 min (2:50 pm)). (a) TCM; (b) HAA9; (c) CAM.

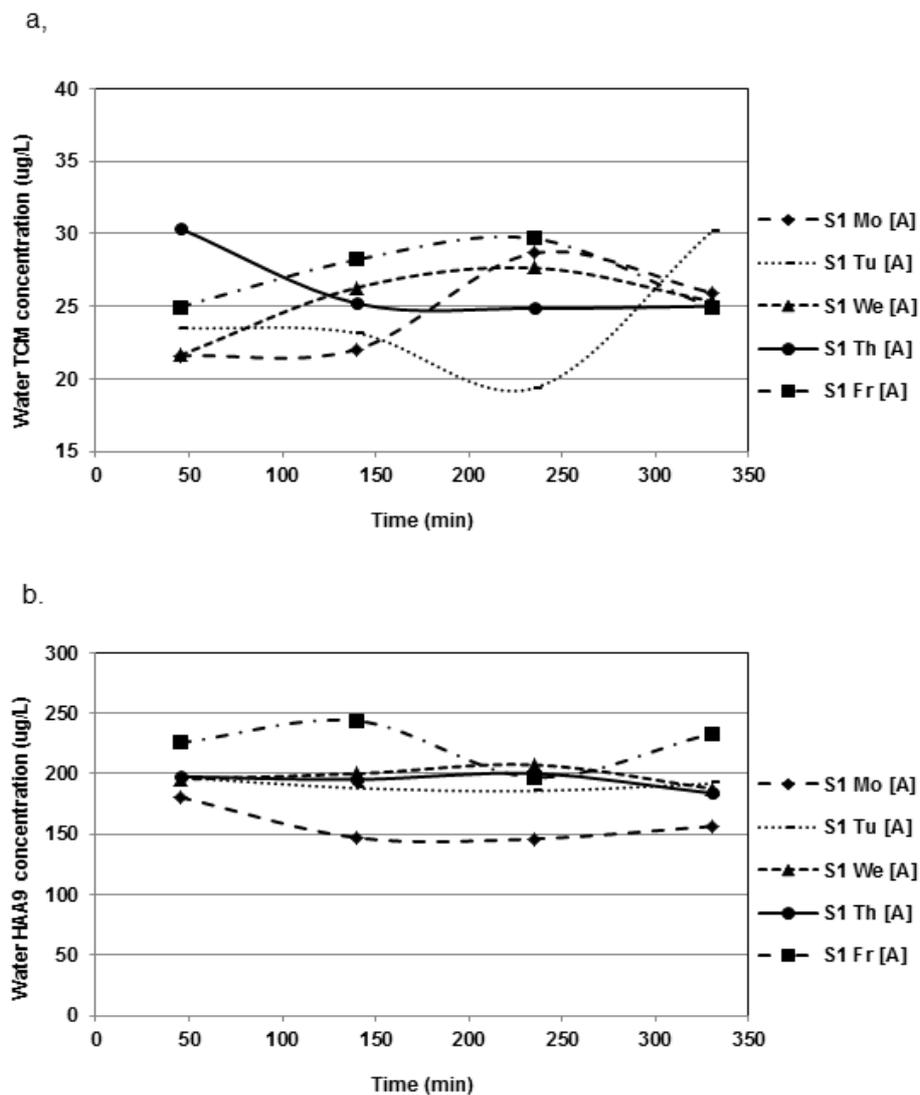
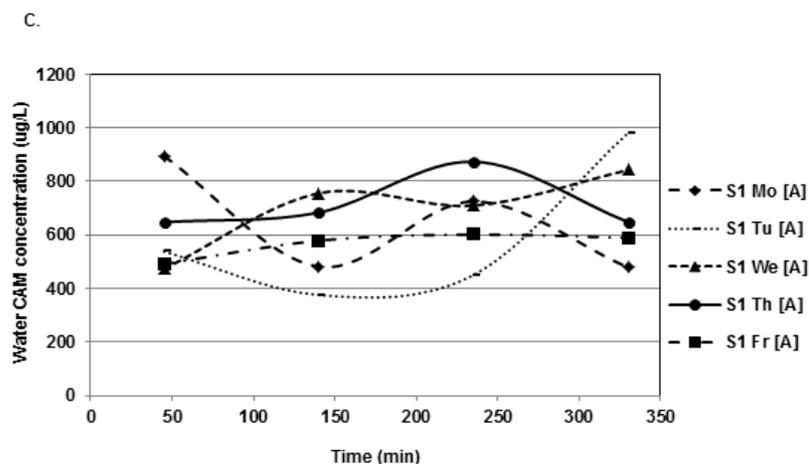


Figure 1. Cont.



No clear trend appears, as the levels measured were sometimes greater in the deep end but sometimes greater in the shallow end. The coefficients of variations of the concentrations measured between the various areas of the pool can reach up to around 40%. However, the average coefficients of variations were quite similar for both pools, ranging between 5% and 15% for the water contaminants (Table 4).

**Table 4.** Coefficients of variations (%) between the levels of DBPs measured at the various sampling places into the pool (for water contaminants) and around the pool (for air contaminants).

	<i>n</i> <sup>a</sup>	Pool [A] Mean (± SD)	[Min–Max]	<i>n</i> <sup>a</sup>	Pool [B] Mean (± SD)	[Min–Max]
TTHMs in water (µg/L)	39	14.1 (± 7.4)	[2.6–31.1]	37	14.1 (± 8.15)	[2.4–38.9]
TCM		14.1 (± 7.4)	[2.6–31.1]		14.1 (± 8.15)	[2.4–38.9]
HAA9 in water (µg/L)	40	9.5 (± 9.5)	[1.3–38.2]	40	7.2 (± 5.0)	[0.9–26.2]
DCAA		10.0 (± 9.9)	[1.5–35.3]		6.6 (± 4.5)	[1.5–23.0]
TCAA		10.8 (± 9.9)	[0.5–40.5]		9.3 (± 6.0)	[0.8–30.9]
BCAA		6.7 (± 6.2)	[0.2–24.8]		6.6 (± 10.3)	[0.4–52.5]
BDCAA		6.5 (± 13.0)	[0.4–82.6]		5.6 (± 5.8)	[0.2–21.6]
TTHMs in air (µg/m <sup>3</sup> )	38	22.1 (± 24.2)	[0.4–87.3]	37	12.9 (± 15.9)	[0.12–63.9]
TCM		22.1 (± 24.1)	[0.1–87.3]		12.8 (± 15.8)	[0.1–62.5]
DCBM		33.0 (± 32.0)	[0–128]		22.9 (± 30.2)	[0–133]

*n*<sup>a</sup> is the number of cases with all concentrations available at a same time in each sampling place.

Regarding TCM, the mixed ANOVA models did not indicate any effect relative to the sampling site for [B], but a slight effect depending on the day for [A] (*p* = 0.0176). In this last case, the samples collected in the shallow end of the pool tended to present slightly lower TCM levels than the samples collected at the deep end. Nevertheless, this effect was only significant at the beginning of the week (Monday and Tuesday). The ANOVA showed no effect of the sampling site on HAA9 levels.

3.1.3. Temporal Variations

TCM concentrations were slightly but significantly higher during S2 than during S1 in both [A] and [B]  $25.4 \pm 4.5 \mu\text{g/L}$  for S1 vs.  $31.7 \pm 6.04 \mu\text{g/L}$  for S2, in [A]; and  $20.7 \pm 3.95 \mu\text{g/L}$  for S1 vs.  $27.95 \pm 4.4 \mu\text{g/L}$  for S2, in [B]) ( $p < 0.001$ ). HAA9 and CAM levels were also higher during S2 in each pool. For HAA9, they were:  $193 \pm 26.3 \mu\text{g/L}$  for S1 vs.  $242.2 \pm 49.3 \mu\text{g/L}$  for S2, in [A]; and  $245.1 \pm 27.5 \mu\text{g/L}$  for S1 vs.  $270.5 \pm 43.8 \mu\text{g/L}$  for S2, in [B]. For CAMs, they were:  $583 \pm 123 \mu\text{g/L}$  for S1 vs.  $791 \pm 568 \mu\text{g/L}$  for S2, in [A]; and  $419 \pm 112 \mu\text{g/L}$  for S1 vs.  $509 \pm 121 \mu\text{g/L}$  for S2, in [B]. Figures 1 and 2 illustrate the within-day and day-to-day variations of DBP concentrations, respectively. Figure 1 does not disclose any evidence of a typical pattern regarding the within-day variations in DBP levels. Mean TCM concentrations did not fluctuate much (around  $10 \mu\text{g/L}$  approximately) between the five days of the week (Figure 2a). More variations were observed for HAAs, the levels of which increased constantly during the week (approximately 1.7-fold) (Figure 2b). Figure 3 presents day-to-day variations of the levels of CAMs in water. These variations were dependent primarily on TCAM levels, while the concentration of MCAM remained quite constant during the week.

Overall, the mixed ANOVA showed no effect relative to sampling day or time for CAMs or TCM. However, interestingly, the factor day was clearly significant in each pool for HAA9 ( $p < 0.0001$ ) (Figure 1b). For instance, in [A], the HAA9 levels were significantly lower on Monday, compared to the other days, and higher on Friday, compared to the first days of the week. This confirms the observation that HAA9 concentrations in pool water would tend to increase during the week. Indeed, the same trends are also observed in pool [B].

**Figure 2.** Mean DBP water concentrations ( $\mu\text{g/L}$ ) in the various zones of pool [A] and [B] during session S1 and S2. (a) TCM; (b) HAA9.

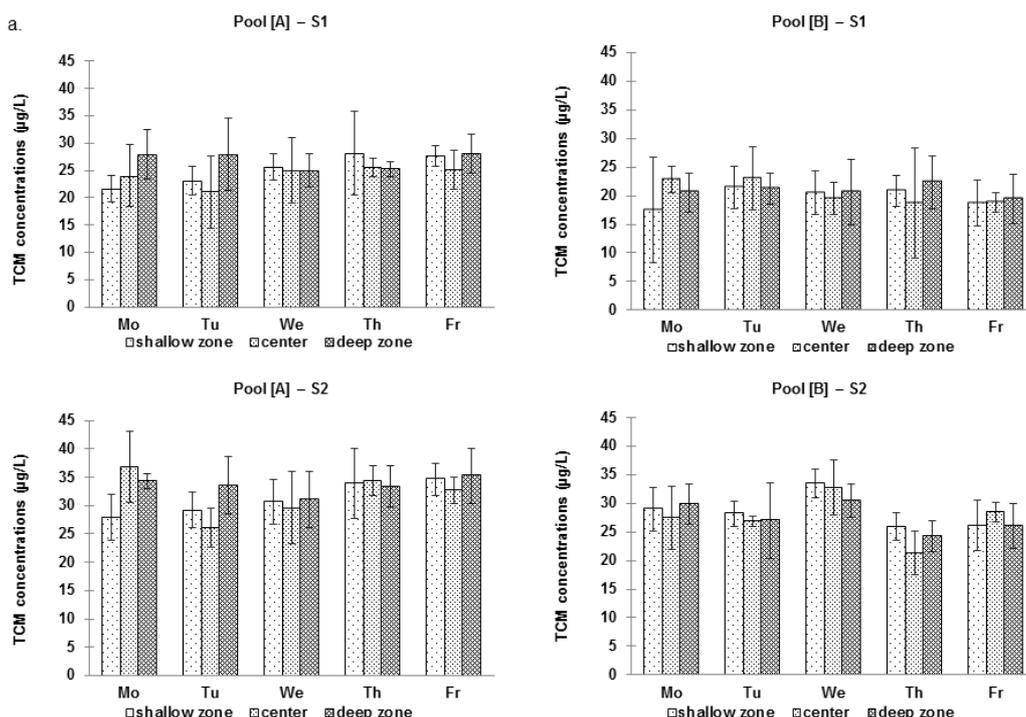


Figure 2. Cont.

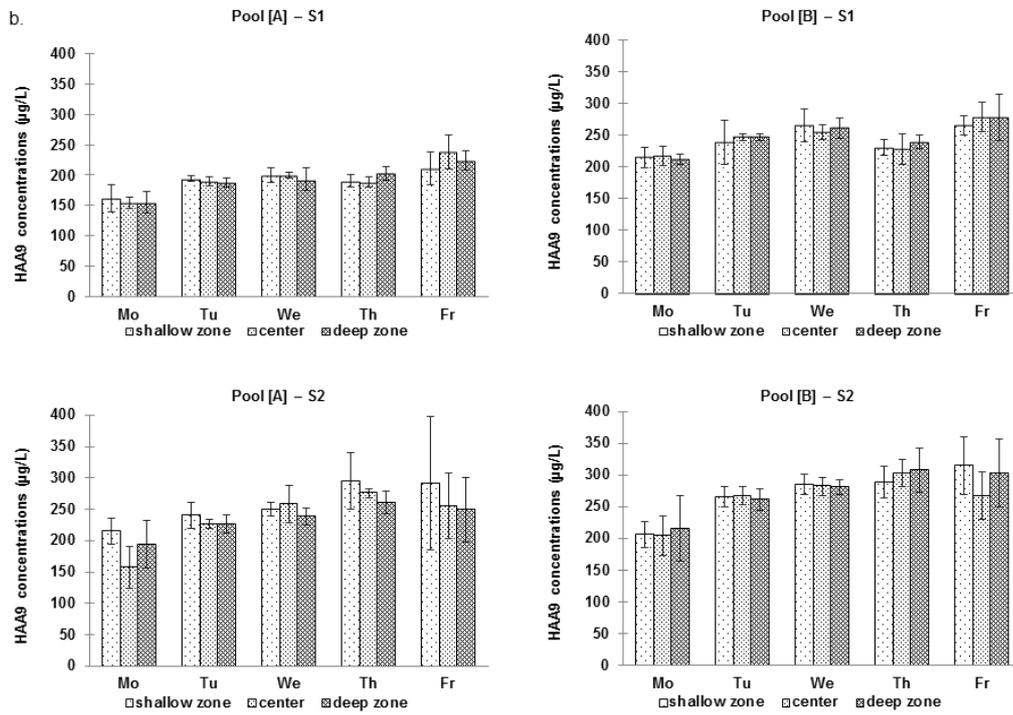
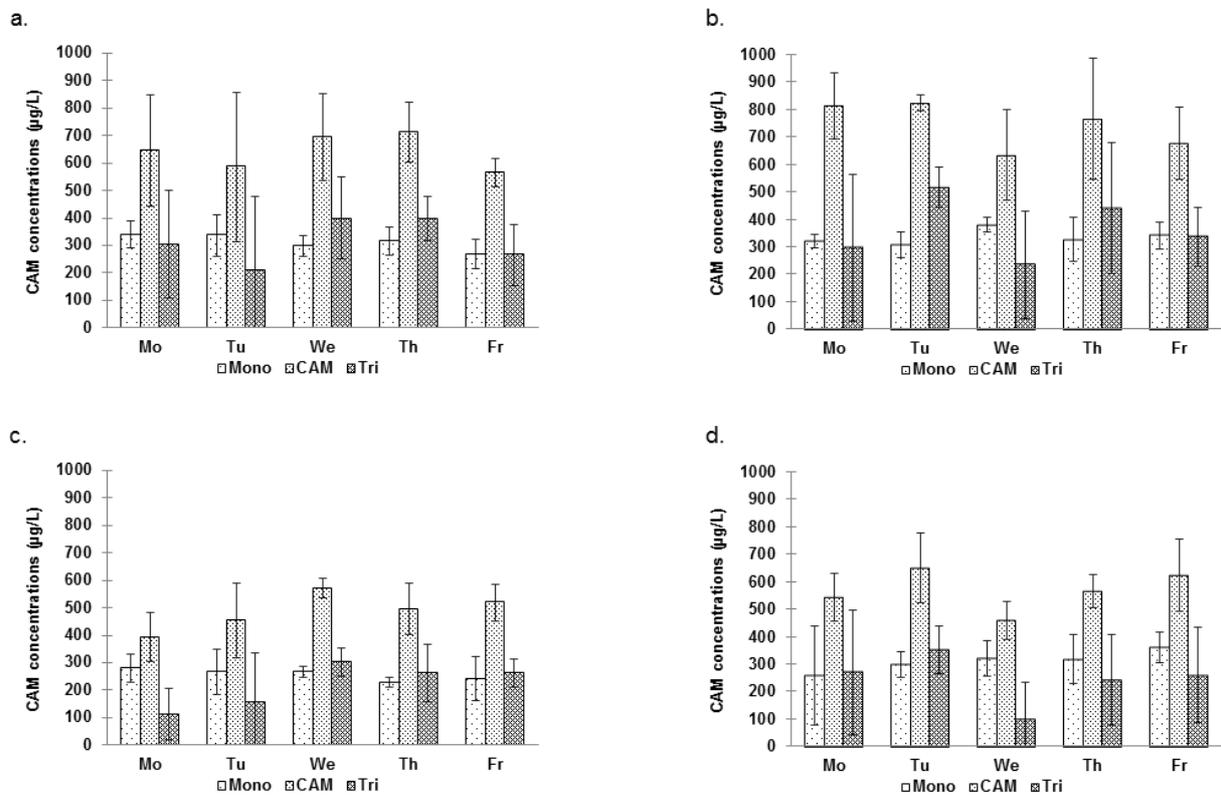


Figure 3. Mean daily concentrations of MCAM (Mono), TCAM (Tri) and Total CAM in water in [A] and [B] during S1 and S2. (a) [A]-S1; (b) [A]-S2; (c) [B]-S1; (d) [B]-S2.



3.2. DBP Levels in Air

3.2.1. THMs

Only TCM was systematically detected above the limit of quantification. DCBM was detected in some samples, but at much lower levels than TCM (Table 2). CDBM and TBM levels remained under their LOD. TCM showed the highest levels in both [A] and [B]. Therefore, the interpretation of spatial and temporal variations of THMs in the air was restricted to TCM and DCBM.

3.2.1.1. Spatial Variations

Significantly higher levels of THMs were measured in the air of pool [A] (Table 2). In addition, as shown in Table 4, the concentrations of THMs measured at 30 cm and 150 cm above the water surface in [A] were much more variable than in [B]. It may be due to usually higher swimmers' attendance in [A] (see Table 3) which cause more turbulence and therefore can contribute to higher volatilization of the THMs. Besides, Figure 4 clearly shows that the THM levels measured at 30 cm of pool [A]–session S2 are higher, compared to the levels measured at 150 cm. Indeed, the mixed ANOVA model for pool [A] indicates the factor sample site (height) is significant ( $p = 0.0176$ ). Such was not the case for pool [B].

**Figure 4.** Mean concentrations ( $\mu\text{g/L}$ ) of TTHMs in the pool air. (a) [A]–S1; (b) [A]–S2; (c) [B]–S1; (d) [B]–S2.

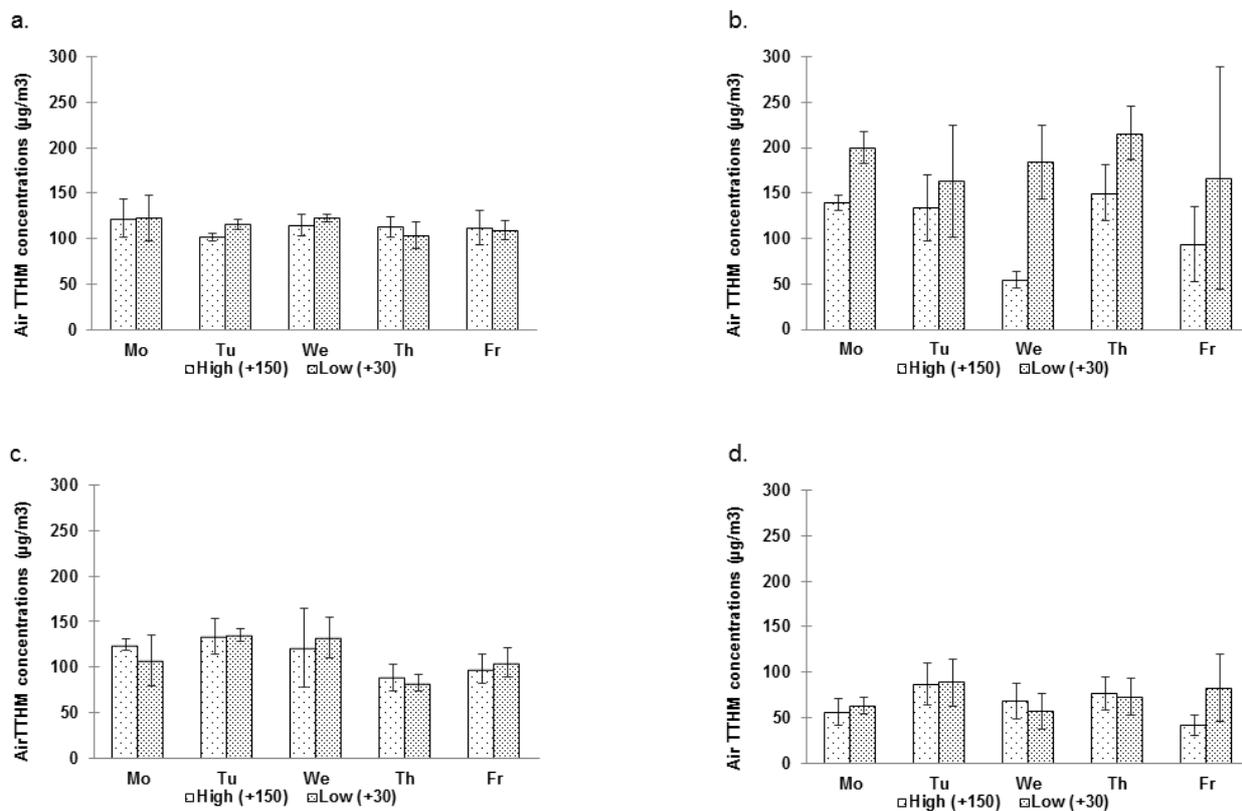


Table 5 summarizes the concentrations of TCM measured in various premises of each swimming pool building. Values measured in the premises of pool [B] were clearly higher, compared to pool [A].

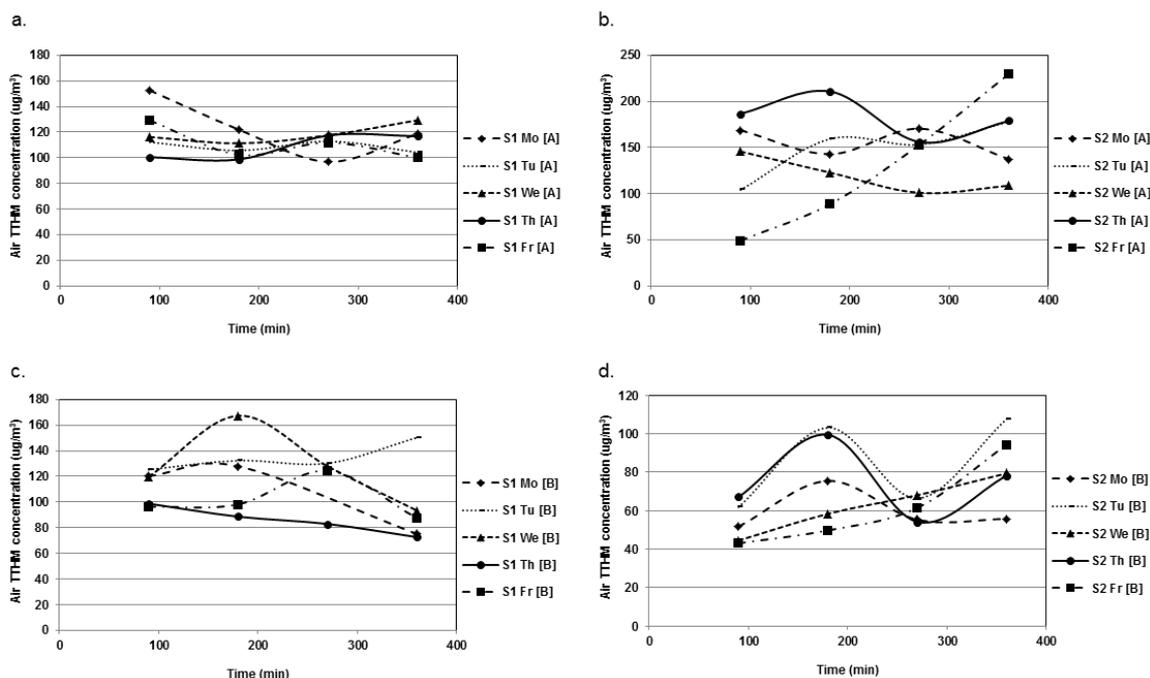
**Table 5.** TCM concentrations ( $\mu\text{g}/\text{m}^3$ ) in the ambient air of various rooms of [A] and [B] (all samples).

Room	Pool [A]			Pool [B]		
	<i>n</i>	Median	[Min–Max]	<i>n</i>	Median	[Min–Max]
Men changing room	20	2.3	[<LOD–4.5]	19	65.6	[43.8–115.5]
Women changing room	20	14.6	[4.6–28.2]	19	66.10	[47.5–111.5]
Lifeguards’ office	18	13.10	[<LOD–38.3]	20	59.3	[22.3–109.3]
Administrative office	-	-	-	11	27.1	[8.5–37.1]
Technical room	14	46.4	[4.7–99.2]	8	62.2	[43.8–117.8]
Bleachers	4	90.5	[81.4–117.9]	-	-	-

3.2.1.2. Temporal Variations

Overall, total THM (TTHM) air levels around the pool were significantly higher during S2 ( $147.6 \pm 67.4 \mu\text{g}/\text{m}^3$ ) than during S1 ( $113.4 \pm 14.6 \mu\text{g}/\text{m}^3$ ) in [A] ( $p = 0.003, p = 0.0316, p = 0.0031$  for TCM, DCBM, TTHM, respectively) as it was also the case for water levels. Conversely, the level of contamination was surprisingly, but significantly greater during S1 in [B] ( $112.5 \pm 26.7 \mu\text{g}/\text{m}^3$  vs.  $69.0 \pm 23.4 \mu\text{g}/\text{m}^3$ ) ( $p < 0.0001$  for each THM), as shown in Figure 4. However, no clear trends could be observed regarding day-to-day variations. Nevertheless, for [B] only, the mixed ANOVA showed an effect of the factor day ( $p = 0.0079$ ) due mainly to higher levels of contamination measured on Tuesday. The within-day variations of TTHM air levels were quite disparate as for water concentrations (Figure 5).

**Figure 5.** Mean TTHM concentrations ( $\mu\text{g}/\text{m}^3$ ) in the air of [A] and [B] during the 4 sampling periods of each day (Time = 0 min (9:00 am)–400 min (3:40 pm)). (a) [A]–S1; (b) [A]–S2; (c) [B]–S1 \*; (d) [B]–S2.



\* One data misses on Monday for [B]–S1.

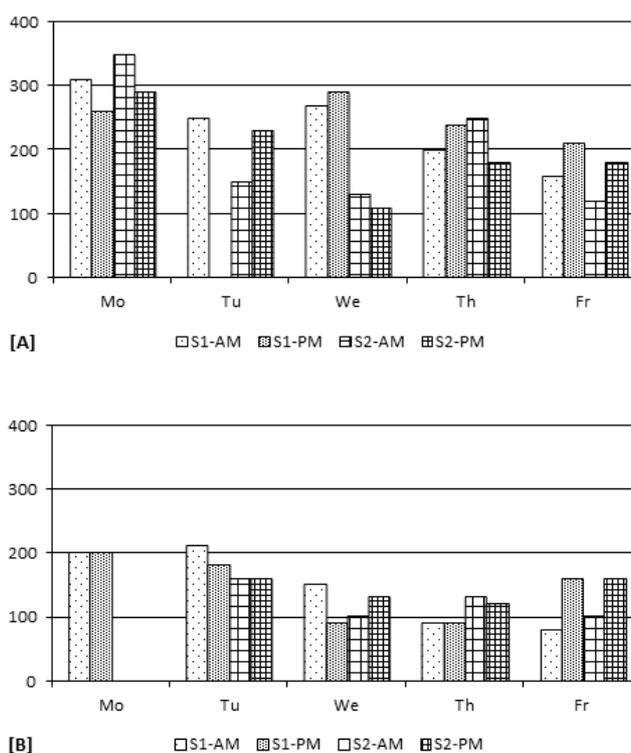
### 3.2.2. TCAMs

No typical patterns could be drawn. However, differences as high as 60  $\mu\text{g}/\text{m}^3$  could be measured between two samples taken at two different times on the same day.

#### 3.2.2.1. Spatial Variations

TCAM air levels around the pool were generally higher in [A] than in [B] (see Table 2) ( $p = 0.0005$ ). Only two samples (both in [A]) were above the suggested protective threshold level of 300  $\mu\text{g}/\text{m}^3$  proposed by Parrat [39] (see Figure 6). TCAM were below this threshold in the few samples collected in the lifeguard’s offices and even below the detection limit (50  $\mu\text{g}/\text{m}^3$ ) in the changing rooms.

**Figure 6.** Concentrations ( $\mu\text{g}/\text{m}^3$ ) of TCAM in the air of [A] and [B] in the morning (AM) and afternoon (PM) of each sampling day during S1 and S2 (Missing data were due to broken samples).



#### 3.2.2.2. Temporal Variations

In [A], the mean concentrations of TCAM were  $243.3 \pm 47.0 \mu\text{g}/\text{m}^3$  and  $199.0 \pm 79.4 \mu\text{g}/\text{m}^3$  during S1 and S2, respectively. In [B], they were  $145.0 \pm 52.8 \mu\text{g}/\text{m}^3$  and  $132.5 \pm 25.5 \mu\text{g}/\text{m}^3$ . In both cases, differences between S1 and S2 were not statistically significant ( $p = 0.1626$  and  $p = 0.5487$ , for [A] and [B], respectively). Figure 6 shows the day-to-day and within-day variability of TCAM concentrations during S1 and S2. It is not clear whether any trends exist regarding these variations. However, the ANOVA points to evidence of an effect relative to the factor day (with TCAM concentrations higher at the beginning of the week), but only for [B] ( $p = 0.0093$ ).

3.3. Relationships between the DBP Concentrations

Pearson coefficients between the various DBP concentrations in each pool and for each session were calculated. No consistent correlations were observed between the various types of DBPs in water, i.e., HAAs, THMs and CAMs.

The results show that TCAM in air was correlated either with THM levels measured at 30 cm above the water surface or at 150 cm. However, this result was not consistent according to the pool and the session, making it difficult to explain. The relationships between THM levels at 30 cm and THM levels at 150 cm were also inconsistent. However, in general, correlations with mean THM levels were always better with THM levels at 30 cm rather than 150 cm.

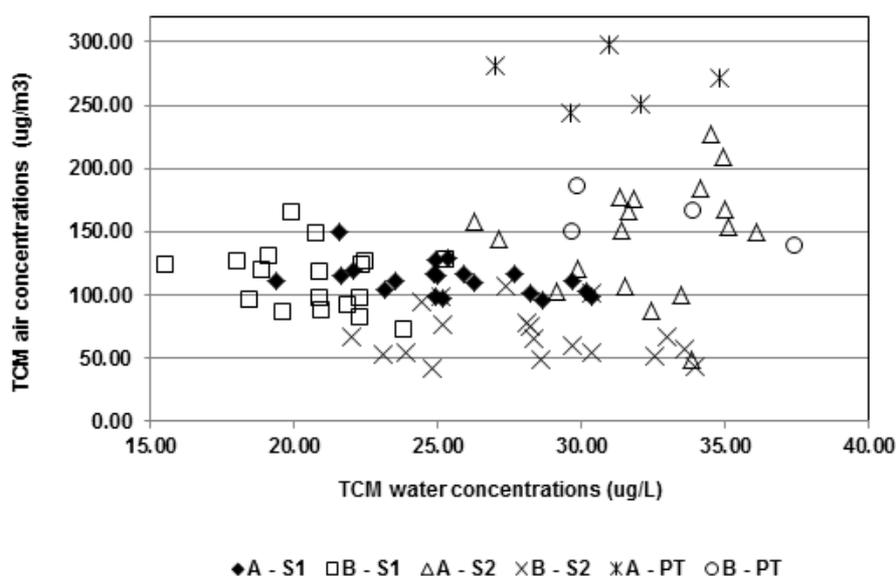
No clear relationship appeared between the DBPs in water and in the air (Table 6). Interestingly, HAA9 levels in water tended to be inversely correlated to air TCAM but the result was not statistically significant. Quite weak correlations were obtained between water CAMs and air THMs, but the relationships are inconsistent if each pool and session were considered independently.

**Table 6.** Pearson coefficient of correlations between DBP concentrations in water and DBP concentrations in air overall sessions and pools.

	TTHM	HAA9	CAM
Air TCAM	0.1821	-0.7139	0.3970
Air TCM	0.1657	-0.1819	0.3218 *
Air DCBM	0.1970	-0.2708 *	0.2021 *
Air TTHM	0.1664	-0.1834	0.3207 *

\*  $p < 0.05$ .

**Figure 7.** Concentrations ( $\mu\text{g}/\text{m}^3$ ) of TCM in the air of [A] and [B] vs. water concentrations ( $\mu\text{g}/\text{L}$ ).



3.4. Predictive Modeling of TCM Air Concentrations from Water Concentrations

We further investigated the relationship between TCM in air and TCM in water and the possibility of predicting air levels from water levels. Figure 7 shows the disparity of TCM air concentrations according to the TCM water concentrations when measured at the same time. However, it is interesting to note in this figure, the paired water and air concentrations measured in one pool during the same week tended to aggregate in the same area that may be regarded as representative of this pool during this week.

Figure 8 shows the predicted TCM air concentrations using the integrated volatilization model (VTM) and the level III fugacity model (FUG) described previously, vs. actual measurements. In all cases, the by-default setting did not allow very precise estimates. Nevertheless, the FUG modeling resulted, interestingly enough, in much more plausible estimates.

Figure 8. Predicted vs. measured TCM air concentrations ( $\mu\text{g}/\text{m}^3$ ) using VTM model and FUG model.

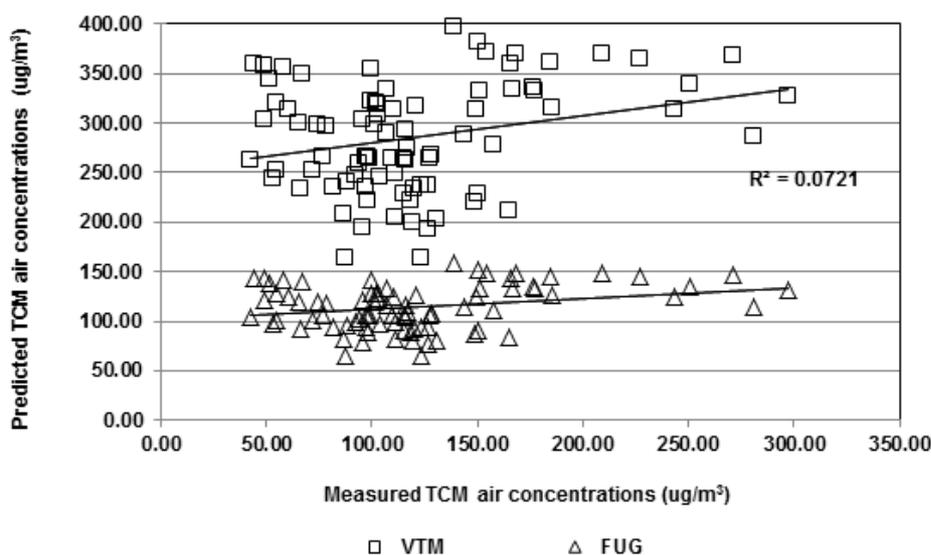


Table 7. Means of square residuals between measured and predicted TCM air concentrations.

Square Residuals	N	Mean	STD	Minimum	Maximum
VTMs <sup>a</sup>	84	31,240	22,670	25.43	99,610
–	32	34,290	26,320	5,214.80	99,610
+	52	29,360	20,140	25.43	79,690
VTMh <sup>b</sup>	84	17,170	16,840	1,688.42	87,340
–	32	10,740	7,120	1,688.42	33,380
+	52	21,120	19,720	2,277.11	87,340
FUG <sup>c</sup>	84	2,760	5,060	0.36	27,750
–	32	1,810	2,690	25.76	9,930
+	52	3,350	6,030	0.36	27,750

<sup>a</sup> volatilization model set by Haddad *et al.* [52], to predict air concentration into the shower room;

<sup>b</sup> volatilization model set by Haddad *et al.* [52], to predict air concentration into the rest of the house;

<sup>c</sup> equation from the fugacity model set by Dyck *et al.* [49]; –: considering periods with no bathers in the pool;

+: considering periods with bathers in the pool.

Indeed, Table 7, where the lower mean indicates the best predictor, suggests a greater reliability of FUG model to predict TCM air concentrations, rather than by-default set VTM model. Interestingly, the FUG estimates were clearly better for periods with no swimming pool attendance. In fact, the situation may be closer to an actual equilibrium state more suitable for such modeling, given that bathers' absence may cause less turbulence and DBP volatilization.

We adjusted the parameterization of VTM model to better fit its predictions on actual measurements. The Adjusted VTM model served to achieve a lower residual square means of 2,758.33 ( $\pm 4,816.76$ ), which indicates more precise predictions. We also adjusted an empirical model (EMP) on our generated database, resulting in the following formula:  $TCMa = 49.44 + 2.646 \times TCM$ , where TCMa is the TCM air concentration predicted in  $\mu\text{g}/\text{m}^3$  and TCM is the TCM water concentration in  $\mu\text{g}/\text{L}$ .

The FUG, adjusted VTM and EMP models were compared on the basis of data extracted from the literature and reported by Dyck *et al.* [49]. TCM air concentrations predicted using each model from reported TCM water concentrations were compared to reported TCM air measurements. Table 8 indicates FUG and EMP models are better predictive models than the adjusted VTM model.

**Table 8.** Comparison between adjusted VTM, EMP and FUG models for their abilities to predict TCM air concentrations from TCM water concentrations on the basis of data reported in the literature.

Square Residuals	N	Mean	STD	Minimum	Maximum
Adjusted VTM <sup>a</sup>	31	83,450	276,000	0.12	1,459,500
EMP <sup>b</sup>	31	36,220	145,880	10.44	830,500
FUG <sup>c</sup>	31	71,480	232,430	0.0017	1,218,600

<sup>a</sup> volatilization model set to fit with our dataset; <sup>b</sup> empirical model adjusted on our database;

<sup>c</sup> equation from the fugacity model set by Dyck *et al.* [49].

## 4. Discussion

We investigated the environmental occurrence of DBPs in two typical swimming pools with particular interest directed at the short-term and spatial variations of both water and air contaminants. Moreover, the database created served to examine the reliability of volatilization models for TCM. It provided interesting information to try to define best practices to assess DBP exposure in swimming pools for risk analysis or regulatory purposes.

### 4.1. Occurrence of DBPs and Health Risks

The high levels of HAAs in the water of the visited pools require particular attention. Indeed, these levels are consistent with those reported in a limited number of studies that documented the occurrence of these compounds in similarly chlorinated pools and also identified DCAA and TCAA as the most abundant HAAs [5,46,47]. While ingestion of swimming pool water is usually considered quite low [57], the impact of the consumption of even small quantities of water so highly loaded in HAAs should be further examined, especially compared to the levels to which people are exposed on a daily basis through drinking water consumption. Previously, Simard *et al.*, indicated HAA levels in swimming pools that could be up to 80 times the HAA levels in the distribution system [5]. The levels

of HAAs we measured in the present study remain lower but are approximately 3–4 times the norm of Québec for HAAs in drinking water (60 µg/L) [58]. Among THMs, only TCM was detected in pool water at quite low levels, ranging close to those reported in the literature, but usually above the German standard of 20 µg/L [2,4,5,41,44,59–69]. TCM was measured in concentrations usually reported in the ambient air of swimming pools [2,4,41,44,59–61,63,65–67,69]. DCBM was also detected but in much smaller concentrations in the air (up to 4.34 µg/m<sup>3</sup> but usually 100 times less than TCM levels), while no brominated THMs were detected in the water (less than 0.6, 1.0 and 0.8 µg/L for DCBM, CDBM and TBM respectively), perhaps due to differences in the sensitivity of analytical methods.

Air contamination was also assessed in various rooms surrounding the pool. To our knowledge, only two Italian studies by Fantuzzi *et al.*, have reported this type of information by measuring THM air concentrations in the reception area or in the engine room [66,67]. Interestingly, TCM levels in the various rooms of the swimming pool building was high, compared to typical household baseline contamination levels ranging between 1 and 10 µg/m<sup>3</sup> reported by Nuckols *et al.* [70]. They are comparable to contamination levels resulting from other household water use activities (*i.e.*, clothes washing (7–33 µg/m<sup>3</sup>), dishwashing (2–28 µg/m<sup>3</sup>), hand washing (19–85 µg/m<sup>3</sup>), bathing (21–98 µg/m<sup>3</sup>)) which the same author points out as potentially significant contributors to daily exposure to TCM. Moreover, swimming pool attendees or workers may spend a much longer time in these premises; therefore, they may even be more exposed to DBPs in these locations than during their usual household water use activities.

The levels of TCAM in ambient air did not comprise a range that seems to be particularly problematic for human health according to the suggested guideline of Parrat [39]. Likewise, it is important to note that for technical reasons we did not take samples during the periods when higher attendance might result in increasing DBP formation and then exposure. In addition, sampling was carried out in summer only; the results may have been greater in winter, especially for air contamination.

#### 4.2. Monitoring and Integrated Modeling for Exposure Assessment

Defining best practices for DBP exposure assessment in swimming pools must consider two main aspects: the first deals with the feasibility of using one particular DBP in a particular medium as a surrogate for the occurrence of other DBPs in other media; the second concerns the availability, ease of use and reliability of *in-situ* monitoring methods and/or predictive environmental modeling, since no standard sampling strategy for DBP exposure in swimming pool exists for attendees or workers.

To use a single DBP as an indicator of the levels of other DBPs would appear unfeasible, given the few correlations obtained in this study, which implies separate monitoring of each DBP. Indeed, contrary to Lee *et al.* [47] or Bessonneau *et al.* [29], we did not observe any consistent correlation between DBPs. Likewise, the analysis of spatial and temporal variations of both water and air contaminant levels pointed to a considerable potential for random disparities that make it inconceivable to predict their presence without using a minimal *in-situ* monitoring campaign.

Regarding the spatial variations, given the differences observed within and between the swimming pools, no location (at a fixed height for air or in a particular zone for water) could be identified as the most representative of the pool contamination for sampling; thus, neither should be regarded as suitable for one-shot monitoring. In the particular case of air TCAM, Parrat [39] points to the different conclusions drawn in the literature regarding possible decreases of contamination levels according to

the height of sample collection. The author assumes that numerous different conditions between pools (e.g., ventilation and attendance) may explain this. Our results, and particularly the differences observed between [A] and [B], support his assumption. In fact, collecting at least two samples of water and two samples of air is recommended, rather than taking some spot samples alone. Systematically collecting two samples of water in the deep and shallow ends of the pool and two samples of air at approximately 30 cm and 150 cm above water surface (*i.e.*, breathing zones of a swimmer and of a man standing at the edge of the pool) could be an interesting strategy for a better assessment of environmental exposure. Indeed, in this study, all air samples were collected in the middle on the poolside and we focused on the “vertical” variations without considering “horizontal” ones. This latter can occur actually as shown by Hsu *et al.* [48]. Nevertheless, in the current state of knowledge, we believe sampling air at the center of the pool may be the most representative sampling place to account for horizontal variations, but further investigations should address that.

Temporal variations can be important within a day, despite the fact that no typical pattern was drawn. Day-to-day variations in one swimming pool appeared to be quite limited in the course of the same week, apart from HAAs that tended to increase over the week in each pool and in each session; this may be due to a water change (back wash) in the pool. As for spatial variations, an appropriate monitoring should account for temporal variations by sampling at least twice a day. To take into account the variations of the number and activities of pool attendants, sampling just after opening and just before closing should be considered. The potential impact of such variations on DBP absorption and internal exposure assessment (and the error measurement associated to accounting for it or not) should be addressed in further investigations.

The issue of modeling the DBP volatilization is another challenging point to address for two reasons. On the one hand, analytical methods to measure TCAM and THMs in the air are not easy to carry out, despite the apparent necessity, given the sanitary impact associated with air TCAM and the allegedly high contribution of inhalation to THM exposure. On the other hand, as previously mentioned, numerous factors influence the formation and the volatilization of DBPs (e.g., number of swimmers, ventilation, and water turbulence) and make the development of alternative modeling tools particularly challenging. So far predicting TCM air concentrations from water levels does not appear to be very reliable, irrespective of the model used, which enforces the need of minimal sampling for both water and air. Between the various models tested, the FUG model proposed by Dyck *et al.* [49] and the EMP model we developed from the data collected in this study resulted in the more realistic predictions but their precision still needs to be greatly improved.

## 5. Conclusions

This study indicates that a minimal sampling strategy should be used for each DBP separately. The water-to-air models available for TCM require further improvement, but given the current state of knowledge where data for TCM air concentrations are not available, the use of the FUG model or the EMP model are alternatives. Overall, accurate DPB exposure assessment in swimming pool still remains very challenging, given the great number of variables (e.g., number of bathers or attendants, turbulence, organic precursors, ventilation) which may influence the amount of each compound and can produce the remarkable differences that this paper relieved within the same environment (water and air). Further research on DBP exposure should deal with the impact on bathers of such high levels

of HAAs in the swimming pool water, integrate both swimming pool and household exposure to DBPs in risk assessment and look at the impact of such exposure on swimming pool workers as they represent the potentially highest exposed population.

### Conflict of Interest

The authors declare no conflict of interest.

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### References

1. Zwiener, C.; Richardson, S.D.; de Marini, D.M.; Grummt, T.; Glauner, T.; Frimmel, F.H. Drowning in disinfection byproducts? Assessing swimming pool water. *Environ. Sci. Technol.* **2007**, *41*, 363–372.
2. Aggazzotti, G.; Fantuzzi, G.; Righi, E.; Predieri, G. Blood and breath analyses as biological indicators of exposure to trihalomethanes in indoor swimming pools. *Sci. Total Environ.* **1998**, *217*, 155–163.
3. Richardson, S.D.; Plewa, M.J.; Wagner, E.D.; Schoeny, R.; Demarini, D.M. Occurrence, genotoxicity, and carcinogenicity of regulated and emerging disinfection by-products in drinking water: A review and roadmap for research. *Mutat. Res.* **2007**, *636*, 178–242.
4. Richardson, S.D.; DeMarini, D.M.; Kogevinas, M.; Fernandez, P.; Marco, E.; Lourencetti, C.; Balleste, C.; Heederik, D.; Meliefste, K.; McKague, A.B.; *et al.* What's in the pool? A comprehensive identification of disinfection by-products and assessment of mutagenicity of chlorinated and brominated swimming pool water. *Environ. Health Perspect.* **2010**, *118*, 1523–1530.
5. Simard, S. *Occurrence des Sous-Produits de la Désinfection Dans L'eau des Piscines Publiques de la Ville de Québec*; Université Laval: Québec city, QC, Canada, 2009.
6. Villanueva, C.M.; Cantor, K.P.; Grimalt, J.O.; Malats, N.; Silverman, D.; Tardon, A.; Garcia-Closas, R.; Serra, C.; Carrato, A.; Castano-Vinyals, G.; *et al.* Bladder cancer and exposure to water disinfection by-products through ingestion, bathing, showering, and swimming in pools. *Am. J. Epidemiol.* **2007**, *165*, 148–156.
7. Tardiff, R.G.; Carson, M.L.; Ginevan, M.E. Updated weight of evidence for an association between adverse reproductive and developmental effects and exposure to disinfection by-products. *Regul. Toxicol. Pharmacol.* **2006**, *45*, 185–205.

8. Jacobs, J.H.; Spaan, S.; van Rooy, G.B.G.J.; Meliefste, C.; Zaat, V.A.C.; Royackers, J.M.; Heederik, D. Exposure to trichloramine and respiratory symptoms in indoor swimming pool workers. *Eur. Respir. J.* **2007**, *29*, 690–698.
9. Kaydos-Daniels, S.C.; Beach, M.J.; Shwe, T.; Magri, J.; Bixler, D. Health effects associated with indoor swimming pools: A suspected toxic chloramine exposure. *Publ. Health* **2007**, *122*, 195–200.
10. Kohlhammer, Y.; Heinrich, J. Chlorine, chlorination by-products and their allergic and respiratory health effects. *Curr. Respir. Med. Rev.* **2007**, *3*, 39–47.
11. Massin, N.; Bohadana, B.; Wild, P.; Héry, M.; Toamain, J.P.; Hubert, G. Respiratory symptoms and bronchial responsiveness in lifeguards exposed to nitrogen chloride in indoor swimming pools. *Occup. Environ. Med.* **1998**, *55*, 258–263.
12. Nemery, B.; Hoet, P.H.; Nowak, D. Indoor swimming pools, water chlorination and respiratory health. *Eur. Respir. J.* **2002**, *19*, 790–793.
13. Pommier de Santi, P.; Andreotti, D.; Lesaint, M.H. Rhinosinusite à la chloramine chez un maître-nageur. *Revue Française d'Allergologie et d'Immunologie Clinique* **2004**, *44*, 400–402.
14. Thickett, K.M.; McCoach, J.S.; Gerber, J.M.; Sadhra, S.; Burge, P.S. Occupational asthma caused by chloramines in indoor swimming-pool air. *Eur. Respir. J.* **2002**, *19*, 827–832.
15. Thoumelin, P.; Monin, E.; Armandet, D.; Julien, M.J.; Massart, B.; Vasseur, C.; Pillon, A.M.; Zilliox, M.; Balducci, F.; Bergeret, A. Troubles d'irritation respiratoire chez les travailleurs des piscines. *Doc. Pour Méd. Trav.* **2005**, *101*, 43–64.
16. Bernard, A.; Carbonnelle, S.; de Burbure, C.; Michel, O.; Nickmilder, M. Chlorinated pool attendance, atopy, and the risk of asthma during childhood. *Environ. Health Perspect.* **2006**, *114*, 1567–1573.
17. Bernard, A.; Carbonnelle, S.; Dumont, X.; Nickmilder, M. Infant swimming practice, pulmonary epithelium integrity, and the risk of allergic and respiratory diseases later in childhood. *Pediatrics* **2007**, *119*, 1095–1103.
18. Bernard, A.; Nickmilder, M. Respiratory health and baby swimming. *Arch. Dis. Child* **2006**, *91*, 620–621.
19. Bernard, A.; Nickmilder, M.; Voisin, C.; Sardella, A. Impact of chlorinated swimming pool attendance on the respiratory health of adolescents. *Pediatrics* **2009**, *124*, 1110–1118.
20. Font-Ribera, L.; Villanueva, C.M.; Nieuwenhuijsen, M.J.; Zock, J.P.; Kogevinas, M.; Henderson, J. Swimming pool attendance, asthma, allergies and lung function in the ALSPAC child cohort. *Am. J. Respir. Crit. Care Med.* **2010**, *83*, 582–588.
21. Weisel, C.P.; Richardson, S.D.; Nemery, B.; Aggazzotti, G.; Baraldi, E.; Blatchley, E.R., III; Blount, B.C.; Carlsen, K.H.; Eggleston, P.A.; Frimmel, F.H.; *et al.* Childhood asthma and environmental exposures at swimming pools: State of the science and research recommendations. *Environ. Health Perspect.* **2009**, *117*, 500–507.
22. Moulin, J.P. Bébés-nageurs: Effets des séances de piscines sur le développement du jeune enfant. *J. Pédiatr. Puéric.* **2007**, *20*, 25–28.
23. Gérardin, F.; Gerber, J.M.; Héry, M.; Quénis, B. Extraction de chloramines par contact gaz/liquide dans les eaux de piscines. *Cah. Notes Doc. Hyg. Séc. Trav.* **1999**, *177*, 21–29.

24. Gérardin, F.; Hecht, G.; Hubert-Pelle, G.; Subra, I. Traitement UV: Suivi de l'évolution des concentrations en chloroforme et en trichlorure d'azote dans les eaux de baignades d'un centre aquatique. *Cah. Notes Doc. Hyg. Séc. Trav.* **2005**, *201*, 19–30.
25. Kogevinas, M.; Villanueva, C.M.; Font-Ribera, L.; Liviac, D.; Bustamante, M.; Espinoza, F.; Nieuwenhuijsen, M.J.; Espinosa, A.; Fernandez, P.; DeMarini, D.M.; *et al.* Genotoxic effects in swimmers exposed to disinfection by-products in indoor swimming pools. *Environ. Health Perspect.* **2010**, *118*, 1531–1537.
26. Liviac, D.; Wagner, E.D.; Mitch, W.A.; Altonji, M.J.; Plewa, M.J. Genotoxicity of water concentrates from recreational pools after various disinfection methods. *Environ. Sci. Technol.* **2010**, *44*, 3527–3532.
27. Plewa, M.J.; Simmons, J.E.; Richardson, S.D.; Wagner, E.D. Mammalian cell cytotoxicity and genotoxicity of the haloacetic acids, a major class of drinking water disinfection by-products. *Environ. Mol. Mutagen.* **2010**, *51*, 871–878.
28. Risques Sanitaires Liés Aux Piscines. Available online: [http://www.afsset.fr/upload/bibliotheque/413996977197053340489872262883/10\\_06\\_piscines\\_reglementees\\_vdef\\_2010.pdf](http://www.afsset.fr/upload/bibliotheque/413996977197053340489872262883/10_06_piscines_reglementees_vdef_2010.pdf) (accessed on 17 July 2012).
29. Bessonneau, V.; Derbez, M.; Clement, M.; Thomas, O. Determinants of chlorination by-products in indoor swimming pools. *Int. J. Hyg. Environ. Health* **2011**, *215*, 76–85.
30. Caro, J.; Gallego, M. Alveolar air and urine analyses as biomarkers of exposure to trihalomethanes in an indoor swimming pool. *Environ. Sci. Technol.* **2008**, *42*, 5002–5007.
31. Font-Ribera, L.; Kogevinas, M.; Zock, J.P.; Gomez, F.P.; Barreiro, E.; Nieuwenhuijsen, M.J.; Fernandez, P.; Lourencetti, C.; Perez-Olabarria, M.; Bustamante, M.; *et al.* Short-term changes in respiratory biomarkers after swimming in a chlorinated pool. *Environ. Health Perspect.* **2010**, *118*, 1538–1544.
32. Font-Ribera, L.; Kogevinas, M.; Zock, J.P.; Nieuwenhuijsen, M.J.; Heederik, D.; Villanueva, C.M. Swimming pool attendance and risk of asthma and allergic symptoms in children. *Eur. Respir. J.* **2009**, *34*, 1304–1310.
33. Kanan, A.; Karanfil, T. Formation of disinfection by-products in indoor swimming pool water: The contribution from filling water natural organic matter and swimmer body fluids. *Water Res.* **2011**, *45*, 926–932.
34. Lee, J.; Ha, K.T.; Zoh, K.D. Characteristics of trihalomethane (THM) production and associated health risk assessment in swimming pool waters treated with different disinfection methods. *Sci. Total Environ.* **2009**, *407*, 1990–1997.
35. Lourencetti, C.; Ballester, C.; Fernandez, P.; Marco, E.; Prado, C.; Periago, J.F.; Grimalt, J.O. New method for determination of trihalomethanes in exhaled breath: Applications to swimming pool and bath environments. *Anal. Chim. Acta* **2010**, *662*, 23–30.
36. Panyakapo, M.; Soontornchai, S.; Paopuree, P. Cancer risk assessment from exposure to trihalomethanes in tap water and swimming pool water. *J. Environ. Sci.(China)* **2008**, *20*, 372–378.
37. Lévesque, B.; Duchesne, J.F.; Gingras, S.; Lavoie, R.; Prud'Homme, D.; Bernard, E.; Boulet, L.P.; Ernst, P. The determinants of prevalence of health complaints among young competitive swimmers. *Int. Arch. Occup. Environ. Health* **2006**, *80*, 32–39.

38. Héry, M.; Hecht, G.; Gerber, J.M.; Gendre, J.C.; Hubert, G.; Rebuffaud, J. Exposure to chloramines in the atmosphere of indoor swimming pools. *Ann. Occup. Hyg.* **1995**, *39*, 427–439.
39. Parrat, J. *Évaluation de L'exposition à la Trichloramine Atmosphérique des Maîtres Nageurs, Employés et Utilisateurs Publics des Piscines Couvertes des Cantons de Fribourg, Neuchâtel et du Jura*; Laboratoire intercantonal de santé au travail—LIST: Peseux, Switzerland, 2008; p. 76.
40. Bonvallot, N.; Glorennec, P.; Zmirou, D. Derivation of a toxicity reference value for nitrogen trichloride as a disinfection by-product. *Regul. Toxicol. Pharmacol.* **2010**, *56*, 357–364.
41. Lévesque, B.; Ayotte, P.; Tardif, R.; Charest-Tardif, G.; Dewailly, E.; Prud'Homme, D.; Gingras, G.; Allaire, S.; Lavoie, R. Evaluation of the health risk associated with exposure to chloroform in indoor swimming pools. *J. Toxicol. Environ. Health* **2000**, *61*, 225–243.
42. Hamel, H. *Etude de L'évolution du Trichlorure D'azote et des Trihalométhanes Dans l'eau et L'air des Piscines Chlorées—Exploration des Voies de Réduction de Cette Contamination*; Université de Rennes I: Rennes, France, 2007.
43. Li, J.; Blatchley, E.R., III. Volatile disinfection byproduct formation resulting from chlorination of organic-nitrogen precursors in swimming pools. *Environ. Sci. Technol.* **2007**, *41*, 6732–6739.
44. Sa, C.S.; Boaventura, R.A.; Pereira, I.B. Analysis of trihalomethanes in water and air from indoor swimming pools using HS-SPME/GC/ECD. *J. Environ. Sci. Health Tox. Hazard. Subst. Environ. Eng.* **2011**, *46*, 355–363.
45. Weaver, W.A.; Li, J.; Wen, Y.; Johnston, J.; Blatchley, M.R.; Blatchley, E.R., III. Volatile disinfection by-product analysis from chlorinated indoor swimming pools. *Water Res.* **2009**, *43*, 3308–3318.
46. Cardador, M.J.; Gallego, M. Haloacetic acids in swimming pools: Swimmer and worker exposure. *Environ. Sci. Technol.* **2011**, *45*, 5783–5790.
47. Lee, J.; Jun, M.J.; Lee, M.H.; Eom, S.W.; Zoh, K.D. Production of various disinfection byproducts in indoor swimming pool waters treated with different disinfection methods. *Int. J. Hyg. Environ. Health* **2010**, *213*, 465–474.
48. Hsu, H.T.; Chen, M.J.; Lin, C.H.; Chou, W.S.; Chen, J.H. Chloroform in indoor swimming-pool air: Monitoring and modeling coupled with the effects of environmental conditions and occupant activities. *Water Res.* **2009**, *43*, 3693–3704.
49. Dyck, R.; Sadiq, R.; Rodriguez, M.J.; Simard, S.; Tardif, R. Trihalomethane exposures in indoor swimming pools: A level III fugacity model. *Water Res.* **2011**, *45*, 5084–5098.
50. US EPA. *Method 552.2. Determination of Haloacetic Acids in Drinking Water by Liquid-Liquid Extraction and Gas Chromatography with Electroncapture Detection*; US EPA: Cincinnati, OH, USA, 1995.
51. American Public Health Association; American Water Works Association; Water Environment Federation. *Standard Methods for the Examination of Water and Wastewater*, 20th ed.; Clesceri, L.S., Greenberg, A.E., Eaton, A.D., Eds.; American Public Health Association: Washington, DC, USA, 1999.
52. Haddad, S.; Charest-Tardif, G.C.; Tardif, R. Development of physiologically based toxicokinetic models for improving the human indoor exposure assessment to water contaminants: Trichloroethylene and trihalomethanes. *J. Toxicol. Environ. Health* **2006**, *69*, 2095–2136.

53. McKone, T.E. Human exposure to volatile organic compounds in household tap water: The indoor inhalation pathway. *Environ. Sci. Technol.* **1987**, *21*, 1194–1201.
54. McKone, T.E.; Knezovich, J.P. The transfer of trichloroethylene (TCE) from a shower to indoor air: Experimental measurements and their implications. *J. Air Waste Manag. Assoc.* **1991**, *41*, 832–837.
55. Mackay, D. *Multimedia Environmental Models: The Fugacity Approach*, 2nd ed.; Lewis Publishers: Boca Raton, FL, USA, 2001.
56. SAS Institute Inc. *SAS OnlineDoc*® 9.2.; SAS Institute: Cary, CA, USA, 2009.
57. Dufour, A.P.; Evans, O.; Behymer, T.D.; Cantu, R. Water ingestion during swimming activities in a pool: A pilot study. *J. Water Health* **2006**, *4*, 425–430.
58. Ministry of Sustainable Development, Environment and Parks. *Règlement Modifiant le Règlement sur la Qualité de l'eau Potable*; Government of Quebec: Quebec, QC, Canada, 2012.
59. Aggazzotti, G.; Fantuzzi, G.; Righi, E.; Predieri, G. Environmental and biological monitoring of chloroform in indoor swimming pools. *J. Chromatogr.* **1995**, *710*, 181–190.
60. Aggazzotti, G.; Fantuzzi, G.; Righi, E.; Tartoni, P.; Cassinadri, T.; Predieri, G. Chloroform in alveolar air of individuals attending indoor swimming pools. *Arch. Environ. Health* **1993**, *48*, 250–254.
61. Aggazzotti, G.; Fantuzzi, G.; Tartoni, P.L.; Predieri, G. Plasma chloroform concentrations in swimmers using indoor swimming pools. *Arch. Environ. Health* **1990**, *45*, 175–179.
62. Aiking, H.; van Acker, M.B.; Scholten, R.J.; Feenstra, J.F.; Valkenburg, H.A. Swimming pool chlorination: A health hazard? *Toxicol. Lett.* **1994**, *72*, 375–380.
63. Cammann, K.; Hubner, K. Trihalomethane concentrations in swimmers' and bath attendants' blood and urine after swimming or working in indoor swimming pools. *Arch. Environ. Health* **1995**, *50*, 61–65.
64. Chu, H.; Nieuwenhuijsen, M.J. Distribution and determinants of trihalomethane concentrations in indoor swimming pools. *Occup. Environ. Med.* **2002**, *59*, 243–247.
65. Erdinger, L.; Kuhn, K.P.; Kirsch, F.; Feldhues, R.; Frobel, T.; Nohynek, B.; Gabrio, T. Pathways of trihalomethane uptake in swimming pools. *Int. J. Hyg. Environ. Health* **2004**, *207*, 571–575.
66. Fantuzzi, G.; Righi, E.; Predieri, G.; Ceppelli, G.; Gobba, F.; Aggazzotti, G. Occupational exposure to trihalomethanes in indoor swimming pools. *Sci. Total Environ.* **2001**, *264*, 257–265.
67. Fantuzzi, G.; Righi, E.; Predieri, G.; Giacobazzi, P.; Mastroianni, K.; Aggazzotti, G. Prevalence of ocular, respiratory and cutaneous symptoms in indoor swimming pool workers and exposure to disinfection by-products (DBPs). *Int. J. Environ. Res. Public Health* **2010**, *7*, 1379–1391.
68. Lévesque, B.; Ayotte, P.; LeBlanc, A.; Dewailly, E.; Prud'Homme, D.; Lavoie, R.; Allaire, S.; Levallois, P. Evaluation of dermal and respiratory chloroform exposure in humans. *Environ. Health Perspect.* **1994**, *102*, 1082–1087.
69. Lindstrom, A.B.; Pleil, J.D.; Berkoff, D.C. Alveolar breath sampling and analysis to assess trihalomethane exposures during competitive swimming training. *Environ. Health Perspect.* **1997**, *105*, 636–642.

70. Nuckols, J.R.; Ashley, D.L.; Lyu, C.; Gordon, S.M.; Hinckley, A.F.; Singer, P. Influence of tap water quality and household water use activities on indoor air and internal dose levels of trihalomethanes. *Environ. Health Perspect.* **2005**, *113*, 863–870.

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