ELSEVIER

Journal of Chromatography A, 710 (1995) 181–190

JOURNAL OF CHROMATOGRAPHY A

Environmental and biological monitoring of chloroform in indoor swimming pools

Gabriella Aggazzotti*, Guglielmina Fantuzzi, Elena Righi, Guerrino Predieri

Cattedra di Metodologia Epidemiologica ed Igiene, Facoltà di Medicina e Chirurgia, Dipartimento di Scienze Biomediche, Sezione di Igiene e Microbiologia, Università degli Studi di Modena, Via G. Campi 287, 41100 Modena, Italy

Abstract

The presence of chloroform as the result of disinfection with sodium hypochlorite was demonstrated in the water and ambient air of indoor swimming pools. Environmental monitoring was performed in 12 indoor swimming pools in northern Italy and the level of human exposure was assessed. Biological monitoring performed by gas chromatography on human plasma and alveolar air samples evidenced that the uptake of chloroform in swimmers varies according to the intensity of the physical activity and age. The elimination of chloroform in alveolar air in one subject showed a very short half-life (from 20 to 27 min) and a complete clearance within 10 h after the end of exposure.

1. Introduction

Surveys from all over the world have reported the presence of trihalomethanes in swimming pools as by-products of treatment with chlorine and its derivatives [1–7]. The most common of these trihalomethanes is chloroform, which is a volatile substance released at the surface of the water and which can be inhaled by swimmers.

Since chloroform is classified in the 2B group by the International Agency for Research on Cancer (IARC), it is worthwhile studying the conditions under which exposure is likely, as well as the amount of chloroform in the environment, in order to assess the risk, if any, for exposed subjects [8,9].

The problem was approached in three ways:

–analysis of chloroform in the water and in the ambient air above the pools,

–biological monitoring of chloroform exposure by means of blood samples and exhaled air (alveolar air) samples,

–study of the kinetics of chloroform elimination from the lungs.

2. Environmental monitoring in indoor swimming pools

Sodium hypochlorite and, more recently, sodium dichloroisocyanurate are the substances most commonly used for the disinfection of swimming pool water. The concentration of active chlorine, and hence that of chloroform (the main by-product of chlorination), is usually higher than in treated drinking water.

Since 1980 we have been monitoring levels of

* Corresponding author.

0021-9673/95/$09.50
SSDI 0021-9673(95)00432-7

chloroform in indoor swimming pools in a region of northern Italy (Emilia Romagna). Concentrations in water and ambient air samples have been measured in 12 indoor swimming pools, several sampling sessions sometimes being performed in the same pool over a period of time. Eighty-eight data sets have been collected.

During each sampling session we took into account variables which could be associated with the release of chloroform from water to ambient air, namely, water and air temperature, pH, and free and combined chlorine residual.

The number of swimmers present in the pool was also noted, as turbulence caused by their movement could influence the release of chloroform into the air.

During each session, three samples of water were collected at a depth of 20 cm at three different positions near the edge of the pool; the mean value was considered as representative of the chloroform concentration in the water. Water samples were collected in screw-capped glass vials (40 cm^3) with silicone-faced septa. The vials were treated just before sampling with 5 mg of sodium thiosulfate to quench residual chlorine reactions.

2.1. Determination of chloroform in water

Samples were analyzed by a head-space gas chromatographic (GC) technique. A Dani HSS 3950 autosampler and a Varian 3400 gas chromatograph equipped with a ^{63}Ni electron-capture detector and a Vocol capillary column [30 m × 0.53 mm I.D., film thickness 3.0 μm (Supelco)] were used. Parameters were: carrier gas (He) flow-rate 8 ml/min; make-up gas, 20 ml/min; initial column temperature, 50°C (1 min); rate, 6°C/min; final temperature, 100°C (7 min); injector temperature, 150°C; detection temperature, 260°C; detector, electron-capture detection (ECD); range, 10; attenuation, 32. Calibration was performed by the external standard method, which is preferred by most workers when studying the presence of chloroform in waters by head-space GS [10,11]. Precision, calculated from five duplicate determinations on five different days, was 2.8% (coefficient of variation, C.V.). The limit of detection was calculated from 30 different series of chloroform determinations of blank value (vials with chloroform-free water). The mean blank concentration was 0.35 μg/l with a standard deviation of 0.04 μg/l. Based on 2.5 times the standard deviation, the limit of detection was 0.1 μg/l.

Chloroform was also measured in the ambient air: during the first session, the sampling of environmental air was performed by collecting spot samples every 15 min at different levels at the edge of the pool, using the same vials as before. As only slight variations were found between chloroform levels at the surface of the pool and those 150 cm above it, we decided to take samples only at 150 cm in order to avoid contamination by splashing. The mean values were considered to be representative of the environmental chloroform concentrations.

Environmental air samples used as controls were collected inside the Department of Hygiene of the University of Modena.

2.2. Determination of chloroform in environmental air

Samples were injected directly into the GC using a gas-tight syringe (Hamilton). Calibration was performed by the external standard method. Precision, calculated as before, was 3.5% as C.V.

The limit of detection was calculated from 30 different series of chloroform determinations of blank value (vials with environmental air). The mean blank concentration was 5.0 $\mu g/m^3$, with a standard deviation of 0.4 $\mu g/m^3$. Based on 2.5 times the standard deviation, the limit of detection was 1 $\mu g/m^3$. Quantitative analysis was performed by a Merck-Hitachi chromato-integrator D2000.

The identity of the chloroform was confirmed by GC–mass spectrometry (GC–MS) as follows: some ambient air samples were collected inside the swimming pools in Carbotrap 300 multibed thermal desorption tubes (Supelco) and injected using a thermal desorption cold-trap injector (Chromopack) into a GC–MS system (GC: HP 5990, MS: 5989A, Hewlett-Packard); identification was based on retention times measured on a

Table 1
Chloroform in water and environmental air samples collected in indoor swimming pools in northern Italy

Swimming pool	No. sampling sessions	Chloroform in water (μg/l)		Chloroform in environmental air (μg/m^3)	
		Arithmetic mean	Range	Arithmetic mean	Range
1	26	88.08	14–167	216.23	16–533
2	26	30.77	9–94	189	60–682
3	9	56.33	31–85	97.56	41–223
4	8	97.38	23–179	338.63	135–656
5	2	36.50	24–49	96	53–139
6	3	77.33	62–96	279.33	104–378
7	2	19.50	13–26	48	44–52
8	2	114.50	84–145	459.50	66–853
9	2	47.50	45–50	302.50	85–520
10	1	23[a]		80[a]	
11	1	56[a]		131[a]	
12	6	99.33	84–113	421.50	67–675

[a] One spot sample.

total ion-current chromatogram and on mass chromatograms of the molecular ion and of the most significant fragments of chloroform (m/z 47–50, 82–87, and 117–124).

The mean levels of chloroform in water and air in 12 indoor swimming pools are reported in Table 1. Chloroform in water ranges from 9 to 179 μg/l, which is in accordance with previous studies [4,12]. Levels in environmental air vary widely, from 16 to 853 μg/m^3.

Correlations among the variables taken into account during each sampling session are reported in Table 2.

The chloroform concentration in water correlates with that in air; the number of swimmers appears to be negatively correlated with the chloroform level in the water and positively with the concentration in air as a consequence of the turbulence induced in the water. Moreover, chloroform in water appears to be significantly correlated with the free and combined chlorine residuals, as all these variables are associated with the process of chlorination. No correlation appears between chloroform in water and the temperature of the water, and between chloroform in air and the temperature of the ambient

Table 2
Spearman's Rank correlation coefficients

	r	p
Chloroform in water (μg/l)/chloroform in air (μg/m^3)	0.2785	0.009
No. of swimmers/chloroform in water (μg/l)	−0.3451	0.001
No. of swimmers/chloroform in air (μg/m^3)	0.3555	0.001
Chloroform in water (μg/l)/free chlorine residual (mg/l Cl)	0.3255	0.002
Chloroform in water (μg/l)/combined chlorine residual (mg/l Cl)	0.2905	0.009
Chloroform in water (μg/l)/water temperature (°C)	0.1931	ns
Chloroform in water (μg/l)/water pH (unit)	0.2135	0.046
Chloroform in air (μg/m^3)/air temperature (°C)	0.0101	ns

Table 3
Multiple regression analysis. Chloroform in environmental air: dependent variable

Independent variable	Variables in the equation				
	B	SE *B*	Beta	*T*	Sig *T*
No. of swimmers	0.010928	0.002071	0.519178	5.277	<0.001
Chloroform in water	0.003532	8.3556×10^{-4}	0.415942	4.227	<0.001
Constant	1.694997	0.096278		17.605	<0.001

air, while a weak correlation exists between the pH and the amount of chloroform in water. It must be noted that in our data sets the values of water and air temperature and pH were within very narrow ranges.

Multiple regression analysis was performed by taking the level of chloroform in ambient air as the dependent variable, while chloroform in water and the number of swimmers were considered independent variables. Both independent variables influence the dependent one (after log transformation): the model accounts for 26.8% of the observed variance (R^2 adjusted: 0.268). The variables in the equation are reported in Table 3.

3. Biological monitoring in indoor swimming pools

People, particularly swimmers, visiting indoor swimming pools are exposed to chloroform by three routes: (1) inhalation of chloroform volatilized into indoor air from chlorinated water; (2) ingestion of chloroform from the water (particularly with children); (3) dermal contact with the water.

As indoor swimming pool visitors are exposed to chloroform for a precise period of time at a known environmental concentration, they form an experimental group from which more detailed information about chloroform exposure can be obtained.

The possibility of exposure via drinking water was excluded, as Modena is supplied with drinking water treated with chlorine dioxide (ClO_2) and is free from chloroform and other halogenated organic compounds. Nevertheless, we analyzed drinking water samples before, during, and after this study to confirm the absence of chloroform.

Chloroform is readily absorbed into the body mainly through the lungs and intestinal mucosa. However, when subjects take a bath or swim in chlorinated water, skin absorption may also be significant. In fact, the skin, the largest organ of the body, acts as a lipid sink for lipid-soluble contaminants [13].

Few data are available on the pharmacokinetics of absorption and excretion of chloroform in humans, particularly at the low rates of exposure normally found in ambient air and drinking water. Some studies show that absorption in man is rapid and complete, occurring by first-order passive absorption processes. Pulmonary uptake and elimination also occur by first-order diffusion processes, with three distinct components, with rate constants corresponding to tissue loading or desaturation of at least three major body compartments (vessel-rich tissues, lean body mass, adipose tissue).

Elimination of chloroform from the body occurs by two major simultaneous processes: pulmonary elimination of unchanged chloroform by first-order kinetics and metabolism. Chloroform is metabolized in the liver and to a lesser extent in the kidneys and other tissues. Metabolism is dose-dependent and saturable, with a greater proportion of small doses being metabolized. The predominant pathway for chloroform is oxidation, which produces phosgene and other active metabolites and may result in an alteration of cellular integrity and viability [14].

Up to now, no by-products have been found in

human fluids which could aid biological monitoring, so we decided to evaluate chloroform concentrations in human plasma. An original head-space GC technique, suitable for determining chloroform in human plasma, was developed in order to obtain a direct measurement in subjects exposed to low levels of chloroform [15].

3.1. Determination of chloroform in plasma

The analysis was performed on plasma aliquots. Samples were analyzed by a head-space GC technique. A Dani HSS 3950 autosampler and a Varian 3400 GC equipped with a ^{63}Ni electron-capture detector and a glass steel column packed with 10% OV-1 on Chromosorb WAW were used. The carrier gas (nitrogen) flow-rate was 30 ml/min; make-up gas, 30 ml/mm; oven temperature, 70°C; inlet temperature, 150°C; detector temperature, 280°C; sensitivity, $8 \cdot 10^{-11}$ AUFS. Calibration was performed by the external standard method.

The identity of the chloroform was confirmed by GC–MS. We examined both standard samples of chloroform in *n*-pentane and standard samples of chloroform-fortified human plasma at increasing concentrations (0.1–1000 mg/l) after extraction with *n*-pentane. The identification of chloroform is based both on retention times measured on a total ion-current chromatogram and on mass chromatograms of the molecular ion and the most significant fragments of chloroform (*m/z* 47–50, 82–87, and 117–124).

Plasma chloroform was evaluated in samples of 127 volunteer subjects (81 men and 46 women) who regularly attended three swimming pools in Modena (Emilia Romagna, Italy). The pools were visited over a period of six months (Nov. 1987–April 1988), and samples were collected in 18 sampling sessions [16].

The kind of activity practised in the swimming pool was recorded, and the subjects were classified into three main groups: competitive swimmers ($n = 102$) in daily training for competitions, non-competitive swimmers ($n = 16$) who were attending swimming courses twice a week, and visitors ($n = 9$) who were present but did not swim. Every subject was asked about frequency of attendance and length of time spent at the swimming pool in the course of a week. The length of the swimming session was noted, as was the time elapsing between the end of the session and blood sampling (only for swimmers). Data were gathered regarding the possibility of exposure outside the swimming pool (e.g. occupational exposure or handling of solvents at home).

A control group of 40 volunteers with no known occupational or environmental exposure and who had never visited an indoor swimming pool were also examined: none of them exhibited plasma chloroform at levels above the limit of detection.

On the other hand, chloroform was always present in all samples collected from exposed subjects, as shown in Table 4, where the levels of plasma chloroform recorded in 18 sampling sessions in the 127 subjects who habitually frequented the swimming pools are shown, together with the chloroform levels found in air and water samples.

Table 4
Chloroform in water, in environmental air, and in plasma samples collected from 127 subjects during 18 sampling sessions

	Geom. mean	Arith. mean	Median	Range
Chloroform in water ($n = 18$) (μg/l)	30.90	32.67	30.50	17–47
Chloroform in air ($n = 18$) (μg/m^3)	178.65	213.61	172.00	66–650
Chloroform in plasma ($n = 127$) (μg/l)	0.82	1.06	0.90	0.1–3.0

Chloroform was never found in the blood samples taken from the 127 investigated subjects before entering the premises; however, after a period of exposure in the swimming pool, their blood samples invariably revealed concentrations of between 0.1 (detection limit by the analytical method) and 3 μg/l, with a geometric mean of 0.82 μg/l and a median of 0.90 μg/l.

The plasma chloroform concentrations correlated significantly ($p < 0.001$) with those in the water and the ambient air, with the number of swimmers in the pool, and with the length of time spent swimming, but not with the number of visits to the pool during the week (Table 5).

Another variable that had a significant influence on plasma chloroform levels was the type of physical activity undertaken in the pool. Comparison by means of the Student Neuman Keuls test, with significance set at $p < 0.05$, of the mean values for competitive swimmers, swimmers attending swimming courses, and non-swimmers present at the sampling sessions showed that the competitive swimmers had significantly higher values (1.22 ± 0.68 μg/l) than either the non-competitive swimmers (0.40 ± 0.15 μg/l) or the non-swimmers (0.29 ± 0.20 μg/l).

Covariance analysis, performed for blood concentration, physical intensity, age of subject, and concentration of chloroform in the ambient air, showed that this model was able to account for 67.88% of the phenomenon: 48.16% of the plasma value was linked to the concentration of chloroform in the ambient air, 4.72% to the intensity of physical activity, and 1.47% to the subject's age, with which it showed a weak negative correlation depending on the amount of time spent in the covered pool.

Since it is not easy to collect numerous blood samples in a broad survey of this type, and as alveolar air sampling is a recognized technique for determining occupational exposure [17,18], we decided to take the alveolar chloroform level as the indicator of exposure for biological monitoring. Accordingly, in a subsequent phase of our study, we studied 163 subjects, comprising swimmers and non-swimming visitors, and 77 control subjects [19].

The exposed subjects, 98 male and 65 female, aged between 5 and 44 years, were classified into three groups, depending on their activity in the pool, namely, learners (12), competitive swimmers (120), and visitors (31). A sample of alveolar air was taken from each subject before entering the premises to check whether chloroform was present before exposure; a second sample was taken at the end of the session, together with a sample of ambient air. Alveolar air samples were collected in 34 cm^3 one-way glass tubes with two valves. Subjects were asked to breath normally into the tube with open valves. At the end of expiration, the valves were closed. For analysis the tubes were heated to 37°C to recreate the conditions at the time of sampling.

3.2. Determination of chloroform in alveolar air

This determination was performed by injecting samples directly into the GC (Varian 3400) using a gas-tight syringe (Hamilton). The analytical procedure has been described previously and was the same as used for the determination of chloroform in the ambient air. Calibration was performed by external standard methods.

Chloroform was found at the detection limit

Table 5
Spearman's Rank correlation coefficients

	r	p
Chloroform in plasma (μg/l)/no. of swimmers	0.322	<0.001
Chloroform in plasma (μg/l)/time spent swimming (min)	0.573	<0.001
Chloroform in plasma (μg/l)/chloroform in water (μg/l)	0.478	<0.001
Chloroform in plasma (μg/l)/chloroform in environmental air (μg/m^3)	0.739	<0.001

value (1 $\mu g/m^3$) in some samples before exposure, while after exposure all the samples of alveolar air revealed varying concentrations of chloroform.

Of the 77 subjects not known to have been exposed to chloroform and therefore serving as controls, it was nevertheless found, albeit in traces, in 53% of the total.

The chloroform concentrations found in the samples of alveolar air taken from the 163 subjects at the end of exposure in the pool are reported in Table 6. Chloroform was always present in the samples of alveolar air collected in the pool, with values ranging between 14 and 312 $\mu g/m^3$.

Our findings show a positive correlation between alveolar chloroform and the concentration of chloroform in the ambient air ($r = 0.907$, $p = 0.002$) and a weak negative correlation with the age of the subjects ($r = -0.310$, $p < 0.001$).

As in the case of plasma chloroform, the nature of the physical activity undertaken in the pool seems to have a considerable influence on the level of alveolar chloroform concentration.

Student Neuman Keuls test, carried out to compare average levels in the three groups (competitive swimmers, learners, and visitors), again showed a significant difference between the last two groups (learners, 74.61 $\mu g/m^3$ and visitors, 58.71 $\mu g/m^3$) and the first (competitive swimmers, 104.35 $\mu g/m^3$).

Covariance analysis was performed on the whole sample (swimmers and visitors) including alveolar concentration as a dependent variable, the intensity of physical exertion as a factor, and ambient air concentrations and age as covariates. The total explained variance by this statistical model was 73.05% ($F = 107.09$, $p < 0.001$), of which 58.36% was due to chloroform in ambient air ($F = 342.14$, $p < 0.001$), 13.13% to the age of the subjects ($F = 78.20$, $p < 0.001$), and 5.11% to the intensity of physical exertion ($F = 14.98$, $p < 0.001$).

4. Kinetics of chloroform elimination

In order to ascertain whether chloroform exposure through swimming in chlorinated water has any injurious effects on health, the pharmacokinetics of this compound needs to be understood. Various studies have been carried out to determine the uptake and elimination of different chemicals in occupationally exposed subjects; the pharmacokinetics of a compound in subjects who are not occupationally exposed, however, is not well documented.

For this reason we decided to study the kinetics of chloroform elimination in exhaled breath following the typical exposure of a swimmer in an indoor swimming pool. We report here the results of a first study carried out in the winter of 1993–1994.

A competitive swimmer (male, non-smoker, 34 years old, weight 104 kg, height 186 cm) who usually swims three times a week was monitored at four swimming sessions, and alveolar air samples were taken at intervals for 10 h after each session.

On each occasion he trained intensively for 45 min, from 20.00 to 20.45, and during this period water and ambient air samples were collected

Table 6
Chloroform in water, in environmental air, and in alveolar air samples collected from 163 subjects during six sampling sessions

	Geom. mean	Arith. mean	Median	Range
Chloroform in water ($n = 6$) ($\mu g/l$)	–	35.97	29.95	19–94
Chloroform in air ($n = 6$) ($\mu g/m^3$)	–	140.33	129.85	49–280
Chloroform in alveolar air ($n = 163$) ($\mu g/m^3$)	78.05	94.09	83	14–312

following the same procedures as reported before. A pre-exposure sample was taken just before the swimmer entered the swimming pool premises; a first post-exposure sample was taken as soon as the swimming session had finished and a second one 15 min after he had left the premises. Further samples were taken every 30 min for the following 3 h while the subject was at home resting, and a final sample was taken the following morning, about 10 h after the end of exposure.

At the same time ambient air samples were collected in the swimmer's house in order to ascertain the background level of any chloroform concentration.

The levels of chloroform in the swimming-pool water ranged between 106 and 144 μg/l, while ambient air values ranged from 92 to 208 $\mu g/m^3$, with a mean value of 139 ± 24.6 $\mu g/m^3$.

The ambient air levels in the swimmer's house were always below the limit of detection by our method.

In the pre-exposure samples of alveolar air, chloroform was not detected; the levels measured after the end of exposure and the corresponding time of collection are reported in Table 7.

Chloroform concentrations in alveolar air as a function of time after exposure found in the four sampling sessions are shown in Fig. 1. The figure shows that the elimination of chloroform in alveolar air after these exposure levels usually stops within 10 h; in the samples collected at the end of this time, chloroform was detected in all the samples but one, where it was measured at a very low concentration.

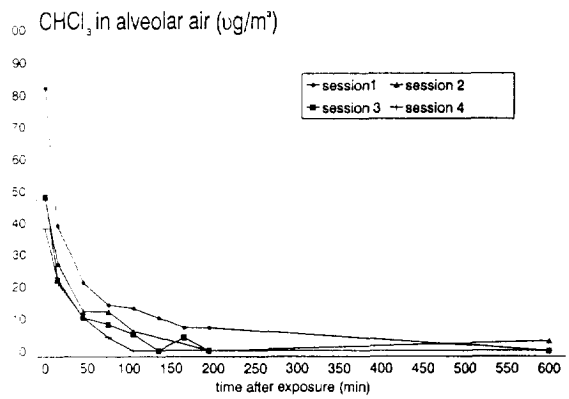

Fig. 1. Chloroform in alveolar air ($\mu g/m^3$) as a function of time after exposure in four sampling sessions.

According to our data, the elimination of chloroform follows first-order, one-compartment pharmacokinetics, and the equation describing breath decay is

$$C = C_0 e^{-kt}$$

where C is the concentration of chloroform at time t, C_0 the concentration of chloroform at time 0, and k is the first-order rate constant of the overall elimination of chloroform.

Table 7
Chloroform in alveolar air samples collected during four sampling sessions after exposure

Time after exposure (min)	Chloroform in alveolar air in sampling session ($\mu g/m^3$)			
	1	2	3	4
0	82	48	48	38
15	39	27	22	21
45	21	12	10	10
75	14	12	8	4
105	13	6	5	0
135	10	–	0	0
165	7	–	4	–
195	7	0	0	0
600	0	3	0	0

Table 8
Pharmacokinetics parameters of chloroform pulmonary elimination

Pharmacokinetics parameters	Sampling session			
	1	2	3	4
C_0 ($\mu g/m^3$)	75	45.4	45.7	37.3
k (min^{-1})	0.026	0.025	0.035	0.033
r^2	0.92	0.95	0.96	0.99
$t_{1/2}$ (min)	26	27	20	21
AUC ($\mu g/m^3$)	2815	1777	1269	1136

The pharmacokinetic parameters derived from the best-fit curves are reported in Table 8, together with the value of r^2, the biological half-time ($t_{1/2}$), and values of the area under the curve (AUC), which were calculated from the end of exposure to the last time at which data were available, 10 h after the end of exposure; r^2 values range between 0.92 and 0.99, which suggests that our model fits our data reasonably well.

The biological half-times ($t_{1/2}$) are very short and range from 20 to 27 min; our data are not sufficient to link this variable to specific factors.

The AUC values can be utilized to obtain information on the total amount of chloroform eliminated in alveolar air; considering a respiratory rate of 1 m^3/h the total amount of chloroform eliminated by the lungs in 10 h after these four swimming sessions was 28.150, 17.770, 12.690, and 11.360 mg, respectively.

5. Discussion

Throughout our investigations in covered swimming pools, chloroform was always present in the water and air at levels corresponding to the number of swimmers present in the pool. It was thus possible to identify an exposure factor of a non-occupational nature that affects subjects frequenting the pool, including those who do not swim but who remain on the premises for varying periods of time.

This population was therefore used for biological monitoring, and the findings show that the swimmers, particularly the competitive swimmers, were more heavily exposed to chloroform. The levels of concentration in the blood and alveolar air were in fact higher, the greater the degree of physical exertion; also, there was a negative correlation between chloroform levels and age, which suggests that younger subjects absorb chloroform more readily.

To complete our study we investigated the kinetics of chloroform elimination from the lungs after a period of exposure in the swimming pool under controlled conditions. Our findings point to single-stage clearance, at variance with that observed by other authors. Research conducted on subjects exposed to chloroform while showering revealed a dual-compartment model featuring a rapid initial phase of clearance from arterial blood followed by a slower second phase connected with the release of chloroform from the body tissues [20–22].

Another study conducted on a subject exposed for 30 min in the swimming pool showed two clearance peaks from the lungs, probably due to two different absorption routes: the first peak was thought to correspond to a very rapid absorption phase, most probably through inhalation, while the second, appearing 60–90 min after exposure, was linked to a slower, probably transdermal, process of absorption [6].

It is likely that the frequency of sampling in our study did not allow two distinct clearance phases to be detected. In two sessions we did in fact detect a slight increase in the amount of chloroform cleared in the 60–100 min interval, but it was statistically insignificant.

Our findings demonstrate that the clearance of chloroform absorbed after a period of exposure in the swimming pool is rapid and is complete after about 10 h from exposure–unlike other volatile halogenated substances such as trieline and perchloroethylene, which take longer to clear. It should be borne in mind, however, that the time taken by the same volatile substance to clear from the lungs may vary considerably from subject to subject [23].

As stated above, chloroform has been identified as a possible carcinogen in man and, as such, classified in group 2B by IARC. We therefore believe that all possible measures should be taken to reduce exposure as far as possible. Covered swimming pools represent a particular situation in which exposure can occur along three routes, absorption taking place through the lungs and the skin, and also by ingestion. Research should therefore be directed at reducing the generation of chloroform during the chlorination process and controlling the concentration of chloroform in the ambient air.

Acknowledgements

Our studies were supported by grants from the Italian Ministry of University and Scientific and Technological Research (40% and 60%). We are indebted to Prof. Sergio Fonda for consulting about pharmacokinetic models.

References

[1] A.J. Beech, R. Diaz, C. Ordaz and B. Palomeque, Am. J. Public Health, 70 (1980) 79.
[2] U. Lahl, K. Batjer, J.V. Duszeln, B. Gabel, B. Stachel and W. Thiemann, Water Res., 15 (1981) 803.
[3] P. Chambon, M. Taveau, M. Morin and R. Chambon, J. Vial. Water Res., 17 (1983) 65.
[4] G. Aggazzotti and G. Predieri, Water Res., 20 (1986) 959.
[5] Y. Penverne and A. Montiel, Trib. CEBEDEAU, 39 (1986) 13.
[6] C.P. Weisel and T.A. Shepard, in R.G.M. Wang (Editor), Water Contamination and Health, Marcel Dekker, New York, 1994, p. 135.
[7] J. Copaken, in Jolley, Condie, Johnson et al. (Editors), Water Chlorination Chemistry, Environmental Impact and Health Effects, Lewis Publishers, Chelsea, MI, 1990, p. 101.
[8] IARC, IARC Monograph on the Evaluation of Carcinogenic Risks to Humans, Suppl. 7, Overall Evaluations of Cancerogenicity: An Updating of IARC Monograph Vols. 1 to 42, Lyon, 1987.
[9] IARC, IARC Monographs on Evaluation of Carcinogenic Risks to Humans, Vol. 52, Chlorinated Drinking Water; Chlorination By-products; Some Other Halogenated Compounds; Cobalt and Cobalt Compounds, IARC, Lyon, 1991.
[10] L. Lukacovic, M. Mikulàs, A. Vanko and G. Kiss, J. Chromatogr., 207 (1981) 373.
[11] R. Otson, G.L. Polley and J.L. Robertson, Water Res., 20 (1986) 775.
[12] G. Aggazzotti, G. Predieri and A. Zavatti, Ig. Mod., 81 (1984) 1416.
[13] R.C. Wester and H.I. Maibach, in R.G.M. Wang (Editor), Water Contamination and Health, Marcel Dekker, New York, 1994, p. 149
[14] United States Environmental Protection Agency, Health Assessment Document for Chloroform, EPA/600/884/004F, Office of Health and Environmental Assessment, Washington, DC, September 1985.
[15] G. Aggazzotti, G. Predieri and G. Fantuzzi, J. Chromatogr., 416 (1987) 125.
[16] G. Aggazzotti, G. Fantuzzi, P.L. Tartoni and G. Predieri, Arch. Environ. Health, 45 (1990) 175.
[17] L. Wallace, E. Pellizzari, T. Hartwell et al., J. Occup. Med., 28 (1986) 603.
[18] A.C. Monster, Arch. Environ. Health, 43 (1988) 90.
[19] G. Aggazzotti, G. Fantuzzi, E. Righi, P.L. Tartoni, M.T. Cassinadri and G. Predieri, Arch. Environ. Health, 48 (1993) 250.
[20] C.P. Weisel, W.K. Jo and P.J. Lioy, J. Expos. Anal. Environ. Epidemiology, (1992), in press. Citation from C.P. Weisel and T.A. Shepard, in R.G.M. Wang (Editor), Water Contamination and Health, Marcel Dekker, New York, 1994, pp. 135–148.
[21] J.K. Raymer, K.W. Thomas, S.D. Cooper, D.A. Whitaker and E.D. Pellizzari, J. Anal. Toxicol., 14 (1990) 337.
[22] J.N. Blancato and N. Chiu, in R.G.M. Wang (Editor), Water Contamination and Health, Marcel Dekker, New York, 1994, p. 217.
[23] E.D. Pellizzari, L.A. Wallace and S.M. Gordon, J. Expos. Anal. Environ. Epidemiology, 2 (1992) 341.