Trihalogenmethanes in Drinking Water and Quantification of Health Risks

Frantisek BOZEK[1)], Milos BOZEK[2)], Jiri DVORAK
[1] Civil Protection Department
University of Defence
65 Kounicova, 662 10 Brno
CZECH REPUBLIC
frantisek.bozek@unob.cz http://www.vojenskaskola.cz
[2] 221 Squadron,
5525 Military Garrison
675 71 Sedlec, Vicenice u Nameste nad Oslavou,
CZECH REPUBLIC
milox@seznam.cz http://www.lznamest.army.cz/kontakt.htm
[3] Language Training Centre
University of Defence
44 Kounicova, 662 10 Brno
CZECH REPUBLIC
jiri.dvorak@unob.cz http://www.vojenskaskola.cz

Abstract: - The paper deals with the quantification of health risks resulting from the disinfection of drinking water and covers both non-carcinogenic and genotoxic risks. The monitoring and calculations were focused on four types of trihalogenmethanes as the main products of such disinfection having potential health risks for the exposed population. Gas chromatography with mass detector was used for the determination of the trihalogenmethanes concentrations in the drinking water. The measured concentrations of individual pollutants with probable human hepatotoxic, nephrotoxic carcinogenic, mutagenic, teratogenic and embryotoxic effects were used for the risk assessment. The risk assessment was carried out in compliance with the national methodology, respecting the standard operational procedure of U.S. EPA. The analysis was carried out in a selected region of the Czech Republic and three age groups of population were considered. It was discovered on the basis of appropriate developed exposure scenarios and the determined concentrations of contaminants that the summary non-carcinogenic risks are acceptable and genotoxic risks are tolerable.

Key-Words: - Dermal contact, Disinfection, Drinking water, Exposure, Genotoxic risk, Ingestion, Inhalation, Non-carcinogenic risk, Risk, Trihalogenmethanes.

1 Introduction

The sanitary treatment of drinking water through disinfection may produce byproducts, which have negative impacts on the quality of drinking water. Such byproducts may occur due to the interaction of oxidation agents with organic substances, which are naturally present also in ground waters. Trihalogenmethanes, including chloroform ($CHCl_3$), bromdichloromethane ($CHBrCl_2$), dibromchloromethane ($CHBr_2Cl$) and bromoform ($CHBr_3$), are the dominant products of the reaction of disinfection means on chlorine base with organic precursors.

Trihalogenmethanes damage kidney and liver and show cytotoxic, hepatotoxic and nephrotoxic effects. Carcinogenic, mutagenic, teratogenic and embryotoxic effects are not excluded either. Therefore their contents in water have to be carefully monitored and in case of higher concentrations both non-carcinogenic and genotoxic risks have to be assessed for the exposed population. The submitted paper is focused on the assessment of health risks in a selected region of the Czech Republic.

2 The Analysis of Current State

Drinking water of high quality is essential for human beings. Drinking water quality were evaluated by applying the physical, chemical and biological parameters that were in accordance with the limit values set in national or international regulations [1]. The disinfection by strong oxidants, e.g. by chlorination and ozonization, is a common practice in the treatment of drinking water with the aim to inactivate pathogens and provide microbial security of water. It was found out that toxic and potentially carcinogenic byproducts are produced during disinfection [2, 3]. It is caused by the

reaction of oxidation agent with organic microcontamination, which is a natural part of not only surface, but also ground waters [4, 5]. At the same time, during the distribution of drinking water there is a deposition of particles of various origins in the areas of the distribution network with favourable hydraulic conditions [6]. Such particles are mostly of organic origin and the analysis of organic deposits proved that the relevant part of organic water contamination are humin acids and other alkyl-derivatives with high content of $–CH_3$ and $–CH_2CH_3$ functional groups. The quantitative data on the composition of organic substances in the distribution water system are stated by Sly, as in [7]. Algae, their metabolites and the metabolites of microflora present in water and distribution network may also produce, to a lesser extent, disinfection byproducts (DBPs) [8].

More than 500 types of halogenderivatives, generally marked as DBPs, were detected by studying the chlorination of raw water containing humin acids. The most significant DBPs identified in water are halogenacetic acids [9], halogenacetonitriles, chloral hydrate, (2,2,2-trichloro-1,1-ethanediol), chlorophenols, chlorine cyan, bromates and last but not least, trihalogenmethanes (THMs) [10].

The reaction rate and the spectrum of produced DBPs depend mainly on the type of disinfection agent, its dose, concentration of precursors, holding time, water temperature and pH [11, 12]. The common disinfection agents used in the Czech Republic are chlorine gas and, to lesser extent, NaClO, ClO_2, weaker chloramine and the combination of chlorination and ozonization [13]. The application of chlorine is accompanied by the least desirable side effects and the highest risks when considering the aspect of DBPs production. The use of weaker disinfection means in comparison with chlorine gas is connected with the necessity to provide standard bacteriological quality of drinking water. Ozone is increasingly applied in the world for its high efficiency against resistant pathogens such as oocysts of *Cryptosporidium*, and lower inclination to the production of DBPs. However, all disinfection agents are oxidants producing DBPs [14].

THMs make up to 90% of DBPs during the chlorination of water. $CHCl_3$ is a dominant component of THMs, while other THMs, i.e. $CHBrCl_2$, $CHBr_2Cl$ and $CHBr_3$, are present in lower concentrations [4]. The THMs are received by inhalation, ingestion and absorption and have toxic effects. In case of a long-term exposure they damage kidneys, liver and thyroid gland. They also have a certain degree of carcinogenity according to the U.S. EPA and are classified, except for $CHBr_2Cl$, into the B2 group as probably carcinogenic to humans. $CHBr_2Cl$ is in the C group of substances classified as possibly carcinogenic to humans [15]. THMs are suspected of having negative reproduction impacts, e.g. on the infants´ lower birth weight, although this fact has not been sufficiently proved [16].

For the above mentioned reasons the THMs represent potential risks to the exposed population even in lower concentrations in drinking water and deserve a more detailed analysis [13, 17].

The international limits for THMs range from 25-250 μg dm^{-3} [12]. The limit of THMs sum in drinking water is 100 μg dm^{-3} in the Czech Republic and complies with the EU Directive [18]. However, it is necessary to emphasize that the risk resulting from the insufficient inactivation of pathogens in drinking water is of higher order priority in comparison with the health risk resulting from the presence of DBPs produced through the interaction of disinfection agent and organic precursor [12, 13].

The removal of DBPs by common water treatment procedures, such as adsorption on activated charcoal powder or granules and air stripping, is not sufficiently effective [13]. Therefore the emphasis is laid either on the removal of precursors or the application of disinfection agents other than chlorine in the water supply practice. Riverbank filtration is of considerable significance in the process of providing the microbial security and eliminating the THMs in less developed countries in which microbial contaminated water is used as a source of drinking water [19].

3 Applied Methods and Devices

The sampling of drinking water was carried out in compliance with standards [20]. The concentration of THMs in drinking water was determined by the liquid-gas extraction technology with the help of the TriPlus static head space dosing device and the Trace GC Ultra gas chromatograph with the Trace DSQ mass detector, produced by Thermoelectron Corporation as it is seen in Figure 1. The limit of determination for individual THMs was 0.1 μg dm^{-3}.

Fig. 1 Gas chromatograph with the mass detector

The assessment of non-carcinogenic and genotoxic risks was carried out in compliance with the valid Czech guidelines and instructions [21], which are based on the method proposed by the U.S. EPA [22].

The prerequisite for assessing the risk of contaminants with threshold effect is the knowledge of reference dose *RfD* [μg kg^{-1} day^{-1}], which may be calculated according to (1):

$$RfD = NOAEL \times UF^{-1} \times MF^{-1} \quad (1)$$

where *NOAEL* is the concentration of contaminant in case of which no adverse health effects are monitored, *UF* is an aggregate uncertainty factor and *MF* is a modification factor. The $UF = 10^x$, and $x \in 0 \wedge \mathrm{N}$, where N represents a symbol for the set of all natural numbers expressing the number of uncertainties and $MF \in (1; 10\rangle$ characterizes the uncertainties not covered by the *UF*.

The second prerequisite of risk quantification is the exposure assessment. The aim is to assess the amount of chronic daily intake *CDI* [μg kg^{-1} day^{-1}] for considered age category *A*, contaminant *S*, and exposure pathway *EP* expressed by equation (2) for ingestion *ING*, (3) for inhalation *INH* and (4) for dermal contact *DC*.

$$CDI_{ING} = c_w \times IR_{ING} \times b \times EF \times ED \times BW^{-1} \times AT^{-1} \quad (2)$$

$$CDI_{INH} = c_a \times IR_{INH} \times ET \times EF \times ED \times BW^{-1} \times AT^{-1} \quad (3)$$

$$CDI_{DC} = c_w \times SA \times K_p \times ET \times EF \times ED \times C_F \times BW^{-1} \times AT^{-1} \quad (4)$$

$$c_a = c_w \times f \times Q \times t \times V^{-1} \times 2^{-1} \quad (5)$$

where c_w [μg dm^{-3}] represents the average concentration of contaminant in water acquired through measurement, IR_{ING} [dm^3 day^{-1}] is the daily rate of consumed water, *b* the rate of consumed water from private sources, *EF* [day year^{-1}] the annual exposure frequency, *ED* [year] the exposure duration, *BW* [kg] the average body weight of population, *AT* [day] the time during which the concentration c_w of contaminant may be considered constant, c_a [μg m^{-3}] the concentration of contaminant in air, IR_{INH} [m^3 hour^{-1}] the rate of air inhaled per hour, *ET* [hour day^{-1}] the daily exposure time, *SA* [cm^2] the skin area which is in contact with contaminated water, K_p [cm hour^{-1}] the coefficient of skin permeability, and finally $CF = 10^{-3}$ dm^3 cm^{-3} is the cm^3 to dm^3 conversion factor. The concentration c_a can be expressed with the help of concentration c_w according to relationship (5), where *f* represents the fraction of releasable contaminant, *Q* [dm^3 hour^{-1}] the water flow per hour, *t* [hour] the showering time, and finally *V* [m^3] is the volume of bathroom.

The hazard quotient *HQ* characterizes non-carcinogenic risks as the ratio of the exposure dose expressed as *CDI* and the reference dose *RfD* according to formula (6).

$$HQ = CDI \times RfD^{-1} \quad (6)$$

If the solely additive effects of particular pathways and individual contaminants of THMs are considered while the synergic effects are neglected, then the summary HQ_{SUM} may be defined by the following relation (7).

$$HQ_{SUM} = \sum_{i=1}^{n} \sum_{j=1}^{m} HQ_{i,j} \quad (7)$$

where $HQ_{i,j}$ is the hazard quotient for *i*-contaminant *S* and *j*-pathway *EP*, *n* means the number of assessed contaminants (in our case the number of THMs) and *m* represents the number of considered pathways, while $i \in \langle 1; n\rangle$, $j \in \langle 1; m\rangle \wedge i \in \mathrm{N}$ and N is a symbol for the set of natural numbers.

When $HQ_{SUM} \leq 1$, the risk is acceptable, when $HQ_{SUM} \in (1; 4\rangle$, the risk is tolerable, and when $HQ_{SUM} > 4$, the risk is unacceptable and it is necessary either to implement corrective measures immediately, or to interrupt the supply of drinking water [21, 22].

The dimensionless quantity of individual excess cancer risk (ECR) showing the increase of the cancer risk over the general average was used for the genotoxic risk description. The individual values of *ECR* for the assessed age periods *A*, contaminants *S*, and exposure pathways *EP* were calculated according to the relation (8), where *CDI* [μg kg^{-1} day^{-1}] represents the corresponding chronic daily intake, and *CSF* [kg day μg^{-1}] is the cancer slope factor for the assessed exposure pathway *EP* and a contaminant *S*.

$$ECR = 1 - e^{-(CSF \times CDI)} \quad (8)$$

It is clear from the equation (8), that the necessary prerequisite for assessing the risk of contaminants without threshold effect is the knowledge of cancer slope factor $CSF_{EP,S}$.

The assessment of corresponding chronic daily intake *CDI* [μg kg^{-1} day^{-1}] for the assessed age category *A*, contaminant *S*, and exposure pathway *EP* was carried out with the use of relations (2), (3), (4), and (5).

The final value of the individual excess lifetime cancer risk $ELCR_{SUM}$ is given according to the formula (9) by the sum of contributions of exposure pathways *EP* for each contaminant *S*, and also the assessment of contributions in different age categories *A*.

$$ELCR_{SUM} = \sum_{k=1}^{q} \sum_{i=1}^{n} \sum_{j=1}^{m} ECR_{k.,i,j} \quad (9)$$

where $ECR_{k,i,j}$ is the individual excess cancer risk for *k*-age category, *i*-contaminant *S* and *j*-pathway *EP*, *q* means the number of assessed age categories, *n* the number of contaminants and *m* the number of pathways, while $k \in \langle 1; q\rangle$, $i \in \langle 1; n\rangle$, $j \in \langle 1; m\rangle \wedge k, i, j \in \mathrm{N}$. The applied procedure assumes the additive effects of both age categories, individual contaminants *S*, and the pathways under consideration.

The final value of ELCR may be also calculated by the weighing average $ELCR_{WEIGHT}$ according to the relation (10), which considers the length of exposure in

each of three age groups.

$$ELCR_{WEIGHT} = \sum_{k=1}^{q} t_k \times \left(\sum_{i=1}^{n} \sum_{j=1}^{m} ECR_{i,j} \right)_k \times t_\alpha \qquad (10)$$

In the equation (10) $\left(\sum_{i=1}^{n} \sum_{j=1}^{m} ECR_{i,j} \right)_k$ represents the sum of individual excess cancer risk *ECR* over the assessed *i*-contaminant *S* and *j*-pathway *EP* for a particular age group *k*. The $\sum_{k=1}^{q} t_k$ means the number of years during which the given age category *k* is assessed and $t_\alpha = 70$ years is the sum of exposure duration of all considered age subpopulations. Other symbols have the same meaning as in formula (9).

There is a consensus in the world that the acceptable limit for the individual genotoxic risk is $ELCR_{SUM} \leq 10^{-6}$. If $ELCR_{SUM} \in (10^{-6};\ 10^{-4}\rangle$, the risk is tolerable. If $ELCR_{SUM} > 10^{-4}$, the risk is unacceptable and it is necessary either to implement corrective measures immediately, or to stop the particular activity [21, 22].

4 Outcomes and Discussion

Drinking water is supplied into the group water system of the assessed region in the Czech Republic with the number of inhabitants $Z \approx 4 \times 10^5$ from two aquifers. There are two siphon mains, which consist of drilled wells 12-21 m deep and water is then supplied into the 5×10^3 m^3 group water tank. The water tank serves for the fixing of hydraulic situation in the siphons and also as an operationally essential accumulation for controlling the water intake from both water withdrawal areas. The water withdrawal area is fed from huge resources of ground waters in cretaceous layers, additionally supplied mainly by the infiltration of atmospheric precipitations into the rock environment.

Permission for water intake from both intake structures is 1.08 m^3 s^{-1}. The ground water is mixed with approximately 10 % of surface water treated through the technology of preionization, coagulation, flocculation, sedimentation and filtration. The origin of water guarantees its constant quality complying with the requirements of the Directive [18]. Before being supplied into the distribution network the water is the subject to homogenization, aggregation, sedimentation, filtration, and finally disinfection with ClO_2 produced directly in water according to equation (11). Thus it is necessary, besides other things, to monitor the remains of unhealthy chlorite.

$$ClO_2^- + Cl_2 = ClO_2 + 2Cl^- \qquad (11)$$

The drinking water was taken at five sampling locations in order to determine the concentration of THMs and cover the assessed region appropriately. The findings are recorded in Table 1.

Table 1 The concentrations of THMs (neg. means the absence of detector response)

Type of THM	Unit	Sampling location					Uncertainty [%]
		A	B	C	D	E	
$CHCl_3$	μg dm^{-3}	0.5	1.1	0.3	< 0.1	1.7	± 35
$CHBrCl_2$	μg dm^{-3}	0.3	1.6	0.2	neg.	0.4	± 35
$CHBr_2Cl$	μg dm^{-3}	< 0.1	1.9	< 0.1	neg.	0.1	± 35
$CHBr_3$	μg dm^{-3}	neg.	0.3	neg.	neg.	neg.	± 35
Sum of THMs concentrations	μg dm^{-3}	0.9	4.9	0.6	0.1	2.2	± 35

It is clear from the Table 1, that the sampling location B used for the characterization of risk is the most problematic from the aspect of summary content of THMs.

The following principles were followed during determining the exposure scenarios of non-carcinogenic effects of THMs and the calculation of individual chronic daily intake *CDI*:

a) Three subpopulations were determined for the risk quantification: newborns up to the age of two months, children up to six years, and adults.
b) Ingestion, inhalation and dermal contact were considered as the exposure pathways.
c) The exposure factors were either adopted from methodologies [21, 22] or estimated in case the data were absent.
d) The reference doses *RfD* for individual exposure pathways and pollutants were adopted from the U.S. EPA [15, 23] and are presented in the Table 2.
e) It was not feasible for the calculation of inhalation exposure to estimate the value of THMs concentration in the indoor air, because the THMs are released during any handling of free water level which is in contact with this air. As a partial compensation of this fact the air exchange was not considered when people stayed in the bathroom.
f) With the absent *RfD* for inhalation and dermal contact the calculation of exposure was carried out by the substitution of common inhalation and dermal pathways by oral intake.

Table 2 Values of reference doses for particular THMs and exposure ways [15, 23]

Reference dose	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$
Ingestion RfD_{ING}	μg kg^{-1} day^{-1}	10.0	20.0	20.0	20.0
Inhalation RfD_{INH}	μg kg^{-1} day^{-1}	8.6E-02	-	-	-
Dermal contact RfD_{DC}	μg kg^{-1} day^{-1}	2.0	-	-	-

The calculated values of *CDI* and non-carcinogenic risks expressed by *HQ* for individual exposure pathways and THMs are presented in Table 3 for the subpopulation of newborns up to the age of two months, in Table 4 for children up to the age of six and finally in Table 5 for adults. At the same time the value of summary risk calculated according to equation (6) is presented in the charts under the assumption of additive effects of individual THMs and the considered exposure pathways.

Table 3 Values of chronic daily intakes and hazard quotients for newborns up to age of two months

Characteristics	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$	Sum of $(HQ_{EP})_A$ for assessed exposure pathways
$CDI_{ING, S}$	μg kg^{-1} day^{-1}	3.75E-02	5.45E-02	6.48E-02	1.02E-02	
$CDI_{INH, S}$	μg kg^{-1} day^{-1}	2.41E-02	-	-	-	
$CDI_{DC, S}$	μg kg^{-1} day^{-1}	1.08E-02				
$HQ_{ING, S}$	non-dimensional	3.75E-03	2.73E-03	3.24E-03	5.10E-04	1.02E-02
$HQ_{INH, S}$	non-dimensional	2.80E-01	2.73E-03	3.24E-03	5.10E-04	2.92E-01
$HQ_{DC, S}$	non-dimensional	5.40E-03				
Sum of $(HQ_S)_A$ for assessed THMs	non-dimensional	2.89E-01	5.45E-03	6.48E-03	1.02E-03	HQ_{SUM}
						3.02E-01

Table 4 Values of chronic daily intakes and hazard quotients for children up to the age of six

Characteristics	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$	Sum of $(HQ_{EP})_A$ for assessed exposure pathways
$CDI_{ING, S}$	μg kg^{-1} day^{-1}	4.57E-02	6.65E-02	7.89E-02	1.25E-02	
$CDI_{INH, S}$	μg kg^{-1} day^{-1}	3.03E-02	-	-	-	
$CDI_{DC, S}$	μg kg^{-1} day^{-1}	3.46E-03				
$HQ_{ING, S}$	non-dimensional	4.57E-03	3.32E-03	3.95E-03	6.23E-04	1.25E-02
$HQ_{INH, S}$	non-dimensional	3.53E-01	3.32E-03	3.95E-03	6.23E-04	3.63E-01
$HQ_{DC, S}$	non-dimensional	1.73E-03				
Sum of $(HQ_S)_A$ for assessed THMs	non-dimensional	3.59E-01	6.65E-03	7.89E-03	1.25E-03	HQ_{SUM}
						3.76E-01

Table 5 Values of chronic daily intakes and hazard quotients for adults

Characteristics	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$	Sum of $(HQ_{EP})_A$ for assessed exposure pathways
$CDI_{ING, S}$	μg kg^{-1} day^{-1}	1.58E-02	2.30E-02	2.73E-02	4.32E-03	
$CDI_{INH, S}$	μg kg^{-1} day^{-1}	9.05E-03	-	-	-	
$CDI_{DC, S}$	μg kg^{-1} day^{-1}	5.42E-04				
$HQ_{ING, S}$	non-dimensional	1.58E-03	1.15E-03	1.37E-03	2.16E-04	4.32E-03
$HQ_{INH, S}$	non-dimensional	1.05E-01	1.15E-03	1.37E-03	2.16E-04	1.08E-01
$HQ_{DC, S}$	non-dimensional	2.71E-04				
Sum of $(HQ_S)_A$ for assessed THMs	non-dimensional	1.07E-01	2.30E-03	2.73E-03	4.32E-04	HQ_{SUM}
						1.12E-01

It is clear from the acquired outcomes that despite the fact the calculated risk is rather overestimated in relation to the developed exposure scenarios, the $HQ < 1$ and the risk is consequently acceptable for all the considered subpopulations. As expected, preschool children are the most sensitive subgroup. Newborns up to the age of two months are slightly less threatened as they consume drinking water mainly from other than private sources. The group of adults from six to seventy years is relatively the most resistant.

The following principles were followed during determining the exposure scenarios of carcinogenic effects of THMs and the calculation of individual chronic daily intakes *CDI* for age category *A*, contaminant *S*, and exposure pathway *EP*:

a) Three subpopulations were determined for the risk quantification: toddlers up to one year of age, children from one to eighteen years, and adults from eighteen to seventy years.
b) Ingestion, inhalation and dermal contact were considered as the exposure pathways.
c) The exposure factors were either taken from the methodical instructions [21, 22] or estimated in case the data were absent.
d) The cancer slope factors *CSF* [kg day μg^{-1}] for contaminants *S* and exposure pathways *EP* were taken from the U.S. EPA [24] and Oak Ridge National Laboratory [24] materials and are shown in Table 6.
e) For the calculation of exposures through inhalation it was not feasible to express numerically the values of individual THMs backgrounds in the „indoor" air, where THMs are released during an arbitrary manipulation with water with the free water level being in contact with the air. In order to partially compensate such an effect, the exchange of air was not considered during the time the persons stayed in the bathroom.
f) As the $CSF_{A,\ S,\ DC}$ cancer slope factor for the age group *A*, contaminant *S* and dermal contact was absent and the $CSF_{A,\ S,\ INH}$ cancer slope factor for the same age group *A*, contaminant *S* and inhalation was known, the excess cancer risk $ECR_{A,\ S,\ DC}$ caused by dermal contact was considered as being of one third of cancer risk caused by inhalation pathway, i.e. $ECR_{A,\ S,\ INH} = 3 \times ECR_{A,\ S,\ DC}$ which is in compliance with the recommendation of the U.S EPA [22].
g) As the cancer slope factors for contaminant *S* and both for inhalation $CSF_{S,\ INH}$ and dermal contact $CSF_{S,\ DC}$ were absent, the cancer risk caused by both inhalation and dermal contact was considered equal to the cancer risk caused by ingestion, i.e. $(ECR_{S,\ INH+DC})_k = (ECR_{S,\ ING})_k$ again in compliance with the recommendation of the U.S EPA [22].

Table 6 Values of chronic daily intakes and hazard quotients for adults

Exposure pathway	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$
Ingestion $CSF_{ING,\ S}$	kg day μg^{-1}	6.1E-06	6.2E-05	8.4E-05	7.9E-06
Inhalation $CSF_{INH,\ S}$	kg day μg^{-1}	8.1E-05	-	-	3.9E-06
Dermal contact $CSF_{DC,\ S}$	kg day μg^{-1}	3.1E-05	-	-	-

The calculated values of *CDI* and genotoxic risks expressed by *ECR* for individual exposure pathways and THMs are presented in Table 7 for the subpopulation of toddlers up to one year of age, in Table 8 for children from one to eighteen years and finally in Table 9 for adults from eighteen to seventy years. At the same time the value of summary risk $(ECR_S)_A$, $(ECR_{EP})_A$, and $(ECR_{S,\ EP})_A$ for every *k*-age category *A* through all considered contaminants *S* and exposure pathways *EP* is presented in the Tables 7, 8, and 9 under the assumption of additive effects of individual THMs and the considered exposure pathways.

Table 7 Individual chronic daily intakes $CDI_{S,\ EP}$, individual excess cancer risks $ECR_{S,\ EP}$, the values of summary risk $(ECR_S)_A$, $(ECR_{EP})_A$, and $(ECR_{S,\ EP})_A$ for toddlers up to one year of age

Characteristics	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$	Sum of $(ECR_{EP})_A$ for assessed exposure pathways
$CDI_{S,\ ING}$	$\mu g\ kg^{-1}\ day^{-1}$	2.48E-02	3.61E-02	4.28E-02	6.76E-03	
$CDI_{S,\ INH}$	$\mu g\ kg^{-1}\ day^{-1}$	1.82E-02	-	-	4.96E-03	
$CDI_{S,\ DC}$	$\mu g\ kg^{-1}\ day^{-1}$	5.16E-03			1.83E-03	
$ECR_{S,\ ING}$	non-dimensional	1.51E-07	2.24E-06	3.60E-06	5.34E-08	6.04E-06
$ECR_{S,\ INH}$	non-dimensional	1.47E-06	2.24E-06	3.60E-06	1.93E-08	7.50E-06
$ECR_{S,\ DC}$	non-dimensional	1.60E-07			6.45E-09	
Sum of $(ECR_S)_A$ for assessed THMs	non-dimensional	1.78E-06	4.48E-06	7.20E-06	7.92E-08	$(ECR_{S,\ EP})_A$
						1.35E-05

The final value of excess lifetime cancer risk was calculated as a simple sum $ELCR_{SUM}$ through the selected age groups *A* according to the equation (9), or by the weighing average $ELCR_{WEIGHT}$ according to the relation (10) which considers the length of exposure in each age of three age groups.

Table 8 Individual chronic daily intakes $CDI_{S,\,EP}$, individual excess cancer risks $ECR_{S,\,EP}$, the values of summary risk $(ECR_S)_A$, $(ECR_{EP})_A$, and $(ECR_{S,\,EP})_A$ for children from one to eighteen years

Characteristics	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$	Sum of $(ECR_{EP})_A$ for assessed exposure pathways
$CDI_{S,\,ING}$	μg kg^{-1} day^{-1}	1.93E-02	2.81E-02	3.34E-02	5.27E-03	
$CDI_{S,\,INH}$	μg kg^{-1} day^{-1}	1.38E-02	-	-	3.77E-03	
$CDI_{S,\,DC}$	μg kg^{-1} day^{-1}	9.70E-04			-	
$ECR_{S,\,ING}$	non-dimensional	1.18E-07	1.74E-06	2.81E-06	4.16E-08	4.71E-06
$ECR_{S,\,INH}$	non-dimensional	1.12E-06	1.74E-06	2.81E-06	1.47E-08	5.72E-06
$ECR_{S,\,DC}$	non-dimensional	3.01E-08			4.90E-09	
Sum of $(ECR_S)_A$ for assessed THMs	non-dimensional	1.27E-06	3.48E-06	5.62E-06	6.12E-08	$(ECR_{S,\,EP})_A$
						1.04E-05

Table 9 Individual chronic daily intakes $CDI_{S,\,EP}$, individual excess cancer risks $ECR_{S,\,EP}$, the values of summary risk $(ECR_S)_A$, $(ECR_{EP})_A$, and $(ECR_{S,\,EP})_A$ for adults from eighteen to seventy years

Characteristics	Unit	$CHCl_3$	$CHBrCl_2$	$CHBr_2Cl$	$CHBr_3$	Sum of $(ECR_{EP})_A$ for assessed exposure pathways
$CDI_{S,\,ING}$	μg kg^{-1} day^{-1}	1.58E-02	2.30E-02	2.73E-02	4.31E-03	
$CDI_{S,\,INH}$	μg kg^{-1} day^{-1}	9.04E-03	-	-	2.47E-03	
$CDI_{S,\,DC}$	μg kg^{-1} day^{-1}	5.42E-04			-	
$ECR_{S,\,ING}$	non-dimensional	9.64E-08	1.43E-06	2.29E-06	3.41E-08	3.85E-06
$ECR_{S,\,INH}$	non-dimensional	7.32E-07	1.43E-06	2.29E-06	9.63E-09	4.48E-06
$ECR_{S,\,DC}$	non-dimensional	1.68E-08			3.21E-09	
Sum of $(ECR_S)_A$ for assessed THMs	non-dimensional	8.45E-07	2.86E-06	4.58E-06	4.69E-08	$(ECR_{S,\,EP})_A$
						8.33E-06

The acquired outcomes are summarized in Table 10. It is obvious that the individual summary cancer risk for all subpopulations $(ECR_{S,\,EP})_A < 10^{-5}$. It approximates the value of 10^{-5} also in the most sensitive subpopulation of toddlers despite the fact that the calculated risk is probably considerably overestimated. The above mentioned fact may be explained by the way of constructing the exposure scenarios and the validity uncertainties of a number of input data, which were used for the genotoxic risk assessment. The genotoxic risk for each subpopulation may therefore be considered as socially tolerable.

It is not surprising that the most sensitive group are toddlers up to one year, followed by the category of children at the age of one to eighteen years and the least threatened group is the population of adults from the age of eighteen.

It is also evident from the Table 10 that the level of excess lifetime of cancer risk *ELCR* over the general average differs depending on the way of quantification for which there were used in principle two numerically different methods.

The value of $ELCR_{SUM}$ was acquired under the assumption of additive effects in the selected *k*-age categories and is approximately six times higher in comparison with the value of $ELCR_{WEIGHT}$, which was calculated as a weighted average considering the duration of exposure in individual age categories. We take it that the value of the weighted excess lifetime genotoxic risk $ELCR_{WEIGHT} = 8.9\times10^{-6}$ is the closest to reality with regard to the existing uncertainties and the used exposure scenarios and factors. This value is not too far from the limit of social acceptability $ELCR \le 10^{-6}$, recognized by the U.S. EPA.

The genotoxic impacts on the population exposed to the effects of one or more carcinogens may be assessed with the help of annual population cancer risk *APCR*. The *APCR* [citizens year^{-1}] represents the average number of cancer in the exposed population of *Z* citizens during one year while the assumed average length of life is 70 years. The $APCR_{SUM}$ accepts the additive effect of carcinomas over the assessed age categories. It is based on the $ELCR_{SUM}$ and is given by the relation (9). The $APCR_{WEIGHT}$ represents the weighted average of annual population risk, stems from the $ELCR_{WEIGHT}$ and was calculated with the help of the relation (13).

$$APCR_{SUM} = ELCR_{SUM} \times Z \times 70^{-1} \qquad (11)$$

$$APCR_{SUM} = ELCR_{WEIGHT} \times Z \times 70^{-1} \qquad (12)$$

The calculated values of $APCR_{SUM}$ and $APCR_{WEIGHT}$ for the assessed region with the number of exposed inhabitants approximately $Z = 4\times10^5$ are shown in the Table 10. It is logical with regard to the relations (9) and (10) that, similarly to the comparison of $ELCR_{SUM}$ and $ELCR_{WEIGHT,}$ the reality is reflected more by the value of $APCR_{WEIGHT}$.

Table 10 Genotoxic risks resulting from the consumption of drinking water containing trihalogenmethanes

Genotoxic risk	Unit	Age group		
		Toddler up to the age of one	Child 1-18 years	Adult 18-70 years
$(ECR_{S,EP})_A$	non-dimensional	1.35E-05[a)]	1.04E-05[b)]	8.33E-06[c)]
$ELCR_{SUM}$	non-dimensional	3.22E-05[d)]		
$ELCR_{WEIGHT}$	non-dimensional	8.91E-06[e)]		
$APCR_{SUM}$	citizen year^{-1}	1.84E-01[f)]		
$APCR_{WEIGHT}$	citizen year^{-1}	5.09E-02[g)]		

The input data used for the quantification of genotoxic risk include certain degree of uncertainties, which undoubtedly affected the outcomes of assessment. The uncertainties are mainly as follows:

a) Data of the concentrations of THMs are objective, with the level of uncertainty ± 35 %.
b) The value of risk (*HQ, ELCR*) within the assessed region is significantly affected by the selection of sampling spot and the fact, whether water is taken from various sources and locations. The risk assessment considered the least favourable option with the maximum sum of concentrations of THMs. The values of risk will be significantly lower at other sampling spots as it results from the measured values of THMs concentrations presented in Table 1.
c) The validity of *RfD* and *CSF*, taken from the U.S. EPA databases, is assessed by the institution itself as „Medium".
d) The calculations of *CDI* are based on the assumption of full absorption of contaminants in the human organism, which is not very likely to happen in practice. This fact also increases the values of *HQ* and *ELCR*.
e) The exposure scenarios, developed for the assessed age categories, try to model the behaviour of people during ingestion and consumption of drinking water. There is not elaborated a standardized model for toddlers and children from one to eighteen years in the national methodical instructions. Therefore a number of exposure factors, especially for these age categories, had to be either adopted from abroad [15, 22, 24] or estimated.
f) The process of ingestion and consumption of drinking water is highly variable and is the function of many factors, such as sex, education, social and cultural environment, household equipment, habits, etc. which bring further uncertainties into the construction of exposure scenarios.
g) There were not found adequate reference doses *RfD* for inhalation and dermal intake in case of $CHBrCl_2$, $CHBr_2Cl$ and $CHBr_3$ in the available materials. Therefore the intake of joint inhalation and dermal pathways was considered as equivalent to the intake through ingestion, which was in compliance with the recommendation of the U.S. EPA [22]. However, the above mentioned substitution may increase the value of $HQ_{INH} + HQ_{DC}$ by one order especially among newborns.
h) The inhalation/dermal ratio of THMs intake was $p_{INH/DC} = 3$ for the needs of risk assessment. It was based on the experiments with $CHCl_3$, although there can be found $p_{INH/DC} \in \langle 1; 3 \rangle$ in the literature. The choice of $p_{INH/DC}$ does not significantly affect the assessment of genotoxic risk expressed in the form of $ELCR_{SUM}$ and $ELCR_{WEIGHT}$, because $p_{INH/DC} = 3$ was applied solely to $CHBr_3$, the concentration of which in the samples of drinking water was much lower compared to other THMs.
i) There were neither found CSF_{INH} for inhalation nor CSF_{DC} for absorption in case of $CHBrCl_2$ and $CHBr_2Cl$ in the available literature. That is why the intake of joint inhalation and dermal pathways was assessed as equivalent to the intake through ingestion, which was in compliance with the recommendation of the U.S. EPA [22]. However, the above mentioned substitution may increase the value of the sum of chronic daily intake $CDI_{INH} + CDI_{DC}$ by one order especially among toddlers up to the age of one.
j) The level of excess lifetime cancer risk *ELCR* over the general average is dependent on the applied

[a)] the value presented in the Table 7
[b)] the value presented in the Table 8
[c)] the value presented in the Table 9
[d)] calculated with the help of equation (9)
[e)] calculated with the help of equation (10)
[f)] calculated from the relation (11)
[g)] calculated from the relation (12)

option of numerical quantification of *ELCR*, as it is demonstrated on the acquired values of $ELCR_{SUM}$ and $ELCR_{WEIGHT}$ calculated according to the relation (9) eventually (10).

k) The degree of genotoxic risk $ELCR_{SUM}$ and $ELCR_{WEIGHT}$ will also be the function of dividing the exposure into the assessed age categories *A*, the selection of which will affect the values $(ECR_{S,\,EP})_k$.

l) The assumption of the additive effect of THMs is another factor increasing the uncertainty of the assessment of HQ_{SUM}, $ELCR_{SUM}$ and $ELCR_{WEIGHT,}$ because the mutual interaction of THMs may show not only synergic, but also antagonistic effects.

m) The interaction of THMs with other compounds present in the analyzed samples of drinking water was not considered either. It also reduces the validity of acquired outcomes.

5 Conclusion

The non-carcinogenic and genotoxic risks were quantified from the long term consumption of drinking water supplied by a group water system to the inhabitants of a selected region of the Czech Republic. The assessed indicator of risk was the group of four THMs, i.e. chloroform, bromoform, bromdichloromethane, and dibromchloromethane. The above mentioned pollutants which are the byproducts of the interaction of disinfection agents with organic precursors are constantly present in the drinking water supplied through the distribution network.

The $HQ_{SUM} < 1$ for all the assessed subpopulations. Therefore there is no reason to worry about the system toxic risk. It is obvious from the standard procedure followed during the calculation of exposures and risk quantification that the HQ_{SUM} value is the least favourable in case of both children subpopulations, especially for preschool children. Newborns up to the age of two months are threatened less, because they drink only a limited amount of drinking water from private sources. The subgroup of adults is significantly less sensitive.

The value of summary excess cancer risk including all exposure pathway *EP* and identified pollutants *S* from the group of THMs was $(ECR_{S,\,EP})_k < 1{,}5\times10^{-5}$ for all the assessed age categories *A*. The genotoxic risk is tolerable for all subpopulations, because the level of acceptability recommended by the U.S. EPA is $ELCR \le 10^{-6}$. Toddlers up to the age of one are the most sensitive subpopulation with $(ECR_{S,\,EP})_k \approx 1.35\times10^{-5}$ followed by the group of children from one to eighteen years with the value of 1.04×10^{-5}. The least endangered group is the subpopulation of adults, for which the value of $(ECR_{S,\,EP})_k$ is in the order of E-06.

Similar outcomes are valid for the *ELCR,* which exceeds the risk over general average. Scooping proved there is a tolerable level of life-long genotoxic risk for the threatened population of the region supplied with the drinking water with the maximal detected concentration of THMs. The final value of *ELCR* is highly affected by the way of numerical quantification. The $ELCR_{SUM} \approx 3.2\times10^{-5}$ assuming there are additive effects of selected age categories, while the application of weighted average considering the duration of exposure in individual age categories resulted in $ELCR_{WEIGHT} \approx 8.9\times10^{-6}$, which was approximately six times lower.

The annual population cancer risk APCR was calculated with the use of $ELCR_{SUM}$ and $ELCR_{WEIGHT}$. The calculated value of $APCR_{WEIGHT} \approx 5.1\times10^{-2}$ people per year reflects the reality rather than the value of $APCR_{SUM} \approx 1.8\times10^{-1}$ people year^{-1}, and it is the same with the $ELCR_{WEIGHT}$ when compared with the $ELCR_{SUM}$.

The non-carcinogenic and genotoxic risks resulting from the long term consumption of drinking water from the regional group water supply may be considered rather overestimated. At the same time the risks are insignificant with regard to the fact that the most problematic sampling spot was selected and considering the construction of exposure scenarios. The above mentioned statement is supported especially by the selection of the sum of concentrations of THMs in drinking water at the most problematic risk analysis sampling site. It can be expected that only a small number of inhabitants of the region will be exposed to these maximal detected concentrations. The concentrations of THMs at other four sampling sites are lower.

The above mentioned statements lead to a legitimate assumption that the calculated values of indicators of both non-carcinogenic and genotoxic risk may currently be accepted by society as a whole. Such a conclusion corresponds with the comparison of the values of THMs concentrations with the currently valid limits for their individual and summary concentrations [18].

Based on the acquired outcomes it may be concluded that the current situation does not require measures to be taken immediately or in a short time period aimed at eliminating or minimizing the origination or presence of THMs in drinking water supplied to the consumers through the regional water supply system. However, in the future it will be suitable to take gradual measures in order to reach the target value $ELCR_{WEIGHT} \le 10^{-6}$ at all sampling sites as recommended by the U.S. EPA.

References:

[1] Zouboulis, A. I., Tzoupanos, N. D., Moussas, P. A. Inorganic Pre-Polymerized Coagulants: Current

Status and Future Trends. In Markatos, N. et al. (Eds.). *Proceedings. of the 3rd IASME/WSEAS International Conference on Energy, Environment, Ecosystems and Sustainable Development*. Agios Nikolaos, Crete Island, Greece : WSEAS Press 2007, pp. 292-300. ISSN 1790-5095. ISBN 978-960-8457-88-1.

[2] Rook, J. J. Formation of Haloforms During Chlorination of Natural Waters. *Water Treatment and Examination*, Vol.23, No.2, 1974, pp. 234-243.

[3] Lekkas, D. F. Identification of Water Quality Changes in Athens Water System - Limitations and Perspectives. *WSEAS Transactions on Environment and Development*. Vol.1, No.1, 2005, pp. 69-74.

[4] Singer, P. C. Control of Disinfection By-Products in Drinking Water. *Journal of Environmental Engineering*, Vol.120, No.4, 1994, pp. 727-744.

[5] Papaioannou, et al. Assessment of Soil and Groundwater Quality and Hydrogeological Profile of Drama's Prefecture, North Greece. *WSEAS Transactions on Environment and Development*. Vol.2, No.10, 2006, pp. 1276-128.

[6] Zouboulis, A. I., Tzoupanos, N. D., Moussas, P. A. Inorganic Pre-Polymerized Coagulants: Current Status and Future Trends. In Markatos, N. et al. (Eds.). *Proceedings. of the 3rd IASME/WSEAS International Conference on Energy, Environment, Ecosystems and Sustainable Development*. Agios Nikolaos, Crete Island, Greece : WSEAS Press 2007, pp. 292-300, ISSN 1790-5095. ISBN 978-960-8457-88-1.

[7] Sly, L. I., Hodgkinson, M., Arunpairojana, V. Deposition of Manganese in a Drinking Water Distribution System. *Applied and Environmental Microbiology*, Vol.56, No.3, 1990, pp. 628-639.

[8] Tzoupanos, N. D., Zouboulis, A. I. Coagulation-Floculation Processes in Water/Wastewater Treatment: The Application of New Generation of Chemical Reagents. In Clussler, E. L. *Proceedings. of the 6th IASME/WSEAS International Conference on Heat Transfer, Thermal Engineering and Environment (HTE´08)*. Rhodes, Greece : WSEAS Press, 2008, pp. 309-317. ISSN 1790-5095. ISBN 978-960-6766-97-8.

[9] Ghomsheh, S. M. T., Soltanieh, M., Mosavi, M. Overview on the Separation and Removal of Haloaceticacids in Drinking Water. In Kallel, A. et al. (Eds.). *Proceedings of the 4th WSEAS International Conference on Waste Management, Water Pollution, Air Pollution, Indoor Climate (WWAI´10)*. Kantaoui, Sousse, Tunisia: WSEAS Press, 2010, pp. 25-31. ISSN 1790-5095. ISBN 978-960-474-190-8.

[10] WHO. *Disinfectants and Disinfection By-Products - Session Objectives World Health Organization Seminar Pack for Drinking Water Quality*. 2002. [on line]. [2010-10-14]. URL: <http://www.who.int/entity/water_sanitation_health /dwq/S04.pdf>.

[11] Whitaker, H. et al. Description of Trihalomethane Levels in Three United Kingdom Water Suppliers. *Journal of Exposure Analysis and Environmental Epidemiology*, Vol.13, No. 1, 2003, pp. 17-23.

[12] WHO. *Guidelines for Drinking-Water Quality. Vol.1 Recommendations*. 1st Addendum to 3rd; Geneva : WHO, 2006.

[13] Bozek, F. et al. Disinfection of Drinking Water and Non-Carcinogenic Risks. In Mladenov, V. et al (Eds.). *Proceedings of the WSEAS International Conference on Development, Energy, Environment, Economics*. Puerto De La Cruz, Tenerife : WSEAS Press, 2010, pp. 245-250. ISSN 1792-6653. ISBN 978-960-454-253-0.

[14] Bonacquisti, T. P. A Drinking Water Utility's Perspective on Bromide, Bromate, and Ozonation. *Toxicology*, Vol.221, No.2-3, 2006, pp. 145-148.

[15] U.S. EPA. Integrated Risk Information System (IRIS). *Database of Toxicological Parameters for Human Health*. [on line]. [2010-09-11]. URL: <http://www.epa.gov/ iris/subst>.

[16] Nieuwenhuijsen, M. J. et al. Chlorination Disinfection By-products in Water and Their Association with Adverse Reproductive Outcomes: A Review. *Occupational and Environmental Medicine*, Vol.57, No.2, 2000, pp. 73-85.

[17] Papaioannou, et al. Quality Control of Drinking Water and Public Health. WSEAS *Transactions on Environment and Development*. Vol.2, No.6, 2006, pp. 845-850.

[18] The Council of the European Union. (CEU). *Council Directive 98/83/EC on the Quality of Water Intended for Human Consumption*. Brussels : CEU, 1998.

[19] Weiss, W. J. et al. Bank Filtration - Fate of DBP Precursors and Selected Microorganisms. *J. of American Water Works Association*, Vol.95, No.10, 2003, pp. 68-81.

[20] Czech Office for Standards, Metrology and Testing (COSMT). ČSNENISO 5667-3. *Water Quality-Sampling-Guidance for the Preservation of Samples and Handling.* Prague : COSMT, 2004.

[21] Ministry of Environment (MoE). *Metodical Instructions No 12 for the Risk Analysis of Contaminated Area*. MoE Bulletin, Vol.XV, article 9, 2005, pp. 1-41.

[22] U.S. EPA. *Risk Assessment Guidance for Superfund: Human Health Evaluation Manual.*

Interim Final. EPA/540/1-89/002, Washington, D.C. : U.S. EPA, 1989.

[23] U.S. EPA. *EPA Superfund, Record of Decision*. EPA/ROD/R04-01/534. Washington, D.C. : U.S. EPA, 2001. [on line]. [2010-09-30]. URL: <http://www.epa.gov/superfund/sites/rods/fulltext/r0401534.pdf>.

[24] Oak Ridge National Laboratory. (ORNL). *The Risk Assessment Information System (RAIS)*. [on line]. [2010-09-11]. URL: <http://risk.lsd.ornl.gov/>.