

RISK ASSESSMENT OF EXPOSURE TO TRIHALOMETHANES (THMs) IN POTABLE WATER OF RURAL AREAS, BEHIRA GOVERNORATE, EGYPT.

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ABSTRACT

Human health risks from exposure to disinfection by-products are of concern based on epidemiological and toxicological evidences of potential adverse effects. Risk assessment of THM compounds; on particular, arises as an urgent need for ensuring safety of drinking water resources. Potential risk associated with exposure to THMs in drinking water of Kafr El-Dawar district as a rural area was investigated. Data revealed that levels of Σ THMs were higher than Egyptian Maximum Contaminant Level (EMCL) and reached its highest value ($264.67 \mu\text{g.L}^{-1}$) during winter 2002. Chloroform was the major THM detected especially during winter and spring seasons. Total chronic daily intakes (CDI) of THM mixture through inhalation, dermal and ingestion exposure pathways were 0.016, 7.6×10^{-6} and $0.024 \text{ mg.kg}^{-1}.\text{day}^{-1}$; respectively at 50th percentile of probability. Estimated cancer risk reached its highest value; 5.8×10^{-3} for chloroform via inhalation at 90th percentile. The measured compounds were classified according to their weight of evidence as B2 for chloroform and BDCM and C for DBCM. Hazard quotient (HQ) was found to be of a high value for chloroform (9.3) through ingestion pathway at 90th percentile. Sum of hazard indices recorded 10.4 for chloroform, BDCM, and DBCM exceeding their reference doses. Accordingly, the obtained data can provide an evidence of potential systemic and/or carcinogenic effects due to exposure to THM through different pathways.

Key words: Health risk assessment; trihalomethanes; drinking water; inhaled air.

INTRODUCTION

Drinking water is always disinfected with chlorine to kill microorganisms that can cause serious illness and death. This kind of disinfection is the most commonly employed in Egypt nowadays but unfortunately chlorination may create tiny amounts of disinfection by-products (DBPs), resulting from reaction of chlorine with natural organic matter (Bellar et al., 1974). Within these DBPs, trihalomethanes (THMs), a carcinogenic organic halogenated byproduct composed of chloroform, bromoform, bromodichloromethane, (BDCM), and dibromochloromethane (DBCM), have been recognized as potentially hazardous to human health and are the major byproducts of chlorination (Bull et al., 1995; Hsu et al., 2001). Correlation between chlorination of drinking water and cancer mortality has been investigated (Morris, 1995; Gallagher, et al., 1998; Yang et al., 1998, 2000) in particular urinary bladder and colorectal cancer (Pilotto, 1995; Cantor et al., 1998; Yang et al., 1998). Additionally, exposure to chlorinated byproducts was related to spontaneous abortion and other adverse reproductive outcomes (Kramer et al., 1992; and Waller et al., 1998). Indeed EPA revised downwards by 20%, the limits of THMs in public drinking water since

prolonged exposure to such chemicals can increase the chance of cancer (US EPA, 2001).

Kafr El-Dawar district, Behira Governorate, has a total area of 601.94 km² and total population of 750,000 consuming $47.7 \times 10^3 \text{ m}^3/\text{day}$ of drinking water and working mainly in agriculture, textile activities and chemicals/dyes production. Most areas of this district are ill-served and have no access to safe drinking water with poor status of distribution network pipelines particularly at remote ends. Therefore, the purpose of this study was to provide an assessment of the potential hazard effects of multipathway exposure to questionable concentrations of THMs detected in drinking water samples collected from Kafr El-Dawar as a rural area.

METHODS

The risk assessment of THMs in drinking water in Kafr El-Dawar is based on the data collected by Abdallah and Gaber 2004, where water samples were collected on spatio-temporal basis during 2002 from eight zones as illustrated in Fig (1). Samples were prepared, extracted and determined on gas chromatography equipped with EC detector using temperature/pressure program (STM-APHA 1998; Yu and Cheng, 1999).



Fig. (1): schematic map showing the total governorate as a whole with specific notation on the main water purification plants in kafr El-Dawar. The study covered 17 villages. Sampling locations covered areas at high environmental pollution risk including, 1-inlet and outlet points of main municipal purification plant, 3 quick filtration units (QFU); 2-tapwater along distribution system network (kitchen taps, zeers, storage containers) and 3-tapwater collected from houses served by purification units inside the industrial complex.

Cross - media transfer

A contaminant concentration in a specific environmental medium can be expressed in another medium to which a person may be exposed using the following equation which was modified by Giardino et al., (1990):

$$Ca = \frac{Cw \cdot F \cdot Fi \cdot ti}{V}$$

Where, C_a is the concentration in bathroom air; C_w is the concentration of the contaminant in water expressed as $\mu\text{g. l}^{-1}$; V is the bathroom volume (9.00 m^3 , average); F is the shower flow rate (600 l. Hr^{-1}); F_i is the fraction of the contaminant volatilized (0.75), and t_i is the shower duration (15 min, average).

Exposure dose assessment

Exposure assessment involves calculation of contaminant concentration in different exposure pathways including ingestion, inhalation, and dermal absorption. The following assumptions of cancer risks were tailored based on EPA guidelines (USEPA, 1985, 1988, 1989, 1999 and 2002).

1. Inhalation

The following equation was used to calculate the THM₅ dose via inhalation pathway:

$$CDI_{inh} = \frac{Ca \cdot Ir \cdot Et \cdot Ed}{Bw \cdot At}$$

Where, C_a is the contaminant concentration in air during showering expressed as mg. m^{-3} ; I_r is breathing rate ($0.6 \text{ m}^3 \cdot \text{hr}^{-1}$ during showering); E_t is the exposure time expressed as hr. day^{-1} ; E_d is the exposure duration expressed in years, B_w is the person body weight (70 kg) and A_t is the averaging time ($365 \text{ day} \cdot \text{Year}^{-1}$).

2. Dermal

Dermal contact with a chemical in water during house activities was estimated as follows:

$$CDI_{dermal} = \frac{Cw \cdot Sa \cdot Et \cdot Ef \cdot Ed \cdot Cf}{Bw \cdot At}$$

Where, C_w is the chemical concentration in water expressed as mg. L^{-1} ; S_a is the skin surface area available for contact (0.86 m^2); E_t is the exposure time (consider local activity patterns is 2.6 hr. day^{-1}); E_f is the exposure frequency ($7 \text{ days. Year}^{-1}$); E_d is the exposure duration expressed in years and C_f is the volumetric conversion factor for water ($1 \text{ L. } 1000 \text{ cm}^3$).

3. Ingestion

Ingestion of chemicals through drinking water was estimated according to the following equation:

$$CDI_{ing} = \frac{Cw \cdot Ir \cdot Ef \cdot Ed}{Bw \cdot At}$$

Where, C_w is the chemical concentration in water expressed as mg. L^{-1} ; I_r is the ingestion rate (1.4 L. day^{-1} ; adult average); E_f is the exposure frequency expressed as day. Year^{-1} and E_d is the exposure duration expressed in years.

Risk Estimation

Estimations of cancer risks through ingestion route, dermal absorption and inhalation exposure depends on the availability of cancer slope factors and unit risk estimates which are provided on USEPA Website (USEPA, 2002; IRIS, 2005). In brief, cancer risk was estimated as follows:

$$\text{risk}_{\text{Cancer}} = \text{CDI} \times \text{S.F}$$

Where, S.F. is the slope factor of a specific cancer substance. In case of inhalation pathway, the risk was calculated based on function of unit risk expressed as ($\text{mg. kg}^{-1} \cdot \text{day}^{-1}$). Total exposure cancer risk was assumed to be the collective risk of exposure pathways. USEPA classified the chemical substances that cause cancer in human in a five main categories depending on their possibility of exerting carcinogenic effects.

On the other hand, non-carcinogenic risk was evaluated based on the reference doses (RfDs) and reference concentrations of contaminants. Thus the hazard quotient of THM₄ in each exposure pathway was calculated for non-carcinogenic risk assessment of ingestion route and dermal absorption as follows:

$$\text{HQ}_{\text{oral}} = \frac{\text{CDI}_{\text{oral}}}{\text{RfD}}$$

Where, RfD is the reference dose for specified substance (USEPA, 2002; IRIS, 2005). Hazard Index (HI) is the sum of more than one hazard quotient for multiple substances and/ or multiple exposure pathways.

Statistical analysis

The obtained data were expressed as mean \pm SE and statistically analyzed using ANOVA to determine the significant differences between treatments (Snedecor and Cochran, 1967).

RESULTS AND DISCUSSION

According to Human Development Report of Egypt (UNDP, 2006), the average life expectancy at birth is 71 year in Behira Governorate which matches with the value stated by USEPA. The average percentage of households with access to piped water and sanitation in Behira is 80.1 and 97.2; respectively compared with 91.3 and 93.6 in whole Egypt. But there was no accurate available data for us about household water consumption for either drinking or showering. Thus EPA consumption rates were used for exposure assessment through mathematical calculations.

THMs residues

Levels of THM₄ residues in collected potable water samples are exhibited in Table (1) and compared with both EMCL values and world health organization (WHO) guideline values (0.02, 0.06, 0.1 and 0.1 mg. l^{-1} for chloroform, BDCM, DBCM and bromoform; respectively) (WHO, 1993, 2004). In general THM₄ residue levels were higher than EMCL. The sum of examined compounds reached the highest value in drinking water (0.27 mg. l^{-1}) during winter while, the lowest value was recorded during autumn (0.05 mg. l^{-1}). During winter and spring seasons, chloroform was the major THM compound detected at high concentrations. Furthermore, estimated values in inhaled air showed that, chloroform and BDCM reached the high values during winter and spring seasons. Such increase in THM₄ levels particularly during winter may be justified where the rate of water consumption in winter is relatively lower than the other seasons, which in turn allow the chlorinated water to stay for longer periods in bad state and dis-repaired network pipelines permitting a chance for the organic matter naturally occurring in water in addition to that present in the pipelines to react with the chlorine in water. This issue is also emphasized through elevation in COD values accompanied with fluctuations in pH values during the defined seasons (Abdallah and Gaber, 2004). Additionally, decreasing of water temperature in pipelines may lower the THM compounds volatilization which consequently may lead to increase in its total concentration. On the other hand, bromo-THM₄ were present in lower concentrations than chloroform in most areas of the district which is consistent with other studies (Chang et al., 1996; Hsu et al., 2001). Also, TCE and 1, 1, 1-TCE were detected at low levels during all seasons.

Exposure dose assessment

Chronic daily intakes (CDIs) of THM through various exposure pathways were presented in Table (2). Inhalation and oral pathways seem to be the most absorbent than dermal pathway for most THM compounds. Chloroform was the major absorbed THM to account for 1.3×10^{-2} , 6.0×10^{-6} and $1.9 \times 10^{-2} \text{ mg. kg}^{-1} \cdot \text{day}^{-1}$, respectively at 50th percentile of probability through inhalation, dermal and ingestion exposure pathways. The lowest absorbed doses were 2.0×10^{-7} and $2.5 \times 10^{-7} \text{ mg.kg}^{-1} \cdot \text{day}^{-1}$ for TCE and 1,1,1-TCE in case of dermal absorption. Inhalation exposure occurs when the air breathed contains compounds volatilized during water usage. Showering has been identified as the activity contributing the greatest amount to inhalation exposure to volatile compounds (Wilkes et al., 1992). Due to its property of a lower boiling point, chloroform is assumed to be the major compound to which people are exposed during showering and bathing. Comparatively, the tolerable daily intake (TDI) of chloroform was calculated by Fawell (2000) to be $0.02 \text{ mg.kg}^{-1} \cdot \text{day}^{-1}$ in drinking water.

Table (1): Average of seasonal residue levels of THMs in drinking water samples and estimated concentrations in inhaled air bathroom in the study area of Kafr El-Dawar district during 2002.

Compound	Winter	Spring	Summer	Autumn	N	mean
1-Drinking water (mg.l⁻¹)						
Chloroform	0.24± 4.02	0.13 ± 2.92	0.043 ± 1.69	0.018± 1.10	32	0.109± 5.99
BDCM	0.008 ± 0.71	0.007 ± 6.8	0.011 ± 0.85	0.009 ± 0.79	32	0.009 ± 1.71
DBCM	0.015 ± 0.99	0.015 ± 1.01	0.019 ± 1.15	0.018± 1.09	32	0.017± 2.36
Total	0.263	0.152	0.073	0.045		0.135
EMCL	0.100	0.100	0.100	0.100		0.100
TCE	0.001 ± 0.39	0.016 ± 0.32	0.001 ± 0.25	0.00	32	0.0011 ± 0.55
1,1,1-TCE	0.001 ± 0.08	0.015± 0.32	0.002± 0.29	0.00	32	0.001 ± 0.57
LSD _{0.05}	11.49	0.52	0.57	0.31		0.59
2-Inhaled air (mg.m⁻³)						
Chloroform	3.04 ± 14.21	1.71 ± 5.99	0.60 ± 5.99	0.23 ± 3.88	32	1.40± 2.13
BDCM	0.093± 2.50	0.086± 2.41	0.136 ± 3.02	0.118 ± 2.80	32	0.11 ± 6.00
DBCM	0.187 ± 3.53	0.191± 3.58	0.26 ± 4.07	0.22± 3.87	32	0.22 ± 7.54
Total	3.32	1.98	0.99	0.568		1.73
TCE	0.024 ± 1.21	0.019 ± 1.14	0.011 ± 0.87	0.00	32	0.014± 2.13
1,1,1-TCE	0.015 ± 1.00	0.019 ± 1.12	0.016 ± 1.02	0.00	32	0.012 ± 2.03
LSD _{0.05}	22.98	1.04	1.14	0.61	-	0.58

Each value is the mean ± SE.

DBCM: Dibromochloromethane, BDCM: Bromodichloromethane, EMCL: Egypt. Maximum contaminant level, TCE: Tetrachloroethane, 1,1,1-TCE: 1,1,1-trichloroethane, and N: number of samples.

Table (2): Chronic daily intake (CDI) of THMs through various exposure pathways.

Compound	Absorbed dose (mg.kg ⁻¹ .day ⁻¹)/ exposure pathway					
	Inhalation		Dermal absorption		Oral ingestion	
	50 th	90 th	50 th	90 th	50 th	90 th
Chloroform	0.013	0.071	6.0x10 ⁻⁶	2.0x10 ⁻⁵	0.019	0.093
BDCM	1.0x10 ⁻³	5.6x10 ⁻³	4.9x10 ⁻⁷	1.7x10 ⁻⁶	1.7x10 ⁻³	7.4x10 ⁻³
DBCM	2.0x10 ⁻³	0.011	9.4x10 ⁻⁷	3.2x10 ⁻⁶	3.0x10 ⁻³	0.014
TCE	1.4x10 ⁻⁴	7.0x10 ⁻⁴	6.0x10 ⁻⁸	2.0x10 ⁻⁷	1.9x10 ⁻⁴	9.0x10 ⁻⁴
1,1,1-TCE	1.9x10 ⁻⁴	6.2x10 ⁻⁴	5.7x10 ⁻⁸	2.5x10 ⁻⁷	1.8x10 ⁻⁴	8.6x10 ⁻⁴
Σ CDI	0.016	0.089	7.6x10 ⁻⁶	2.6x10 ⁻⁵	0.024	0.116

Carcinogenic risk effect

The slope factors of the four contaminants that are associated with lifetime cancer risk for the exposed individuals are shown in Table (3). Estimated values of cancer and non-cancer risks attributable to exposure to each THM component in drinking water and inhaled air are presented in Table (4). Chloroform was the major THM induced cancer risk effect (1.1x10⁻⁴) in drinking water *via* ingestion route of exposure, 1.1x10⁻³ and 3.6x10⁻⁸ in case of inhalation and dermal exposures; respectively at 50th percentile. Σ risk of exposure pathways reach the highest value 8.0x10⁻³ at 90th percentile. The checked compounds were classified according to their weight of evidences as B₂ for chloroform and BDCM and C for DBCM. These estimates represent theoretical excess cancer risk higher than 10⁻⁶, the demonisms or negligible risk level defined by the USEPA.

There is a lack of information concerning exposure and risk associated with THM_s in drinking water in Egypt. Worldwide, few studies have been measured THM_s in drinking water and estimated the health risks through ingestion route (Clayton et al., 1999; Hsu et al., 2001; Sofuaglu et al., 2003; Lee et al., 2004 and Kavcar et al., 2006). The highest estimated risk values were 1.8x10⁻⁴ in Taiwan (Hsu et al., 2001) and 2.1x10⁻⁷ in Arizona (Sofuaglu et al., 2003) for chloroform and 6.82 x10⁻⁵ in Hong Kong (Lee et al., 2004) for BDCM. Epidemiological studies showed an association between breast cancer risk and chlorinated by-products in the Finnish investigation (Koivusalo and Vartiainen, 1997), bladder cancer (King and Marrett, 1996) and spontaneous abortion, low birth weight and defects (Mills et al., 1998). Also, consumption of drinking water with high THM content may increase the risk of melanoma and

possibly of hormone-dependent cancers such as neoplasm of the prostate, breast and the ovary (Vinceti et al., 2004). Additionally, increased risk of chronic myeloid leukemia was associated with

increasing years of exposure to different chlorination disinfection by-product indexes, with an adjusted odds ratio of 1.72 for the highest exposure duration to total THM of more than 40 µg. l⁻¹ (Kasim et al., 2006).

Table (3): Slope factors (SF), unit risk values and reference doses (RfD) for THMs (USEPA, 1999).

Compound	SF oral (mg.kg ⁻¹ .day ⁻¹) ⁻¹	Unit risk inhalation (mg.kg ⁻¹ .day ⁻¹) ⁻¹	RfD (mg.kg ⁻¹ .day ⁻¹)
Chloroform	6.1x10 ⁻³	1.7x10 ⁻⁷	1x10 ⁻²
BDCM	6.2x10 ⁻²	1.8x10 ⁻⁶	2x10 ⁻²
DBCM	8.4x10 ⁻²	2.4x10 ⁻⁶	2x10 ⁻²

Table (4): Cancer and non-cancer risk estimates for THMs in different exposure pathways.

Compound	Weight of evidence	Cancer risk value		HQ	
		50 th	90 th	50 th	90 th
1- Oral ingestion					
Chloroform	B2	1.1x10 ⁻⁴	5.6x10 ⁻⁴	1.9	9.3
BDCM	B2	1.1x10 ⁻⁴	4.7x10 ⁻⁴	0.09	0.40
DBCM	C	2.5x10 ⁻⁴	1.2x10 ⁻³	0.15	0.70
Σ risk		4.7x10 ⁻⁴	2.2x10 ⁻³	2.14	10.4
2-Dermal absorption					
Chloroform	B2	3.6x10 ⁻⁸	1.2x10 ⁻⁷	6.0x10 ⁻⁴	2.0x10 ⁻³
BDCM	B2	3.1x10 ⁻⁸	1.1x10 ⁻⁷	2.5x10 ⁻⁵	8.5x10 ⁻⁵
DBCM	C	8.0x10 ⁻⁸	2.8x10 ⁻⁷	4.7x10 ⁻⁵	1.6x10 ⁻⁴
Σ risk		1.5x10 ⁻⁷	5.1x10 ⁻⁷	6.7x10 ⁻⁴	2.3x10 ⁻³
3-Inhalation					
Chloroform	B2	1.1x10 ⁻³	5.8x10 ⁻³	ND	ND
BDCM	B2	1.8x10 ⁻⁹	1.0x10 ⁻⁸	ND	ND
DBCM	C	4.8x10 ⁻⁹	2.6x10 ⁻⁸	ND	ND
Σ risk		1.1x10 ⁻³	5.8x10 ⁻³	ND	ND
Total risk		1.6x10 ⁻³	8.0x10 ⁻³	2.14	10.4

ND: not determined due to lack of RfC values for inhalation studies.

Non-carcinogenic risk effect

The hazard indexes of THM_s of different exposure routes were also presented in Table (4). Hazard quotient and hazard index that exceed 1.0 for a single or combinations of chemicals or exposure routes indicate the possibility of non-cancer toxic risks from the exposure. HQ was found to be of a high value for chloroform (9.3) at 90th percentile through oral ingestion exposure pathway indicating a great potential toxic effect. The sum of hazard indices did not exceed 10.4 for all checked chemicals at 90th percentile.

Toxicological studies revealed that exposure to mixture of THM_s led to higher blood levels of unchanged THM_s in animals, which may induce risks at low concentrations of THM mixtures (DaSilva et al., 1999). Exposure to multiple toxicants in a mixture may result in additive and/or interactive effects, that could be synergistic or antagonistic (Hsu et al., 2001). Long-term exposure to these byproducts increases the risk of cancer and creates problems for

liver, kidney, gastrointestinal and urinary tracts and central nervous system (USEPA, 2001) in addition to reproductive and developmental effects that are warranting further investigations..

Uncertainty analysis

Uncertainties exist in the risk assessment of exposure. These include uncertainties in measurement (Fritz and Schenk, 1987), in values assigned to population exposure variables (Wallace, 1991) and the uncertainties introduced in risk characterization due to day-to day, place to place variations in concentrations (Kim et al., 2002). Even in well-designed and well-conducted analytical studies, relatively poor exposure assessments were conducted. A major uncertainty surrounds the interpretation of the observed associations, as exposure to relatively few water contaminants has been considered. With the current data, it is difficult to evaluate how unmeasured DBPs or other water contaminants may have affected the observed relative risk estimates.

CONCLUSION

Our data highlights how much risk imposed on the consumers of such water and provides an evidence that tap water THM concentration is a reliable predictor of accrual exposure to these disinfection by-products, which occurs *via* ingestion and even more so through inhalation and dermal exposure. Association between THMs exposure through these pathways and lifetime cancer risk disclosed that the total cancer risk from inhaled air (5.8×10^{-3}) was higher than those from ingested water (2.2×10^{-3}). Non-carcinogenic risk assessment for THM, was calculated as well. The highest value of HQ was 9.3 for chloroform. Also, chloroform levels exceeded its reference dose indicating a great potential toxic effect.

The risk estimation only not enough for inducing cancer from exposure to THMs, but more toxicological studies must done to confirm its.

According to the above results, quality of drinking water in the study area is considered in a partial accordance with WHO guidelines for drinking water. To obtain better quality, DBP, can be controlled through removal of DBP precursors at the first place, using granular activated carbon, and membrane/ozone-bio-filtration which can all remove organic matter.

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REFERENCES

- Abdallah, S. M. 2002. Assessment of chemical contamination of drinking water in Alexandria and Kafr El-Dawar". WHO/EMRO. Final technical report, File # R6/18/3.
- Abdallah, S. M., Gaber, H. M., 2004. Occurrence of disinfection by-products in drinking water of some rural areas. *Alex. J. pharm. Sci.* 18 (1), 3-11.
- Bellar, T. A., Lichtenberg, J. J., Kroner, R. C., 1974. The occurrence of organohalids in chlorinated drinking waters. *J. Am. Water Works Assoc.* 66 (12), 703-706.
- Bull, R. J., Birnbaum, L. S., Cantor, K. P., Rose, J. B., Butterworth, B. E., Pergram, R., Tuomisto, J., 1995. Water chlorination: essential process or cancer hazard. *Fund. Appl. Toxicol.* 28, 155-166.
- Cantor, K. P., Lynch, C. F., Hildesheim, M. E., Dosemeci, M., Lubin, J., Alavanja, M., Craun, G., 1998. Drinking water source and chlorination by-products. 1. Risk of bladder cancer. *Epidemiology* 9, 21-28.
- Chang, E. E., Chao, S. H., Chiang, P. C., Lee, J. E., 1996. Effects of chlorination on THMs formation in raw water. *Toxicol. Environ. Chem.* 56, 211-225.
- Clayton, C. A., Pellizzari, E. D., Whitmore, R. W., Perritt, R. L., Quackenboss, J. J., 1999. National human exposure assessment survey (NHEXAS): distributions and associations of lead, arsenic and volatile organic compounds in EPA Region 5. *J. Exposure Anal. Environ. Epidemiol.* 9, 381-392.
- DaSilva, M. L., CharestTradif, G., Krishnan, K., Tardif, R. 1999. Influence of oral administration of quaternary mixture of trihalomethanes on their blood kinetics in the rat. *Toxicol. Lett.* 106, 49-57.
- Fawell, J., 2000. Risk assessment case study-chloroform and related substances. *Food Chem. Toxicol.* 38, 91-95.
- Fritz, J. S., Schenk, G. H., 1987. *Quantitative Analytical Chemistry.* Prentice-Hall, Englewood Cliffs, NJ, USA.
- Gallagher, M. D., Nuckols, J. R., Stallones, L., Savitz, D. A., 1998. Exposure to trihalomethanes and adverse pregnancy outcomes. *Epidemiology* 9 (5), 484-489.
- Giardino, N., Gumerman, E., Andelman, J., Wilkes, C., Small, M., Borraro, J., C. Davidson 1990. Real-Time Measurements of Trichloroethylene in Domestic Bathrooms using contaminated water. Pre- print indoor Air 90 conference; Toronto.
- Hsu, C. H., Jeng, W. L., Chang, R. M., Chein, L. C., Han, B. C., 2001. Estimation of potential lifetime cancer risks for trihalomethanes from consuming chlorinated drinking water in Taiwan. *Environ. Res. Section A*, 85, 77-82.
- IRIS, 2005. Integrated Risk Information System, US Environmental Protection Agency/ Cincinnati, OH. Accessed at: <<http://www.epa.gov/iris>>.
- Kasim, K., Levallois, P., Johnson, K. C., Abdous, B., Auger, P., 2006. Chlorination disinfection by-products in drinking water and the risk of adult leukemia in Canada. *Am. J. Epidemiol.* 163, 116-126.
- Kavcar, P., Odabasi, M., Kitis, M., Inal, F., Sofuoglu, S. C., 2006. Occurrence, oral exposure and risk assessment of volatile organic compounds in drinking water for Izmir. *Water Res.* 40, 3219-3230.
- Kim, Y. M., Harrad, S., Harrison, R. M., 2002. Levels and sources of personal inhalation exposure to volatile organic compounds. *Environ. Sci. Technol.* 36, 5405-5410.
- King, W. D., Marrett, L.D., 1996. Case - control study of bladder cancer and chlorination by products in treated water (Ontario, Canada). *Cancer causes* 1, 596 - 604.
- Koivusalo, M., Varitainen, T., 1997. Drinking water chlorination by-products and cancer. *Rev. Environ. Health* 87, 1168-1176.

- Kramer, M. D., Lynch, C. F., Isacson, P., Hanson, J. W., 1992. The association of waterborne chloroform with intrauterine growth retardation. *Epidemiology* 3, 407-413.
- Lee, S. C., Guo, H., Lam, S. M. J., Lau, S. L. A., 2004. Multipathway risk assessment on disinfection by-products of drinking water in Hong Kong. *Environ. Res.* 94, 47-56.
- Mills, C. J., Bull, R. J., Cantor, K. P., Reif, J., Hrudey, S. E., Huston, P., 1998. Health risks of drinking water chlorination by-products: report of an expert working group *Chronic Diseases in Canada* 19 (3), 91-102.
- Morris, R. D., 1995. Drinking water and cancer. *Environ. Health Perspect.* 103 (Suppl. 8), 225-231.
- Pilotto, L. S., 1995. Disinfection of drinking-water, disinfection by-products and cancer-what about Australia. *Aust. J. Pub. Health* 19 (1), 89-93.
- Snedecor, G. V., Cochran, W. C. 1967. *Statistical Methods* 6th Ed., Iowa state Univ. USA.
- Sofuoglu, C. S., Lebowitz, M. D., O'Rourke, M. K., Robertson, G. L., Dellarco, M., Moschandreas, D. J., 2003. Exposure and risk estimates for Arizona drinking water. *J. Am. Water Works Assoc.* 95 (7), 67-79.
- STM-APHA 1998. *Standard Methods for Examining of Water and Wastewater*. 20th edition, APHA, Washington D.C., USA.
- UNDP, 2006. *The Egypt Human Development Report*. United Nations Development Program, Institute of National Planning, Project Document EGY/01/006.
- USEPA, 1985. *Development of statistical distributions or ranges of standard factors used in exposure assessments*. Office of health and environmental assessment.
- USEPA, 1988. *Proposed guidelines for exposure-related measurements*. 53 Federal register 48830.
- USEPA, 1989. *Exposure factors handbook*. Office of health and environmental assessment. EPA /600/8 - 89/043.
- USEPA, 1999. *Guidelines for Carcinogen Risk Assessment*. Risk Assessment Forum, U.S. Environmental Protection Agency, Washington DC. NCEA- F-0644 (Revised draft).
- USEPA, 2001. *Integrated Risk Information System (IRIS): chloroform*. U.S. Environmental Protection Agency/ Cincinnati, OH. Accessed at: <<http://www.epa.gov/iris>>.
- USEPA, 2002. *Integrated Risk Information System (Electronic data base)*. U.S. Environmental Protection Agency, Washington DC. Available on line: <http://www.epa.gov/iris>.
- Vinceti, M., Fantuzzi, G., Monici, L., Cassinadri, M., Predieri, G., Aggazzotti, G., 2004. A retrospective cohort study of trihalomethane exposure through drinking water and cancer mortality in northern Italy. *Sci. Total Environ.* 330, 47-53.
- Wallace, L. A., 1991. Comparison of risks from outdoor and indoor exposure to toxic chemicals. *Environ. Health Perspect.* 95, 7-13.
- Waller, K., Swan, S. H., DeLorenze, G., Hopkins, B., 1998. Trihalomethanes in drinking water and spontaneous abortion. *Epidemiology* 9, 134-140.
- WHO, 1993. *World Health Organization Guidelines for Drinking water quality*. 2nd ed. Vol. 1, recommendations, WHO, Geneva.
- WHO, 2004. *Guidelines for drinking water quality* 3rd ed. Vol. 1, Recommendation, WHO, Geneva.
- Wilkes, C., Small, M., Andelman, J., Giardino, N., Marshall, J., 1992. Inhalation exposure model for volatile chemical from indoor uses of water. *Atmos. Environ.* 26a, 2227.
- Yang, C. Y., Cheng, B. H., Tsai, S. S., Wu, T. N., Lin, M. C., Lin, K. C., 2000. Association between chlorination of drinking water and adverse pregnancy outcome in Taiwan. *Environ. Health Perspect.* 108 (8), 765-768.
- Yang, C. Y., Chiu, H. F., Cheng, M. F., Tsai, S. S., 1998. Chlorination of drinking water and cancer mortality in Taiwan. *Environ. Res.* 78, 1-6.
- Yu, J. C., Cheng, L. N., 1999. Speciation and distribution of trihalomethanes in drinking water of Hong Kong. *Environ. Int.* 25 (5), 605-611.

الملخص العربي

تقييم مخاطر لتعرض لمركبات الترياهالوميثان في مياه الشرب في المناطق الريفية
بمحافظة البحيرة-مصر

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تعد المخاطر الصحية للإنسان التي قد تنتج عن التعرض لنواتج التفاعل الثانوية لعملية كلورة مياه الشرب من الأهمية استنادا الى وجود دلائل وبائية وسمية تفيد إمكانية حدوث تأثيرات صحية عكسية. وقد ظهرت الحاجة لاجراء تقييم لمخاطر مركبات الترياهالوميثان على الأخص كأمرا ضروريا للتأكد من سلامة مصادر مياه الشرب. وبناء على هذا فقد تم بحث المخاطر المحتملة والتي قد تصاحب التعرض لمركبات الترياهالوميثان في مياه الشرب في منطقة كفر الدوار كاحدى المناطق الريفية. وقد أظهرت النتائج أن المستوى للكلية لمركبات الترياهالوميثان كان أعلى من الحد الأقصى المصرى للملوثة وقد وصل الى أعلى مستوي له خلال شتاء ٢٠٠٢ (٢٦٧ و٢٦٤ ميكروجرام/لتر). وكان للكلورفورم هو أكثر مركبات الترياهالوميثان تواجدا خاصة خلال فصلى الشتاء والربيع. وقد سجل المقدار للكلية للدائم المأخوذ يوميا لخليط الترياهالوميثان من خلال طرق التعرض المحتملة (الاستنشاق- للتلامس- البلع) للقيم الآتية ٠.٠١٦ و ٧.٦ × ١٠^{-٦} و ٠.٠٢٤ مجم/كجم/يوم على التوالي عند احتمالات ٥٠%. ووصلت القيمة المقدرة لخطر الإصابة بالسرطان الى أعلى قيمة لها ٥.٨ × ١٠^{-٦} نتيجة التعرض للكلورفورم عن طريق الاستنشاق عند احتمالات ٩٠%. وقد صنفت مركبات الترياهالوميثان وفقا للدلائل المقاسة الى B2 لكل من الكلورفورم و BDCM و C لمركب DBCM. كما سجلت قيمة محصلة الخطر (HQ) أعلى قيمة لمركب الكلورفورم (٩.٣) عن طريق البلع عند احتمالات ٩٠%. وسجل مجموع مؤشرات الخطر قيمة ١٠.٤ للمركبات الكلورفورم و BDCM و DBCM متجاوزا الجرعات القياسية.

وأجمالا فإن النتائج المتحصل عليها يمكن أن تفيد إمكانية حدوث تأثيرات جهازية بالإضافة الى/ أو سرطانة كنتيجة للتعرض لمركبات الترياهالوميثان من خلال طرق التعرض المختلفة.