

## Research Article

# Cancer Risks from Disinfection Byproducts of Drinking Water: The Neglected Issue in the Global South

Ambelu A<sup>1\*</sup>, Fikre T<sup>2</sup> and Mekonen S<sup>1</sup><sup>1</sup>Jimma University, College of Public Health, Department of Environmental Health Science and Technology, Jimma, Ethiopia<sup>2</sup>Gambella Regional Health Bureau, Gambella, Ethiopia**\*Corresponding author:** Argaw Ambelu, Gambella Regional Health Bureau, Gambella, Ethiopia**Received:** September 29, 2017; **Accepted:** October 31, 2017; **Published:** November 07, 2017**Abstract**

Surface water treated with chlorine is known to have undesirable disinfection by-products. Many disinfection by-products such as chloroform are known to cause chronic illness such as cancer. In most sub-Saharan Africa, bacteriological treatment of drinking water is much more emphasized than the risk of disinfection by-products. The purpose of the present study was to determine chloroform concentration from municipal water distribution system of surface and groundwater sources. Water samples were collected from municipal water distribution systems of Jimma and Agaro town before and after chlorination. Chloroform concentration was determined using gas chromatography with electron capture detector (GC-ECD). In addition, household survey was conducted to determine the water consumption, bathing habit, and body weight of the consumers. Human exposure and risk assessment was done using USEPA exposure estimation method. From the findings, the mean concentrations of chloroform were  $93.75\mu\text{g/L} \pm 77.19$  and  $4.67\mu\text{g/L} \pm 5.33$  in Jimma and Agaro town water sources, respectively. About 50% of the water samples collected from Jimma town was greater than the United States Environmental Protection Agency (USEPA) maximum concentration level (MCL) for trihalomethanes (THMs). Chlorine dose, pH, and residence time predicts the occurrence of chloroform. The cancer risk from chloroform exposure via ingestion and inhalation was greater than World Health Organization (WHO) acceptable cancer risk value. Hence, attention is required to the disinfection by-products to safeguard consumers' health.

**Keywords:** Chloroform; Cancer Risk; Exposure Assessment; Surface and Groundwater**Introduction**

Access to safe drinking water is essential to human health and a component of effective policy for health protection [1]. To provide potable water for drinking, food preparation and recreation, destruction of pathogenic microorganisms is an important issue of concern. Due to this needs, the water is commonly treated by the use of reactive chemical agents such as chlorine for the safety of consumers [1]. However, chlorine can easily react with natural organic matter (NOM) present in surface water which results in the production of disinfection byproducts (DBPs) [2,3]. Trihalomethanes (THMs) are the primary disinfection byproduct where the major are chloroform, bromodichloromethane, dibromochloromethane, and bromoform [4].

Chloroform is the most prevalent among THM compounds in chlorinated water and has been classified as possibly carcinogenic substance to humans, based on sufficient evidence from experimental animals [5]. Since early 1974 up to present, many studies were carried out to evaluate health impact of chloroform using laboratory animals. Some studies revealed that, chloroform has damaged different cells in the body and has risk on cell mutation and develops cancer in exposed organs [6–9]. Even though there is a difficulty for direct evaluations of the effects of THM on human subjects, there are studies which indicated, THMs are known to increase risk of bladder cancer, intestine, anal, esophagus, and some reproductive health impacts like

abortion, low birth weight [10–12].

The concentration of THMs vary based on the level of Organic Matter (OM), ultraviolet absorption, pH, temperature, chlorine or bromine dosage and residence time [12–17]. Surface water sources in sub-Saharan African countries are rich in OM, which can easily exposed to direct sunlight and often with elevated water temperature. On the other side, the water supply system in many cities of such countries is abstracted from surface water source such as rivers, ponds, lakes, canals, etc., which is subsequently treated with chlorine. This apparently makes the water to have elevated concentration of THM and exposure of consumers to these chemicals. As the result many countries promulgated guidelines to control DBPs [18] (Figure 1). Special concerns are associated with the THMs, because they have been recognized as potentially hazardous and are the major by-products of chlorination [19].

Regular monitoring of DBPs in the public water supply system of some sub-Saharan African countries is hardly available. However, there is no information available on the nature, distribution and typical concentrations of DBPs in Ethiopian and other sub-Sahara African countries. Unlike the attention given to microbial and physical contamination, level of DBPs in municipal water supply system is missing, the level of consumers' exposure and risks is not known. Therefore, the main aim of this study is to investigate the level of DBPs in particular to chloroform in the water distribution systems

of Agaro and Jimma southwestern Ethiopia, and to undertake consumer cancer risk assessment.

## Materials and Methods

### Study area

Jimma is situated in the southwestern Ethiopia at a distance of 352km far from Addis Ababa. The existing water distribution network covers an area of approximately 31km<sup>2</sup> and the source is surface water. The distribution system uses conventional water treatment such as coagulation, sedimentation and filtration and addition of free chlorine for disinfection [20]. While in the second study area which is Agaro there is no water treatment system except chlorination before distribution [21].

### Sampling

To determine the concentration of chloroform and its precursors, fresh water samples were collected from the treatment plant and distribution taps. The analytical procedures for collection and analyses of water samples were made according to USEPA Method 551.1 of 1995 [22]. Duplicated raw and treated water samples were collected in 125ml glass bottles. The glass bottles were previously washed with phosphate free detergents and tap water, then rinsed thoroughly with distilled water and allowed to dry at room temperature and then placed in an oven at 400°C for 30 minutes. Before sampling, 1.5mg of ascorbic acid was added to bottles to eliminate any residual chlorine and to stop additional chloroform formation. The tap water were allowed to slowly flow about 5 minutes before sampling and then the bottles were filled just to overflowing without passing air bubbles through the sample. The samples were stored between 0°C to 4°C during transportation from field to the laboratory.

Samples for Total Organic Carbon (TOC) and UV-absorbance measurements were collected in 125ml brown glass bottles which were adequately washed in a similar way with the above procedures. Once collected, samples were carefully stored in the dark below 4°C and were transported to Addis Ababa, JEJE Labo Analytical Testing Laboratory for analysis. To evaluate the water consumption rate, bathing habits and their body weight 768 individuals (384 in each town) were interviewed and the data were used for human exposure assessment.

### Analytical procedures

The pH and temperature of the water samples were determined using a pH meter and digital Thermometer, respectively within an hour time following the sample collection. Free and total chlorine residual, for each chlorinated samples was determined using HACH, CN-66 model free or total chlorine test kit using colorimetric DPD. The information on residence time and distance from treatment plant to sampling sites were obtained from water supply offices of each town. After samples were filtered at 45µm, UV absorbance was measured by UV/visible spectrophotometry of DR 5000 Hach model at 254nm with 5mm optical path quartz cells. TOC was analyzed using a Shimadzu TOC analyzer (Shimadzu TOC 5000) following method 10129.

For the determination of chloroform concentration a 10mL of water was taken and poured in 15mL glass bottle and 1.5g anhydrous sodium sulphate and 2mL n-hexane was added as an extraction solvent

and shaken by hand for 4 minutes and left undisturbed for 2 minutes. Then 2µL the extracted sample was injected into an Agilent 7890A Gas Chromatography system equipped with an Electron Capture Detector (ECD) for the quantitative determination of chloroform.

### Chromatographic condition

The gas chromatographic separation was achieved on a capillary column HP-5 (30m length x 0.32mm internal diameter (I.D) and 0.25µm film thickness). The oven temperature was kept at 80°C for 15 minutes. The temperature of the injector and detector were set at 200°C and 250°C respectively. The extraction procedures were undertaken at room temperature. Nitrogen gas (99.99% pure) was used as both carrier and make up gas with constant pressure of 10psi and a flow rate of 1mL/min.

### Exposure assessment

Ingestion of water is one of exposure pathway to THM among many, such as inhalation and dermal contact during bathing, swimming, dishwashing and clothes washing [23]. In current study, population exposure to chloroform via ingestion was estimated using chronic daily intake (CDI) estimation method as described in USEPA exposure estimation method [24]. Parameters like body weight, age and amount of daily water intake for adults were obtained from the survey and used in the following equation.

$$CDI_{\text{Ingestion}} = Cw * (IRw/BW) * (EF * ED)/AT \quad (1)$$

where;  $CDI_{\text{Ingestion}}$  = Chronic daily intake through oral ingestion exposure (mg/kg-day), Cw (mg/L) = Concentration of chloroform in drinking water of the study site (mg/L), IRw (L/day) = Ingestion rate of drinking water, 2L for adults (USEPA, 2005), BW (kg) = body weight; 70 for adults [25], EF (days/year) = exposure frequency; 365 days/year [24],

ED (years) = exposure duration; for average of 70 years [25] but life expectancy of Ethiopians were taken as 54 and 59 years for males and females respectively [27],

AT (days) =  $ED \times 365 \text{ days/year}$  [24] and absorptivity of body is assumed to be 100% [23].

### Cancer risk estimation

The target cancer risk estimation model is adopted from the method used by Ching-Hung Hsu and his colleagues in 2000 for estimation of Potential lifetime cancer risks for THMs from Consuming chlorinated drinking water in Taiwan. For carcinogenic effects THMs, risk is expressed as excess probability of contacting cancer over a lifetime (70 years). Because contact rates with tap water for children and adults are different, cancer risks during the 1st 30 years of life were calculated using age-adjusted factors. The model considers mainly ingestion and inhalation route for cancer risk estimation. In the current study, we used some parameters depending to our countries situation and we compared with adopted model standards. The model for estimating target cancer risks (lifetime cancer risks) is presented below in equation 2. Additionally, cancer risk is also predicted for ingestion exposure alone by multiplying CDI via ingestion with its carcinogenic slope factor of  $6.10 \times 10^{-3}$  [26].

$$TR = Cc * EFr * [(K * IFAadj * CPSi) + (IFWadj * CPSo)] / ATc * 1000 \mu\text{g}/\text{mg}$$

**Table 1:** The recovery concentration analyzed by using n-hexane as extraction solvent.

Analyte	Fortification Conc. (µg/L) (n=7)	Mean conc. (µg/L) (n=7)	% RSD	% Recovery
Chloroform	150	153.32	9.52	102.21
	1.5	1.46	6.5	97.33

and

$$IFWadj (L^* year/ Kg^* day) = (EDc^* IRWc)/BWc + ((EDtot-EDc)^*IRWa)/BWA \tag{2}$$

where:

Cc is contaminants in water (µg/L), TR is target cancer risk, CPSO is carcinogenic potency slope oral (risk per mg/kg/day) = 6.10x10<sup>-3</sup>,

CPSi is carcinogenic potency slope inhaled (risk per mg/kg/day), 3.05x10<sup>-2</sup>, BWa is body weight, adult (70kg); in our case we obtained from survey, BWc is body weight for age1-6 years is 15kg, ATc is averaging time carcinogens (25,550 days), in our case, 54 years (19710 days) for males and 59 years (21535 days) for females (according to CSA, 2007 [27]; life expectancy in Ethiopia is 54 and 59 years for males and females respectively); IFAadj is inhalation factor, age-adjusted (11.66m<sup>3</sup>-years/kg-days), IRWa is tap water ingestion, adult (2L/day) ( in our case we obtained from survey), IRWc is tap water ingestion of children age 1-6 years (1L/day), IFWadj is tap water ingestion factor, age-adjusted (1.09L-years/kg-days), EFR is exposure frequency (365 days/year), EDtot is exposure duration total (30 years), EDc is exposure duration, age 1-6 years (6 years) and K is volatilization factor (0.5L/m<sup>3</sup>).

**Quality control**

All the procedures were undertaken per standards to keep quality of procedure to get reliable result. Laboratory reagent blanks (LRB) was analyzed before processing any samples. In addition, each time when a set of samples were extracted or reagents were changed, a LRB was analyzed. Each new bottle of solvents was analyzed for interferences before use. For calibration, chloroform standard of 99.99% minimum assay was purchased from Central Drug House, New Delhi, Ltd, India. Standard solutions of chloroform were prepared in methanol as it described in USEPA methods 551. From this solution different serial dilutions were prepared on methanol. Thus, two sets of five level concentrations ranging from 0.5 to 10µg/L and 25 to 400µg/L were prepared for calibration of GC-ECD of chloroform analysis. The r<sup>2</sup> values were 0.9984 and 0.9990 for lower and higher concentrations respectively. For the reliability of this

calibration continuing calibration verification was used in which 175µg/L chloroform was injected and analyzed to be 173.5µg/L (99.14% recovery). Minimum detection limit was estimated and found to be 0.16µg/L. As indicated in Table 1, the mean percent recoveries and repeatability (%RSD) were in the accepted analytical range (% recover= 70-120 and %RSD<20) [28].

**Results**

Among the participants, 73(60.8%) and 57(47.5%) were males and mean ages were 36.37 ± 11.02 and 35.86 ± 11.1 years in Jimma and Agaro towns, respectively. About 52(43.3%) and 77(64.2%) of study participants reported as they take shower twice per week in Jimma and Agaro town respectively. The mean duration of time to take shower was 14.4 and 13.9 minutes for Jimma town and Agaro towns respectively.

As indicated in Table 2, the mean concentrations of Chloroform were 93.75µg/L and 4.67µg/L in Jimma and Agaro towns respectively. The highest concentration of chloroform is observed in water distribution system of Jimma. While for TOC the mean concentration were 15.04mg/L and 13.88mg/L respectively, in Jimma and Agaro towns.

Using bivariate correlation, there is statistically significant strong positive correlation between chloroform concentration and pH, Temperature and distance of the water point from the treatment plant, and residential time with their respective r<sup>2</sup> and p-values of 0.949, 0.98 (p<0.000, <0.000), 0.672, 0.92 (p<0.009, <0.000), 0.694, 0.91 (p<0.006, <0.000) and 0.764, 0.95 (p<0.001, <0.000) in Jimma and Agaro town respectively. In Jimma town, when pH and residential time controlled in partial correlation, there is strong positive correlation between chloroform concentration and TOC and UV absorbance with r<sup>2</sup> values of 0.86 (p<0.000) and 0.58 (p<0.048), respectively. For Agaro town water supply system, there was positive correlation between chloroform and TOC and UV absorbance at 254nm when controlled for residence time r<sup>2</sup> values of 0.73(p<0.025) and 0.77 (p<0.015) for TOC and UV absorbance respectively. Over all, the chloroform concentration of water samples from Jimma town treatment plant was significantly different from the Agaro town (Figure 2).

Multiple regression tests revealed that chloroform concentration in the distribution systems were determined by chlorine dose, pH and residence time with adjusted r<sup>2</sup> value of 0.867. The model fit between predicted and observed values is presented on Figure 3.

Chronic daily intake (CDI) of chloroform via ingestion route

**Table 2:** Summary statistic chloroform concentration and water quality parameters.

Source	Summary Statistics	Chloroform (µg/L)	Temp. (°C)	pH	TOC (mg/L)	UV abs	R time	Distance	Res. Chlorine
Jimma town	Min	0	18	5.81	6.36	0.1	0	0	0
	Max	220.32	22.1	7.4	15.03	1.85	6	7.23	1.5
	Mean	93.75	19.34	6.76	9.41	0.33	2.44	4.25	0.33
	Stdev	77.19	1.33	0.58	2.82	0.49	1.76	2.6	0.51
Agaro town	Min	0	20.8	6.67	2.31	0.016	0	0	0
	Max	17.33	23.4	7.75	13.88	0.372	4	7.17	0
	Mean	4.67	22	7.21	9.14	0.133	1.52	2.69	-
	SDev	5.33	0.85	0.35	3.64	0.114	1.37	2.81	-

Temp. = Temperature, TOC=Total Organic Carbon, UV abs = UV absorbance, R time=Residence time and Res. Chlorine = Residual free Chlorine

**Table 3:** Chronic daily intake (CDI) and age adjusted cancer risk estimated for ingestion exposure.

Parameters used	Drinking water (L/day)		CDI (ingestion)		Cancer risk	
			Jimma	Agaro	Jimma	Agaro
USEPA	1 L/day		1.8x10 <sup>-3</sup>	1.1x10 <sup>-4</sup>	1.1x10 <sup>-5</sup>	6.7x10 <sup>-7</sup>
(3 scenarios)	2 L/day		3.54x10 <sup>-3</sup>	2.2x10 <sup>-4</sup>	2.2x10 <sup>-5</sup>	1.3x10 <sup>-6</sup>
	3 L/day		5.3x10 <sup>-3</sup>	3.3x10 <sup>-4</sup>	3.2x10 <sup>-5</sup>	2x10 <sup>-6</sup>
This study	Minimum	Male	4.5x10 <sup>-4</sup>	2.7x10 <sup>-5</sup>	2.8x10 <sup>-6</sup>	1.65x10 <sup>-7</sup>
	(0.25 L/day)	Female	4.4x10 <sup>-4</sup>	2.6x10 <sup>-5</sup>	2.7x10 <sup>-6</sup>	1.58x10 <sup>-7</sup>
	Mean	Male	2.4x10 <sup>-3</sup>	1.4x10 <sup>-4</sup>	1.46x10 <sup>-5</sup>	8.54x10 <sup>-7</sup>
	(1.32 L/day)	Female	2.3x10 <sup>-3</sup>	1.4x10 <sup>-4</sup>	1.4x10 <sup>-5</sup>	8.54x10 <sup>-7</sup>
	Maximum	Male	4.5x10 <sup>-3</sup>	2.7x10 <sup>-4</sup>	2.75x10 <sup>-5</sup>	1.65x10 <sup>-6</sup>
	(2.5 L/day)	Female	4.4x10 <sup>-3</sup>	2.6x10 <sup>-4</sup>	2.68x10 <sup>-5</sup>	1.59x10 <sup>-6</sup>

**Table 4:** Cancer risk estimation of population exposed to chloroform in drinking water.

Concentration of Chloroform (µg/L)		TR (calculated with USEPA standard parameters)		TR (age, weight and amount of drinking water obtained from survey)	
		Jimma	Agaro	Jimma	Agaro
Min	Jimma = 41.67 Agaro = 2.49	1.098x10 <sup>-4</sup>	6.56x10 <sup>-6</sup>	1.46x10 <sup>-4</sup>	1.34x10 <sup>-4</sup>
Mean	Jimma = 93.75 Agaro = 4.67	3.14x10 <sup>-4</sup>	1.96x10 <sup>-5</sup>	4.19x10 <sup>-4</sup>	3.84x10 <sup>-4</sup>
Max	Jimma = 220.32 Agaro = 17.33	5.81x10 <sup>-4</sup>	4.57x10 <sup>-5</sup>	7.74x10 <sup>-4</sup>	7.08x10 <sup>-4</sup>

Min = minimum, Max = maximum, TR = target cancer risk

alone was calculated for populations of both study areas (Table 3). In Jimma town for both males and females; the CDI for mean chloroform concentration with 2L of water ingestion (USEPA’s standard adult water intake value) is greater than the CDI with mean water intake (1.32L) of current study. There is difference in CDI and cancer risk values for different water source, different water intake amount, and sex. We can also observe that the CDI and cancer risk values calculated using USEPA’s standard parameters were greater than values calculated using parameters of this study.

Age adjusted cancer developing risk was estimated (Table 4). The result indicated that risk was varied with different water sources and intake rates. Cancer risk varied based on the the variation of the concentrations of chloroform in drinking water. The cancer risk estimated for ingestion and inhalation exposure was 14 times higher than cancer risk calculated for ingestion exposure alone. This study revealed that, the cancer target risk of the population of Jimma town was higher than Agaro town.

## Discussion

The present study indicated that, water treatment targeting microbial and physical contaminants may not be sufficient to safeguard consumers’ health. A water source distributed after chlorination often contains chloroform at various levels while, not in raw water sources. The concentration of chloroform, which is an indicator of trihalomethane varied from 0 to 220.3 ± 77.2 µg/ in Jimma town which uses surface water sources. A similar study done in Russian city [29] also showed that the concentration of chloroform in tap water was 198±70 µg/L. While in Agaro town, the concentration ranges from 0 to 17.33 ± 5.3 µg/L. This is a clear indication that after chlorination, groundwater sources produce very trace amount of trihalomethane compared with chlorinated water from the surface water sources. The huge concentration difference between towns is possibly due to the

difference in precursors, mainly chlorin dose in which 5mg/L in the case of Russian city where as 3mg/L and 0.5mg/L in and Jimma and Agaro, respectively. The chloroform concentration in in both study areas are below the WHO guideline value for chloroform which is 300µg/L [1] but half of of the water samples from Jimma town water distribution system were greater than USEPA’s standards for THMs (80µg/L (initial stage), 60µg/L (stage 2) and 40µg/L (stage 3)) but no standard for chloroform [24].

Our study is almost in consistent with the study done in Canada according to [30], during the evaluation of THMs, chloroform was 80% of TTHMs in Beauport city (7 to 228 µg/L) which is almost in similar range with the results from Jimma town.

In the correlation test, there is statistically significant positive correlation between chloroform concentration and most of water quality and operational parameters (p<0.05). In general, water temperature appears to be the most influential parameter on chloroform occurrence in the distribution systems, for both Jimma and Agaro water distribution system. The chloroform concentrations were directly proportional with temperature. The reason behind is that in higher temperature, the reaction between chlorine and Natural Organic Matter (NOM) will be enhanced and formation of THMs gets faster. Few studies indicated that elevated concentration of chloroform was associated with increasing water temperature [30,31]. On top of that, residual chlorine is also one of the factors which determine the occurrence of DBPs [14]. It was seen that when residual free chlorine decreases, the concentration of chloroform production increases in Jimma town, which is in agreement with the study done by Garcia and his colleagues [32], but this condition will be true as long as free chlorine is abundant like in the case of Jimma town water supply system. Using Pearson correlation, there was no correlation between residual chlorine and chloroform concentration. Since the parameters where measured from actual working condition

of water distribution system and there is multi-collinearity between these parameters thus it may be the reason for absence of correlation.

In Jimma town, when pH and residence time controlled in partial correlation, there is strong positive correlation between chloroform concentration and TOC and UV absorbance with  $r^2$  values of 0.86 ( $p < 0.000$ ) and 0.58 ( $p < 0.048$ ) respectively. For Agaro town water supply system, there was positive correlation between chloroform and TOC and UV absorbance at 254nm when controlled for residence time with  $r^2$  values of 0.73 ( $p < 0.025$ ) and 0.77 ( $p < 0.015$ ) for TOC and UV absorbance respectively. The positive correlation agrees with the theoretical assumption that water with higher organic carbon content generates higher chloroform levels when chlorinated [2,18,32,33]. The most predicting factor of the elevated concentration of chloroform is pH. From the finding of this study, the concentration of chloroform increases with pH from 5.81 to 7.41 and 6.67 to 7.75 in the case of Jimma town and Agaro town, respectively. Using Pearson correlation, there was strong positive correlation ( $r^2 = 0.945$  and  $0.98$ ) in Jimma and Agaro towns respectively. This agrees findings of other researches [32,34].

In current study, the residence time is also found to be one of the predicting variables for the occurrence of chloroform. This means that having sufficient time for NOM to react with residual chlorine formation of chloroform will increase. The same is true for distance from treatment plant to sampling point. But the increment of chloroform is observed only when the residual chlorine is abundant. Thus, it is clearly observed that there was an increase in chloroform concentration between the treatment plant and the water distribution points, such as the public stand points and household taps. This result agrees with study of Rodriguez and his colleagues in 2001 but disagrees with finding of ye and his colleagues in 2009 in which the correlation between THMs with residence time was indefinite and they suggested that volatile DBPs' evaporation and biodegradability over time when the disinfectant residual is low as the cause of poor correlation.

Regression equations were tested using different explanatory variables representing water quality parameters (UV-absorbance, TOC) as well as operational conditions (water temperature, pH, residence time, distance and chlorine dose) from which pH, residence time and chlorine dose found to be best predictors of chloroform, which is also in agreement with the findings of [35]. However the other parameters were unable to predict occurrence of chloroform which disagrees with the theoretical thought of TOC and UV absorbance believed to be good predictors. The disagreement is possibly due to high collinearity between the parameters. Analysis of chloroform formation under real conditions of utility operation did not allow us to include all the operational and water quality parameters within the prediction model which theoretically influence the formation of THMs in drinking water. This is possibly due to the uncertainty on water residence time, water flow rate and distance.

As indicated in Table 2, the mean chloroform concentration in Jimma town surface water source is 19 fold higher than chloroform concentrations of Agaro town where the source is groundwater. Independent t- test indicted statistically significant difference between mean chloroform concentrations of the two towns. The differences in chloroform levels for the utilities being studied are mainly related to

the type of water being chlorinated, which is to say, the difference in THM precursor content, as well as the chlorine dose. This result agrees with other studies done by Chang et al. (1996) and Wang et al. (2006) where chloroform in chlorinated surface water was greater than chloroform from chlorinated water of groundwater [36,37].

The volume of tap water consumed is an essential element in quantitative microbial and chemical risk assessment. As most of the respondents responded, the average volume of water consumed were 1.23L/day and 1.32L/day in Jimma and Agaro towns respectively. This result is lower than study done in Korea [17] where the amount of water drunk was 1.36L/day. Our result is also lower than the WHO's and USEPA's daily water intake which is 2liter/day where the difference could be due to geographical and cultural differences.

CDI for chloroform from ingestion exposure is estimated and varied from  $4.4 \times 10^{-4}$  to  $5.3 \times 10^{-3}$  mg/kg day<sup>-1</sup> in Jimma town and from  $2.6 \times 10^{-5}$  to  $3.3 \times 10^{-4}$  mg/kg day<sup>-1</sup> in Agaro town. CDI for males which ranges from  $4.5 \times 10^{-4}$  to  $4.5 \times 10^{-3}$  mg/kg day<sup>-1</sup> in Jimma town are a little bit greater than for females which ranges from  $4.4 \times 10^{-4}$  to  $4.4 \times 10^{-3}$  mg/kg day<sup>-1</sup>. These values are less than USEPA's daily allowable intake value of chloroform which is 0.01mg/kg day<sup>-1</sup> [24].

The CDI estimated using USEPA standard parameters was greater than CDI estimated using exposure parameters from the survey data of this study. The difference is possibly due to differences in body weight and life expectancy. This result agrees with study in South China where CDI of males were greater than females. The CDI levels for both males and females in this study are greater than CDI's levels for both males and females of one study done in China [23]. The difference is may be due to high mean concentration of chloroform in the case of Jimma.

Cancer risk for 30 years ingestion and inhalation exposure was calculated for both Jimma town and Agaro town populations from drinking chlorinated water. It varied from  $1.098 \times 10^{-4}$  to  $3.14 \times 10^{-4}$  for Jimma town and from  $1.96 \times 10^{-5}$  to  $3.84 \times 10^{-4}$  in Agaro town. In both cases, the risk values are greater than WHO acceptable risk of cancer from drinking water in average of 70 years exposure which is  $1 \times 10^{-5}$  or 1 cancer case from 100,000 individuals [1]. According to USEPA, acceptable value is risk less than  $10^{-6}$  in 100,000 populations [38]. In the current study, the cancer risk was estimated for only chloroform via ingestion and inhalation exposure. From this, we may suspect that cancer risk value may much more higher than the calculated number if it is estimated for all known DBPs and other routes of exposure as well for 70 years. From this result we can also generalize that cancer risk from chloroform of surface water is greater than the risk from chloroform of groundwater. Our finding is in agreement with the study done in South China where the cancer risk from consumption of chlorinated surface water was greater than from chlorinated groundwater where estimated cancer risk was  $1.80 \times 10^{-4}$  (based on 3L/day) for chloroform in tap water which is abstracted from surface water; and where for well water the risk was  $2.73 \times 10^{-5}$ . The discrepancy between the surface and groundwater supply systems is most probably due to the differences in concentration of chloroform. Chloroform is known to contribute the majority of the lifetime cancer risks (ranging from 87.5 to 92.5%) of the total risks [26].

Our study gives an insight that the chemical risk from drinking chlorinated water should not be neglected in developing countries.

Although the microbiological quality of drinking water cannot be compromised, there is a need to better understand epidemiology of chemical disinfectants and their associated DBPs in order to develop a better understanding of the health risks associated with drinking water and to seek a balance between microbial and chemical risks. Possibilities have to be sought to decrease the chemical risk due to DBPs without compromising microbiological quality of drinking water in sub-Saharan Africa. This is because different studies already concluded that the risk of chlorination byproducts like chloroform is not only causing cancer but also it has reproductive effects like birth defects, abortion and low birth weight [12].

## Conclusion

This study indicated that attention is required to reduce disinfection by-products in the treatment plants. The presence of chloroform is confirmed in chlorinated treated water and but not observed in raw water sources. The mean concentration of chloroform in this study was greater than MCL set by USEPA. TOC, UV absorbance, water temperature, residential time, distance from treatment plant to sampling point and pH were found to be important parameters influencing chloroform formation. Chlorinating water from surface water makes more chloroform formation than from groundwater source. The exposure varied with source of chlorinated drinking water, amount of water intake, amount of chlorine used for treatment, amount of water consumed per day and life expectancy. The estimated cancer risk from chloroform exposure via ingestion alone, ingestion and inhalation exposure together in Jimma and Agaro town was greater than WHO's acceptable cancer risk value. Cancer risk estimated also varied with route of exposure, concentration of chloroform in water, source of drinking water, sex and estimation parameters from USEPA standard and some parameters from this study.

## References

1. WHO. Guidelines for drinking-water quality: first addendum to the third edition, volume 1: recommendations. Geneva: WHO. 2006.
2. Rook JJ. Formation of haloforms during chlorination of natural waters. *J Water Treat. Exam.* 1974; 23: 234–243.
3. Yin J, Wu B, Zhang X-X, Xian Q. Comparative toxicity of chloro- and bromo-nitromethanes in mice based on a metabolomic method. *Chemosphere.* 2017; 185: 20–28.
4. CDC. Disinfection By-Products [Internet]. *Safe Water Syst.* 2016.
5. IARC. Chloroform. Group 2B. IARC Monographs for the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol 73. Lyon: International Agency for Research on Cancer. 1999.
6. National Toxicology Program. Report on the Carcinogenesis Bioassay of Chloroform (CAS No. 67-66-3). *Natl. Cancer Inst. Carcinog. Tech. Rep.* 1976: 1–60.
7. IARC, editor. Some chemicals that cause tumours of the kidney or urinary bladder in rodents and some other substances: this publication represents the views and expert opinions of an IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, which met in Lyon. Lyon: IARC. 1999.
8. IARC, editor. IARC monographs on the evaluation of carcinogenic risks to humans, volume 97, 1,3-Butadiene, ethylene oxide and vinyl halides (vinyl fluoride, vinyl chloride and vinyl bromide): this publication represents the views and expert opinions of an IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, which met in Lyon, 5 - 12 June 2007. Lyon: WHO. 2008.
9. Larson JL, Wolf DC, Butterworth BE. Induced cytotoxicity and cell proliferation in the hepatocarcinogenicity of chloroform in female B6C3F1 mice: comparison of administration by gavage in corn oil vs ad libitum in drinking water. *Fundam. Appl. Toxicol. Off. J. Soc. Toxicol.* 1994; 22: 90–102.
10. Morris RD, Audet AM, Angelillo IF, Chalmers TC, Mosteller F. Chlorination, chlorination by-products, and cancer: a meta-analysis. *Am. J. Public Health.* 1992; 82: 955–963.
11. Chowdhury S, Rodriguez MJ, Sadiq R. Disinfection byproducts in Canadian provinces: Associated cancer risks and medical expenses. *J. Hazard. Mater.* 2011; 187: 574–584.
12. Grazuleviciene R, Nieuwenhuijsen MJ, Vencloviene J, Kostopoulou-Karadanelli M, Krasner SW, Danileviciute A, et al. Individual exposures to drinking water trihalomethanes, low birth weight and small for gestational age risk: a prospective Kaunas cohort study. *Environ. Health Glob. Access Sci. Source.* 2011; 10: 32.
13. Amy G. International Programme on Chemical Safety, editors. Disinfectants and disinfectant by-products. Geneva: World Health Organization. 2000.
14. Amy GL, Chadik PA, Chowdhury ZK. Developing Models for Predicting Trihalomethane Formation Potential and Kinetics. *Am. Water Works Assoc.* 1987; 79: 89–97.
15. Singer PC, Obolensky A, Greiner A. DBPs in Chlorinated North Carolina Drinking Waters (PDF). *Am. Water Works Assoc.* 1995; 87: 83–92.
16. Sadiq R, Rodriguez MJ. Disinfection by-products (DBPs) in drinking water and predictive models for their occurrence: a review. *Sci. Total Environ.* 2004; 321: 21–46.
17. Kim J. Fate of THMs and HAAs in low TOC surface water. *Environ. Res.* 2009; 109: 158–165.
18. Rizzo L, Selcuk H, Nikolaou A, Belgiorno V, Bekbolet M, Meric S. Formation of chlorinated organic in drinking water of Istanbul (Turkey) and Salerno (Italy). *Glob. NEST J.* 2005; 7: 95–105.
19. Bull RJ, Birnbaum LS, Cantor KP, Rose JB, Butterworth BE, Pegram R, et al. Water chlorination: essential process or cancer hazard? *Fundam. Appl. Toxicol. Off. J. Soc. Toxicol.* 1995; 28: 155–166.
20. Jimma Town Water and Sewerage Enterprise. Jimma Town Water Supply & Sewerage Enterprise, Business Plan Volume I. Jimma, Ethiopia. 2008.
21. ATWSO, Agaro Taown Water Office. Agaro town water supply office annual report of 2010. Ethiopia; 2010 p. 35.
22. Munch DJ. METHOD 551.1: Determination of chlorination disinfection by-products, chlorinated solvents, and halogenated pesticides/herbicides in Drinking water by liquid-liquid extraction and gas chromatography with electron-capture detection. USEPA; 1995.
23. Lee SC, Guo H, Lam SMJ, Lau SLA. Multipath way risk assessment on disinfection by-products of drinking water in Hong Kong. *Environ. Res.* 2004; 94: 47–56.
24. USEPA, Office of Water. Stage 2 Disinfectants and Disinfection Byproduct Rule (Stage 2 DBP rule). USEPA. 2010.
25. USEPA. Guidelines for Carcinogen Risk Assessment [Internet]. US Environmental Protection Agency; 2005.
26. Hsu CH, Jeng WL, Chang RM, Chien LC, Han BC. Estimation of potential lifetime cancer risks for trihalomethanes from consuming chlorinated drinking water in Taiwan. *Environ. Res.* 2001; 85: 77–82.
27. CSA Ethiopia. Census of Ethiopian Population. Central Statistics Agency of Ethiopia; 2007.
28. AOAC International. AOAC Guidelines for Single Laboratory Validation of Chemical Methods for Dietary Supplements and Botanicals. Association of Official Analytical Chemists, Arlington; 2002.
29. Egorov AI, Tereschenko AA, Altshul LM, Vartiainen T, Samsonov D, LaBrecque B, et al. Exposures to drinking water chlorination by-products in a Russian city. *Int. J. Hyg. Environ. Health.* 2003; 206: 539–551.
30. Rodriguez MJ, Vinette Y, Sérodes J-B, Bouchard C. Trihalomethanes in

- Drinking Water of Greater Québec Region (Canada): Occurrence, Variations and Modelling. 2003; 89: 69–93.
31. Wei J, Ye B, Wang W, Yang L, Tao J, Hang Z. Spatial and temporal evaluations of disinfection by-products in drinking water distribution systems in Beijing, China. *Sci. Total Environ.* 2010; 408: 4600–4606.
32. Garcia-Villanova RJ, Garcia C, Gomez JA, Garcia MP, Ardanuy R. Formation, evolution and modeling of trihalomethanes in the drinking water of a town: II. In the distribution system. *Water Res.* 1997; 31: 1405–1413.
33. Kristiana I, Gallard H, Joll C, Croué J-P. The formation of halogen-specific TOX from chlorination and chloramination of natural organic matter isolates. *Water Res.* 2009; 43: 4177–4186.
34. Ye B, Wang W, Yang L, Wei J, E X. Factors influencing disinfection by-products formation in drinking water of six cities in China. *J. Hazard. Mater.* 2009; 171: 147–152.
35. Rodriguez MJ, Sérodes JB. Spatial and temporal evolution of trihalomethanes in three water distribution systems. *Water Res.* 2001; 35: 1572–1586.
36. Chang EE, Chao S, Chiang P, Lee J. Effects of chlorination on THMs formation in raw water. *Toxicol. Environ. Chem.* 1996; 56: 211–225.
37. Wang W, Ye B, Yang L, Li Y, Wang Y. Risk assessment on disinfection by-products of drinking water of different water sources and disinfection processes. *Environ. Int.* 2007; 33: 219–225.
38. USEPA. General Principles for Performing Aggregate Exposure and Risk Assessments. United States Environmental Protection Agency, Office of Pesticide Program. 2001.