UC Davis
UC Davis Electronic Theses and Dissertations

Title

From Source to Household to Toxicity: Disinfection By-Product Formation Potential in California Drinking Water

Permalink

Author

Anderson, Berkley Nadine

Publication Date

2022

Peer reviewed|Thesis/dissertation

From Source to Household to Toxicity: Disinfection By-Product Formation Potential in California Drinking Water

By

BERKLEY ANDERSON
THESIS

Submitted in partial satisfaction of the requirements for the degree of

MASTER OF SCIENCE

in

Civil and Environmental Engineering

in the

OFFICE OF GRADUATE STUDIES

of the

UNIVERSITY OF CALIFORNIA

DAVIS

Approved:

Thomas M. Young, Chair

Heather N. Bischel

Jeannie L. Darby

Committee in Charge

2022

ABSTRACT

      With climate change, water shortages, and high anthropogenic activity, California faces many water quality challenges. One challenge includes the formation of disinfection by-products (DBPs) in drinking water due to reactions between disinfectants, natural organic matter, and ions present in source waters. While only a few compounds are regulated, significantly more toxic unregulated DBPs are emerging. In addition, DBPs can have significant variability within a given distribution system due to changes in demand, different water ages, changes in formation kinetics, and transformations. Thus, a better understanding on the occurrence, variability, and toxicity of DBPs in California drinking water is needed. As a component of the Drinking Water Project funded by the California Breast Cancer Research Program (CBCRP), the purpose of this study was to (1) investigate disinfection by-product formation potential (DBP–FP) in household drinking water across different water systems in California; (2) examine DBP variability within each distribution system; and (3) identify compounds that may be contributing to breast cancer toxicity. The results of this study indicate that regulatory monitoring approaches may not provide an accurate representation of household DBP exposure due to high variability and higher toxicity associated with unregulated compounds including dibromoacetonitrile (dBAN), bromochloroacetonitrile (BCAN), and iodoacetic acid (IAA). Due to their occurrence and role in driving toxicity, these specific compounds are recommended for prioritization in future research on long term exposure and breast cancer risks. In addition, higher unregulated DBP concentrations and toxicity were observed in public water systems that utilize complex water sources including high salinity water, brackish groundwater, and indirect potable reuse of recycled water. Potentially higher DBP exposures in these public water systems raises concern because such complex water sources are an increasingly important component of the California drinking water supply.

<p style="text-align: center;">CONTENTS</p>

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

1,1,1–TCE	1,1,1–trichloroethane	IAN	Iodoacetonitrile
1,1,2–TCE	1,1,2–trichloroethane	IDSE	Initial Distribution System Evaluation
AWWA	American Water Works Association	IQR	Interquartile Range
BAA	Bromoacetic Acid	IR	Irvine
BAN	Bromoacetonitrile	LA	Los Angeles
BCAA	Bromochloroacetic Acid	LLE	Liquid–Liquid Extraction
BCAN	Bromochloroacetonitrile	LOD	Limit of Detection
BCIM	Bromochloroiodomethane	LOQ	Limit of Quantification
BdCAA	Bromodichloroacetic Acid	LRAA	Locational Running Annual Average
BdCM	Bromodichloromethane	MC	Merced
BW	Bottled Water	MCL	Maximum Contaminant Level
CAA	Chloroacetic Acid	MD	Madera
CBCRP	California Breast Cancer Research Program	MS	Mass Spectrometry
CdBAA	Chlorodibromoacetic Acid	MTBE	Methyl Tert–Butyl Ether
CdIM	Chlorodiiodomethane	MW	Molecular Weight
CHO	Chinese Hamster Ovary	ND	Not Detected
CIAA	Chloroiodoacetic Acid	NOM	Natural Organic Matter
CT	Carbon Tetrachloride	PCE	Tetrachloroethylene
dBAA	Dibromoacetic Acid	PDMS	Polydimethylsiloxane
dBAN	Dibromoacetonitrile	QC	Quality Control
DBCP	1,2–dibromo–3–chloropropane	RSD	Relative Standard Deviation
DBP	Disinfection By–Product	SM	San Mateo
DBP–FP	Disinfection By–Product Formation Potential	tBAA	Tribromoacetic Acid
dCAA	Dichloroacetic Acid	TBM	Tribromomethane
EB	East Bay	tCAA	Trichloroacetic Acid
EC	Effect Concentration	TCE	Trichloroethylene
ECD	Electron Capture Detector	TCM	Trichloromethane
EDB	1,2–dibromoethane	TCP	1,2,3–trichloropropane
EPA	U.S. Environmental Protection Agency	TF–SPME	Thin–Filmed Solid–Phase Microextraction
GC	Gas Chromatography	THM	Trihalomethane
HAA	Haloacetic Acid	TOS	Total Oxidative Stress
HAN	Haloacetonitrile	WHO	World Health Organization
HLB	Hydrophilic Lipophilic Balanced	WV	Weaverville
IAA	Iodoacetic Acid	YT	Yurok Tribe

1. INTRODUCTION

Drinking water disinfection is notably recognized as one of the greatest public health achievements of the 20th century, significantly reducing waterborne pathogens responsible for disease outbreaks including typhoid, cholera, and salmonellosis (NRC, 1980). Chlorine remains the most widely used disinfectant in the United States. Other commonly used disinfectants include chlorine dioxide, chloramines, and ozone. Although disinfection has successfully controlled waterborne diseases and increased overall life expectancy, an unintended consequence includes the formation of toxic and carcinogenic compounds known as disinfection by-products. Disinfection by-products (DBPs) are formed when such chemical disinfectants react with natural organic matter (humic and fulvic acids or other organic compounds), bromide, iodide, and nitrogen in the source water.

1.1 DBP Regulatory Compliance and Methods

Currently eleven disinfection by-products are regulated in the U.S. under the EPA Stage 2 Disinfectants and Disinfection Byproducts Rule (DBPR) including 4 trihalomethanes (THM_4), five haloacetic acids (HAA_5), bromate, and chlorite (USEPA, 2005). The maximum contaminant level (MCL) for THM_4 and HAA_5 are 80 µg/L and 60 µg/L, respectively. Under the Stage 2 DBPR, community water systems and non-transient non-community water systems must conduct an initial distribution system evaluation (IDSE) to identify locations with the highest DBP concentrations. These locations are then used as monitoring sites where compliance is based on the locational running annual average (LRAA) for each monitoring site (USEPA, 2005). The number of required monitoring sites and monitoring frequency is dependent on the source water type and size of population served.

Monitoring of regulated DBPs must be conducted by a certified laboratory using EPA approved analytical methods (USEPA, 1995). These approved methods all require the use of preservatives or dechlorinating agents such as ammonium chloride or sodium thiosulfate to prevent further reactions between disinfectants and precursors. For each monitoring location this represents DBP formation at a specific point in time. However if disinfectants and precursors are present, they will continue reacting as water age increases. Thus, DBP formation potential (DBP–FP) experiments are designed to examine DBP formation by allowing reactions between disinfectants and precursors to approach completion (Chen and Westerhoff, 2010; Krasner et al., 2008). While this allows plant operators to better understand DBP precursor removal efficacy and DBP speciation, it also represents potential household exposure if tap water is allowed to sit for extended periods of time before consumption.

1.2 DBP Formation, Speciation, and Variability

While only 11 compounds are regulated, there are about 600 known DBPs. Only a small fraction of those have been studied for their quantitative occurrence and associated health effects (Richardson, 2011). Emerging unregulated DBPs of interest include iodo-acids, iodo-THMs halonitromethanes, haloacetonitriles, and haloketones (Richardson et al., 2007; Richardson, 2011). DBP formation and speciation varies according to the type and amount of NOM and ions present in source water, type of disinfectant, disinfectant concentration, contact time, pH, and temperature (Guilherme and Rodriguez, 2015). Due to changes in source water quality and treatment operations, DBP formation and speciation can exhibit significant temporal variability. In addition, DBPs are also known to have considerable spatial variability throughout the distribution system itself. Such spatial variability may be related to the nature of the distribution

system (changes in demand, storage tank turnover, varying water ages); nature of the pipelines (pipe failures, leaching of pipe material, presence of biofilms); and transformations (biodegradation, volatilization) (Baribeau, 2006). Other studies have observed the highly variable nature of both regulated and unregulated DBPs within a given distribution system (Charisiadis et al., 2015; Guilherme and Rodriguez, 2015, 2014; Shanks et al., 2013; Wei et al., 2010).

Considering the highly variable nature of DBPs and emergence of more unregulated DBPs, this raises the question – *do regulatory monitoring approaches and location-specific running annual averages provide an accurate representation of household exposure to DBPs?*

1.3 DBP Toxicity (Epidemiology, *In Vivo*, *In Vitro*)

Exposure to disinfection by-products in drinking water have been correlated with numerous adverse health effects in epidemiologic, *in vivo*, and *in vitro* studies. Epidemiologic evidence has shown a strong correlation between bladder cancer and long–term exposure to THM in drinking water (Villanueva et al., 2015). *In vivo* studies in rats have observed tumor induction in the kidney, large intestine, liver, lung, mammary gland, oral cavity, peritoneal mesothelium, thyroid gland, and stomach organs with long-term exposure to DBPs (Melnick and Hooth, 2011). *In vivo* studies in rats have also correlated DBP ingestion in drinking water with adverse reproductive and developmental outcomes including fetotoxic effects, adverse sperm effects, impaired fetal development, delayed sexual maturation, changes in reproductive organs/placenta, and skeletal defects (Tardiff et al., 2006). However, there are some limitations to epidemiologic and *in vivo* studies including variability in genetic factors, health status, diets, lifestyles, and extrapolation from observed animal to predicted human response (Melnick and

3

Hooth, 2011). In addition, regulated compounds are typically used as surrogates in these studies to represent overall DBP toxicity and health outcomes. However, studies using *in vitro* mammalian cell bioassays have revealed that emerging unregulated compounds can be significantly more toxic (Plewa et al., 2017, 2008; Plewa and Wagner, 2011; Richardson et al., 2007)

In vitro bioassay tests are emerging as a useful tool to assess the relative toxicity of chemicals such as disinfection by-products in drinking water (Neale and Escher, 2019). The use of *in vitro* bioassay tests allows systematic testing of chemicals with reproducible cell lines and a range of biological endpoints. Such biological endpoints are indicative of different cellular toxicity pathways including cytotoxicity, genotoxicity, mutagenicity, xenobiotic metabolism, hormone or endocrine receptors, reactive modes of action, and adaptive stress responses (Neale and Escher, 2019). Although *in vitro* bioassay tests are not useful in projecting cancer risks, they can be used to identify which disinfection by-products and toxicity pathways are of concern. Using assays based on Chinese hamster ovary (CHO) cells, studies have found that the decreasing order of DBP cytotoxicity and genotoxicity includes iodo- > bromo- >> chloro-containing DBPs (Plewa, 2008). Cytotoxicity and genotoxicity endpoints are used to evaluate damage to the cell and damage to DNA, respectively. Nitrogen containing DBPs (N-DBPs) such as haloacetonitriles (HANs) are emerging as the most significant drivers of cytotoxicity and genotoxicity in treated drinking water (Plewa et al., 2017, 2008).

In addition to cytotoxicity and genotoxicity, studies have investigated DBP toxicity for other biological endpoints. One study tested 50 DBPs with nine different bioassays representative of different toxicity pathways and biological endpoints (Stalter et al., 2016). The results suggest that DBPs are more toxic through oxidative stress induction or enzyme inhibition

rather than direct cell damage. One of the tested bioassays for oxidative stress induction includes the AREc32 assay, which is based on the MCF7 human breast cancer cell line (Stalter et al., 2016). This toxicity pathway has been shown to be particularly sensitive to DBPs with HANs, haloketones, mono-HAAs, and iodo-DBPs as the most potent groups (Stalter et al., 2020, 2016). In this study, effect concentrations from the study conducted by (Stalter et al., 2016) will be used to investigate which DBPs detected throughout the distribution are contributing to oxidative stress induction in the human breast cancer cell line.

Currently only chloro- and bromo- containing DBPs are regulated under the Stage 2 DBPR. While these constitute the bulk of detected DBP concentrations, emerging unregulated DBPs are orders of magnitude more toxic and may be driving toxicity in disinfected drinking water. This raises another question – *are regulated DBPs accurate representations of overall toxicity?*

1.4 CBCRP Drinking Water Project and Objectives

This study is a component of the Nontarget Chemical Analysis of California Drinking Water, funded by the California Breast Cancer Research Program (CBCRP). To our knowledge, this project is the largest and most comprehensive investigation of target and non-target chemicals in California drinking water at the household level and their role as mammary gland carcinogens or estrogenic endocrine disruptors. This paper focuses specifically on the role of DBPs and aims to (1) investigate DBP formation potential (DBP–FP) across different drinking water systems and water sources in California (2) examine DBP spatial and temporal variability within each distribution system (3) identify DBP compounds that are driving oxidative stress induction in the AREc32 breast cancer cell line.

DBPs examined in this study include THM$_4$ (trichloromethane, bromodichloromethane, dibromochloromethane, tribromomethane); HAA$_5$ (chloroacetic acid, dichloroacetic acid, trichloroacetic acid, bromoacetic acid, dibromoacetic acid); HANs (bromoacetonitrile, bromochloroacetonitrile, dibromoacetonitrile, iodoacetonitrile); unregulated HAAs (bromochloroacetic acid, bromodichloroacetic acid, chlorodibromoacetic acid, tribromoacetic acid, chloroiodoacetic acid, iodoacetic acid); unregulated THMs (bromochloroiodomethane, chlorodiiodomethane); and other chlorinated solvents as presented in Table S1.

1.5 Overview of Studied Drinking Water Systems

This study includes the investigation of eight public water systems in California as well as various brands of bottled water. These public water systems were chosen to encompass a range of characteristics including varying water source type, water treatment and disinfection processes, population size served, and nature of the distribution system. Four of the selected public water systems including San Mateo, East Bay, Los Angeles, and Irvine, have been identified as serving areas that have historically elevated breast cancer rates (PHI and CBCRP, 2012) An overview of studied public water systems and their characteristics is shown in Table 1 and Figure 1. Additional information and water quality reports for each region are presented in Table S2.

Table 1. Overview of Studied Public Water Systems

REGION	WATER SYSTEM	SOURCE WATER TYPE	DISINFECTION TYPE
San Mateo	California Water Services, San Mateo	Large Surface Water	Chloramination
East Bay	East Bay Municipal Utility District		
Weaverville	Weaverville C. S. D.	Small Surface Water	Chlorination
Yurok	Yurok Tribal Environmental Program		
Los Angeles	LA City Department of Water & Power	Mixed Water	Chloramination
Irvine	Irvine Ranch Water District		
Madera	City of Madera	Groundwater	Chlorination
Merced	City of Merced		
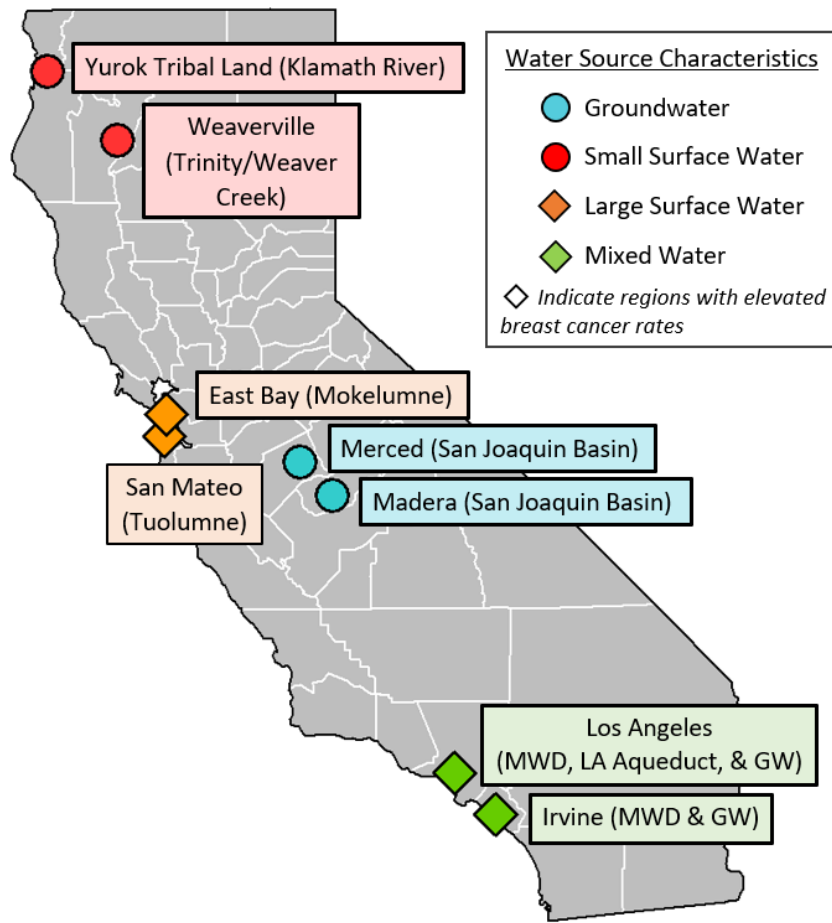
Figure 1. Map of Studied Public Water Systems in California

1.6 Overview of Studied Bottled Water Brands and Local Refill Stations

In addition to household drinking water, various brands of bottled water and local refill stations were also included in this study. All bottled, canned, and boxed water brands source their water from either natural springs or municipal drinking water which is then further purified by a range of processes including filtration, carbon filtration, distillation, reverse osmosis, or additional disinfection. Refill stations typically use on-site water treatment or point-of-use technologies including filters, activated carbon filters, and ultraviolet lamps. Studied bottled water brands and local refill stations are summarized in Table S3.

2. METHODS AND MATERIALS

2.1 Public Water Systems Selection and Sampling

The eight studied water systems were selected to include different water source types (large surface water, small surface water, mixed water, and groundwater); disinfection processes (chlorination and chloramination); and distribution system characteristics (population and geographic area served). For each water system, the California Public Water Supply Systems Search Tool (Drinking Water Watch) was used to select neighborhoods within service areas and (CalEnviroScreen 3.0) was used to select neighborhoods with the greatest economic diversity. After collaboration with advocacy partners and a recruiting process, 15 households from each water system were selected for participation. Sampling locations within each region are shown in Figure S1. In addition to household samples, water from various bottled water brands from local grocery stores and local refill stations were collected and analyzed.

Regions were sampled consecutively each week in the following order: San Mateo, East Bay, Bottled Water, Weaverville, Yurok, Los Angeles, Irvine, Madera, and Merced, for a total of 9 weeks. Sampling kits were shipped to all selected households within a region and then

collected, transported back, and processed on the same day the following week. Winter samples were collected from January to April 2020 and summer samples were collected from July to October 2020.

2.2 Materials and Reagents

All materials and reagents were purchased at the highest purity available. Standards were purchased from Accustandard Inc., New Haven, CT; Sigma-Aldrich, St. Louis, MO; and Toronto Research Chemicals Inc., Toronto, Ontario. All compounds and solvents used for extraction including sodium bicarbonate, sodium sulfate, acetone, hexane, methanol, methyl tert-butyl ether (MTBE), and sulfuric acid were purchased from Thermo Fisher Scientific, Waltham, MA and Sigma-Aldrich, St. Louis, MO. Carrier gas tanks for gas chromatography (GC) instruments including nitrogen, helium, and argon (5% methane) were purchased from Airgas, Radnor, PA.

2.3 Analytical Methods

2.3.1 Disinfection By-Product Formation Potential Tests

Household samples were collected in amber glass bottles where participants were instructed to fill bottles completely to the top to avoid volatilization during transport. The collected sample bottle was then placed in a cooler with 3 ice packs. Coolers were picked–up from each household and transported to the UC Davis Civil and Environmental Engineering Department via Rapidus courier service. Upon arrival, samples were processed and aliquoted into 40 mL amber glass vials for HAA (GC–ECD) extraction and 50 mL amber glass vials for THMs, HANs, and other chlorinated solvents (GC–MS) extraction. Aliquots were also filled to

the top to avoid volatilization. The aliquots were then allowed to continue reacting for 7 to 14

days at 4°C and extracted using the following methods:

2.3.2 Haloacetic Acids (HAAs)

All HAAs were extracted using a liquid-liquid extraction (LLE) with 40 mL of sample, 4

mL of methyl tert-butyl ether (MTBE), and 18 g of sodium sulfate. Samples were acidified with

sulfuric acid to ensure pH < 0.5 and derivatized by sample methylation with acidified methanol.

Extraction methods were derived from EPA Method 552.3, however a dechlorinating agent was

not used (USEPA, 2003). Extracts were then analyzed using GC–ECD (Agilent 6890) and

methods outlined in Section S2.1. Results were analyzed using Agilent ChemStation Software

and Microsoft Excel.

2.3.3 Trihalomethane (THMs), Haloacetonitriles (HANs), Chlorinated Solvents

All remaining compounds including THMs, HANs, and other chlorinated solvents were

extracted using thin-filmed solid-phase microextraction (TF–SPME) with polydimethylsiloxane/

hydrophilic lipophilic balanced (PDMS/HLB) fibers. Preconditioned fibers were immersed in 10

mL of sample in 12 mL amber vials which were then placed in a tube rotator for 30 minutes at 30

rpm. The fibers were allowed to dry fully before being placed in thermal desorption tubes and

analyzed using GC–MS (Agilent 6890) paired with an automated thermal desorption system

(Markes International ULTRA–xr) and methods outlined in Section S2.2. Results were analyzed

using Agilent MassHunter Quantitative Analysis Software.

The limit of detection (LOD) and limit of quantification (LOQ) for all compounds are

summarized in Table S6. The LOQ for all compounds ranged from 0.025–5 µg/L however most

were at 0.1 µg/L. With each batch of samples, a quality control (QC) sample was analyzed to monitor method precision and accuracy for all compounds. The relative standard deviation (RSD) between QC recoveries (GC–ECD) and QC peak response (GC–MS) are presented in Table S6. Accuracies for all points used in the calibration curve from the LOQ to the maximum calibration point are also reported in Table S6. Retention Times for GC–ECD and GC–MS are presented in Table S4 and Table S5, respectively.

2.4 Statistical Analysis

Variance within each distribution system was determined by dividing or normalizing detected concentrations by the total average of each dataset. This serves to better understand what the locational running annual average (LRAA) represents in monitoring compliance and how DBP formation might vary from this value throughout the distribution system. Reported boxplots are characterized by the minimum, 1st quartile, 2nd quartile, 3rd quartile, and maximum in each data set. All quartile calculations made in RStudio and Microsoft Excel were based on the inclusive median method and outliers were included. Values between the LOD and LOQ were included in variability and toxicity analysis for compounds that had high detection frequencies (>60%) within a given distribution system. Although these values have a higher uncertainty, they were included to better represent potential exposure and toxicity.

2.5 Estimated AREc32 Total Oxidative Stress Induction

This approach was based on the TIC–TOX method developed by (Plewa et al., 2017) which uses GC peak response (TIC) and Chinese hamster cell cytotoxicity (TOX) to identify DBP compounds driving toxicity in drinking water. In this paper quantitative concentrations

were analyzed and effect concentrations for the AREc32 biological endpoint were used. Effect

concentrations ($EC_{IR1.5}$) for individual DBPs were used from the study conducted by (Stalter et

al., 2016). These values elicit an induction ratio of 1.5 or a 50% effect increase relative to the

negative control. The AREc32 total oxidative stress (TOS) induction for each sample was

calculated using the following equation:

$$Total\ Oxidative\ Stress\ (TOS) = \Sigma\ [DBP] * (\ MW * EC_{IR1.5})^{-1} * 10^6 \quad (Eq.\ 1)$$

Where [DBP] is the concentration of each compound in (g/L), MW is the molecular

weight of the compound (g/mol), $EC_{IR1.5}$ is the effect concentration (mol/L), and 10^6 is a

normalization factor. Additional information on the AREc32 reporter gene assay is provided in

Section S2.3 and $EC_{IR1.5}$ values used for each compound are summarized in Table S7.

3. RESULTS AND DISCUSSION

3.1 Detection Frequencies

Detection frequencies for all compounds and regions are shown in Table S8 and represent

the percentage of samples within each region that have compounds detected above the LOD. In

chlorinated drinking water systems, chloroform is typically the primary DBP formed and

constitutes ~90% of total THM concentrations (WHO, 2005). Chloroform was detected in every

region. However, 64% of summer detects and 41% of winter detects saturated the GC–MS

detector and were beyond the linear range of the calibration curve. Thus, the regulated THM$_4$

class was broken down into chloroform (TCM) and the remaining THM$_3$. While extrapolated

chloroform concentrations are reported, it should be noted that these values have a high degree of

uncertainty and were not considered in the variability analysis. Extrapolated chloroform

concentrations were used in toxicity analysis. However, chloroform was one of the least potent

DBP for AREc32 oxidative stress induction and did not have a significant impact on toxicity to the MCF7 breast cancer cell line.

All "other" chlorinated solvents had sparse detects with the majority below the LOQ. Madera winter samples had reporting values for TCE ranging from ND–0.66 µg/L and PCE ranging from ND–3.10 µg/L. However, 98% of remaining chlorinated solvent detects across all other samples were below the LOQ. Due to higher uncertainty of detected concentrations and lack of $EC_{IR1.5}$ values for these compounds, all "other" chlorinated solvents were not included in the variability and toxicity analyses. While our results suggest that some compounds including EDB, PCE, CT, and TCE may be present in one or more regions at the *ng/L* level, more research on the occurrence, toxicity, and associated health effects of these compounds may be needed.

When analyzed using the methods described in section 2.3.2, chloroacetic acid (CAA) coeluted with an unidentified compound in samples from some regions. Duplicates of all winter and summer samples were analyzed using GC–ECD and methods outlined in Section S2.1. However, an Agilent J&W DB-5MS GC column was used and the method was modified to a shorter run time to exclusively analyze CAA. CAA concentrations across regions and seasons ranged from ND–8.15 µg/L.

3.2 Disinfection By-Product Formation Potential (DBP–FP)

3.2.1 Bottled Water and Local Refill Stations

The DBP–FP concentrations for all bottled water and local refill station samples across winter (W) and summer (S) seasons are shown in Figure 2. TCM was the most detected compound, accounting for more than 68% of the total DBP–FP detected across all bottled, boxed, canned, and refill water samples. The remaining detects include primarily the other

regulated THM4 and HAA5 compounds as well as a few detects of unregulated BCAA, BdCAA, and BCAN. All bottled, boxed, and canned water brands had relatively low concentrations. This is reasonable considering that studied brands all source their water from either natural spring water or tap water which is further purified by a combination of processes including filtration, distillation, reverse osmosis, and additional disinfection. Refill stations overall had higher concentrations which could be due to onsite treatment and less extensive purification processes. Large variations in concentrations between sampling periods in refill station water could also be due to systems being at different points within their treatment service lives.
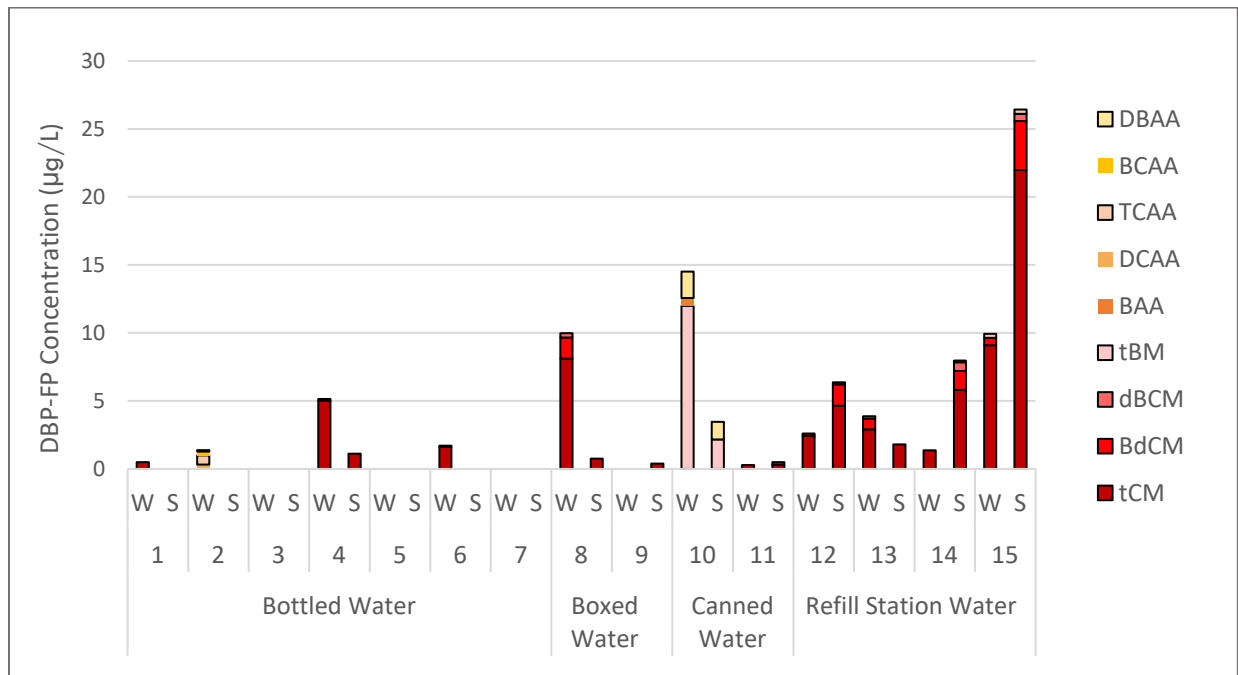
Figure 2. DBP–FP for Bottled Water Brands and Refill Stations across Winter (W) and Summer (S) Sampling. Compounds with the highest detected concentrations are outlined in black.

(1–Dasani, 2–Nestle, 3–Arrowhead, 4-Kirkland, 5–Crystal Geyser, 6–Aquafina, 7–Smart Water, 8–Boxed Water is Better, 9–Just Water, 10–Ever and Ever, 11–Pathway, 12–H20 to Go Refill, 13–Safeway, 14–Davis Food Co–op, 15–Nugget)

3.2.2 Public Water Systems

DBP–FP averages for each DBP class and region are shown in Table 2. The highest

DBP–FP levels by source water type include large surface water ~ small surface water > mixed

water > groundwater. Regions with similar water source type and disinfection processes had

comparable DBP–FP concentrations and DBP speciation. Observed similarities and results by

grouped regions are discussed in the following sections.

Table 2. Disinfection By-Product Formation Potential Averages (µg/L)

	SUMMER							
DBP Class	SM	EB	WV	YT	LA	IR	MD	MC
TCM	35.69*	83.87*	30.58*	64.53*	18.36*	24.20*	< LOD	1.45
THM_3	0.55	4.93	2.03	4.83	23.12	18.76	1.09	1.64
THM_{ur}	< LOD	< LOD	< LOD	< LOD	0.27	0.16	< LOD	< LOD
HAA_5	26.24	30.14	16.20	18.39	7.97	7.72	0.13	0.14
HAA_{ur}	0.70	3.07	1.07	1.66	4.65	2.91	< LOD	0.07
HAN	< LOD	< LOD	0.16	0.11	1.77	1.00	0.09	0.21
Total	63.19	122.06	50.04	89.53	56.13	54.75	1.41	3.52
	WINTER							
DBP Class	SM	EB	WV	YT	LA	IR	MD	MC
TCM	13.05*	33.56*	26.76*	51.76*	2.41	5.34	< LOD	0.42
THM_3	1.79	0.43	0.24	2.20	7.89	6.99	2.24	0.93
THM_{ur}	0.18	< LOD	< LOD	< LOD	0.64	0.39	< LOD	< LOD
HAA_5	15.02	22.88	22.68	21.84	7.36	4.79	0.11	0.26
HAA_{ur}	2.49	1.89	0.76	1.52	3.76	3.77	< LOD	< LOD
HAN	0.06	< LOD	< LOD	0.05	3.63	3.19	0.52	0.03
Total	32.63	58.77	50.44	77.36	25.70	24.46	3.65	1.73

* More than 80% of samples saturated detector for chloroform (extrapolated values included)

3.2.2 (a) Los Angeles and Irvine – Mixed Water Source

Los Angeles and Irvine had relatively low concentrations for both seasons with

household level total DBP–FP ranging from 18.95–65.14 µg/L and 2.74–77.54 µg/L,

respectively. However, Los Angeles and Irvine samples had the highest unregulated DBP–FP

sum and the most diverse speciation, with a total of 12 unregulated compounds detected.

Household level total unregulated DBP–FP in Los Angeles and Irvine ranged from 3.82–12.91

μg/L and ND–10.24 μg/L, respectively and comprised primarily of BCAA, BdCAA, CdBAA, BCAN, dBAN, and BCIM. The diversity of compounds detected in these samples may be attributed to the complex nature of Los Angeles and Irvine's water supplies. Figure 3 shows the annual breakdown of Los Angeles and Irvine's water sources as reported in their 2020 consumer confidence reports. While these percentages may not reflect water composition at the time of sampling, they provide a snapshot of potential water sources.

Both Los Angeles and Irvine rely on water imported by the Metropolitan Water District of Southern California (MWD) which consists of surface water from Northern California via the State Water Project and the Colorado River via the Colorado River Aqueduct. The Colorado River is known to have historical elevated salinity levels (Tuttle and Grauch, 2009). Both regions also use groundwater that is impacted by seawater intrusion. Higher bromide and iodide levels in high salinity source waters and brackish groundwater may result in the increased formation of bromo- and iodo- DBPs (Li and Mitch, 2018). Los Angeles and Irvine had the highest reported bromide levels in their 2020 consumer confidence reports which ranged from 30–200 μg/L and ND–190 μg/L, respectively. To combat seawater intrusion and recharge aquifers both regions also inject highly treated wastewater or recycled water into their groundwater supplies, a process referred to as indirect potable reuse. However, DBPs are emerging as a concern for both direct and indirect potable reuse applications due to the increased amount of organic material, dissolved organic nitrogen, ammonia, bromide, and iodide present in recycled waters (Alexandrou et al., 2018; Farré and Gernjak, 2021). Although we cannot identify which water source is contributing specifically to observed DBP speciation in Los Angeles and Irvine household samples, future disinfection by-product formation potential experiments on each individual water source may help identify which sources are contributing DBP precursors of concern.
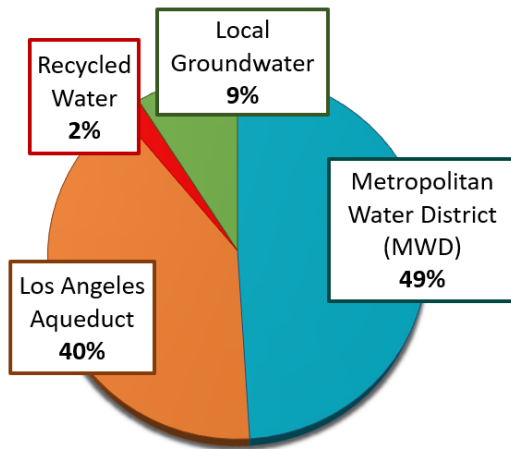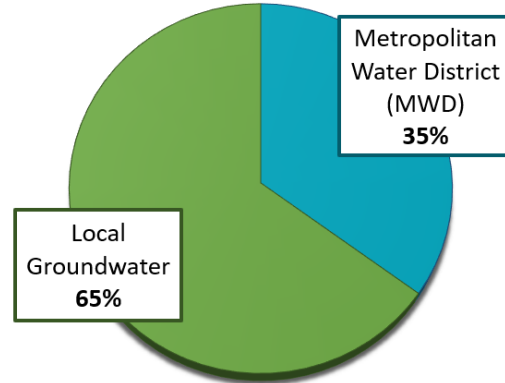
Figure 3. Breakdown of Los Angeles and Irvine Water Sources as Reported in 2020 Consumer Confidence Reports

In addition, both Los Angeles and Irvine use ammonia to form chloramines as a secondary disinfectant. While chloramination typically results in reduced overall DBP formation (Bougeard et al., 2010) increases in HANs and iodo-THMs have been observed (Richardson, 2011; Richardson and Plewa, 2020). Bromochloroiodomethane (BCIM) was consistently present in both regions and seasons at concentrations ranging from 0.016–1.41 µg/L with a few non–detects. HAN concentrations were highest in Los Angeles and Irvine with concentrations ranging from 0.82–7.44 µg/L and ND–6.13 µg/L, respectively. The removal of HAN precursors has been linked to nitrification efficiencies in wastewater treatment plants (Krasner et al., 2009), indicating that observed formation of HANs may also be a result of high levels of organic amines in the recycled water sources. Figure 4 shows the breakdown of compound averages detected in Los Angeles summer samples. These trends were similar across seasons in Los Angeles and Irvine. These results demonstrate the occurrence of HANs, iodo-THMs (BCIM), and other brominated DBPs in drinking water sourced with complexed water supplies.
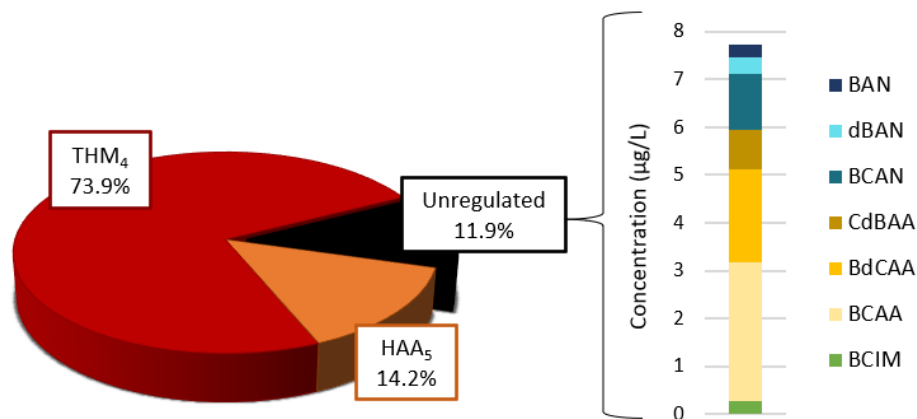
Figure 4. Breakdown of Compound Averages Detected in Los Angeles Summer Sampling

3.2.2 (b) San Mateo, East Bay, Weaverville, and Yurok – Surface Water Source

San Mateo and East Bay rely on large surface water systems including the Hetch Hetchy Regional Water System and Mokelumne River Watershed, respectively. Weaverville and Yurok Tribal Land also rely on surface water sources, but these are primarily local rivers or creeks. DCAA and tCAA were detected in every sample across the four regions at concentrations ranging from 2.85–33.08 µg/L. This is reasonable considering that dCAA and tCAA are typically the most dominant HAAs found in standard drinking water (Plewa et al., 2010). Chloroform was also detected at higher concentrations in the surface water sources, and remaining concentrations primarily consisted of THM_4, HAA_5, BCAA, BdCAA, BCAN, and IAA in the summer. Interestingly, BCIM was only detected in San Mateo winter samples at concentrations ranging from 0.16–0.24 µg/L which may be due to San Mateo's use of chloramines. East Bay also uses chloramines however BCIM was not detected in either sampling period. Unlike the mixed water chlormainated systems, San Mateo and East Bay had relatively low or not detected HAN concentrations. This indicates that high observed HAN concentrations in Los Angeles and Irvine are likely due to higher levels of organic amines in the mixed water sources.

3.2.2 (c) Madera and Merced – Groundwater Source

Madera and Merced both rely on groundwater from the San Joaquin Basin. Madera and Merced overall had the lowest total DBP–FP concentrations ranging from ND–10.92 µg/L and 0.62–11.25 µg/L, respectively. THM4, dBAA, BCAN, and dBAN were the primary compounds detected in both regions with more brominated compounds formed compared with their chlorinated analogs. This shift in speciation could be due to high bromide levels (>50 µg/L) in the source water (Plewa et al., 2010). While bromide levels in Madera are not reported, Merced reported bromide levels ranging from 24–170 µg/L in their 2020 consumer confidence report. These values were comparable to the reported bromide levels in Los Angeles and Irvine.

3.3 Spatial and Seasonal Variability

3.3.1 Spatial Variability

The spatial variability for summer and winter sampling is shown in Figure 5 and Figure 6, respectively. Presented boxplots represent the spread of DBP–FP concentrations in each distribution system relative to the regional average as shown in Table 2. It should be noted that regional averages differ significantly and while some boxplots may appear to be smaller, the absolute difference in concentrations may be larger. For example, the difference between the 3rd and 1st quartile or interquartile range (IQR) for HAA5 in Weaverville winter sampling was 8.70 µg/L while Merced winter sampling was only 0.11 µg/L. The IQRs for each DBP class across all regions and both seasons varied as follows: THM3 (0.03–4.34 µg/L), HAA5 (0.08–8.70 µg/L), THM$_{UR}$ (0.03–0.32 µg/L), HAA$_{UR}$ (0.03–1.45 µg/L), and HAN (0.01–1.73 µg/L). IQRs further broken down by each region and season are presented in Table S9. Implications for observed

DBP variability in terms of potential toxicity to the AREc32 breast cancer cell line is discussed in a later section.

Overall, concentrations by DBP class ranged anywhere from ND to 4.3 times the regional average supporting the highly variable nature of DBPs within a given distribution system. Observed outliers across both GC–ECD and GC–MS methods indicate potential "dead zones" or areas of stagnant water in the distribution system, locations of storage tank turnover, or presence of pipeline biofilms. These results support other studies' conclusions that locations selected for monitoring purposes may not comprehensively represent DBP exposures due to the high spatial variability within the distribution system (Guilherme and Rodriguez, 2015).

3.3.2. Temporal Variability

DBP temporal or seasonal variability can be caused by changes in source water quality and treatment operations. Overall DBP–FP concentrations were highest in the summer versus the winter except for Weaverville and Madera which remained about the same across both seasons. Typically, DBP concentrations are highest in the summer due to higher amount of organic matter present in source waters. Additionally, the surge use of chlorine–based disinfectants during the COVID–19 pandemic has also been linked to higher DBP levels in wastewater and drinking waters (Chu et al., 2021). The COVID–19 shutdown in California occurred between winter and summer sampling periods (late March 2020) which could also explain higher detected concentrations in the summer.
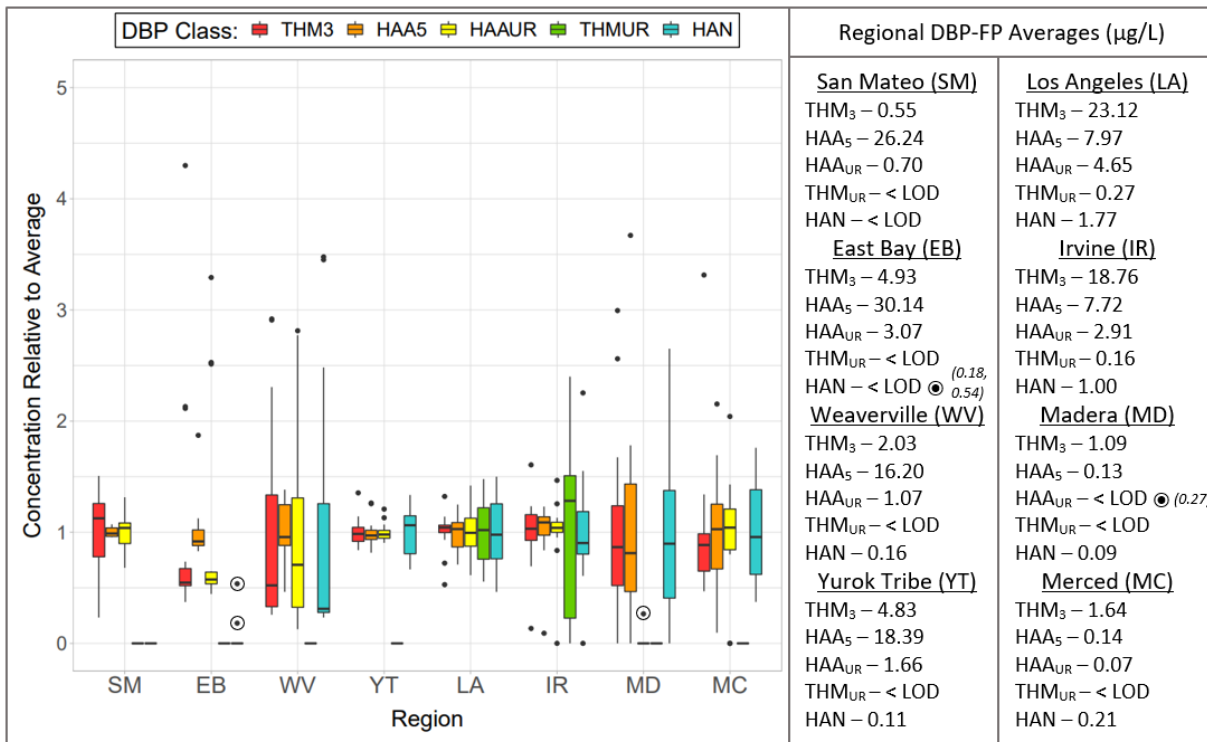
Figure 5. Spatial Variability of DBP Classes in Summer Season (July – October 2020)

◉ *Denotes concentrations of spikes in regions, spike concentrations (µg/L) are reported in the tabulated values on the right*
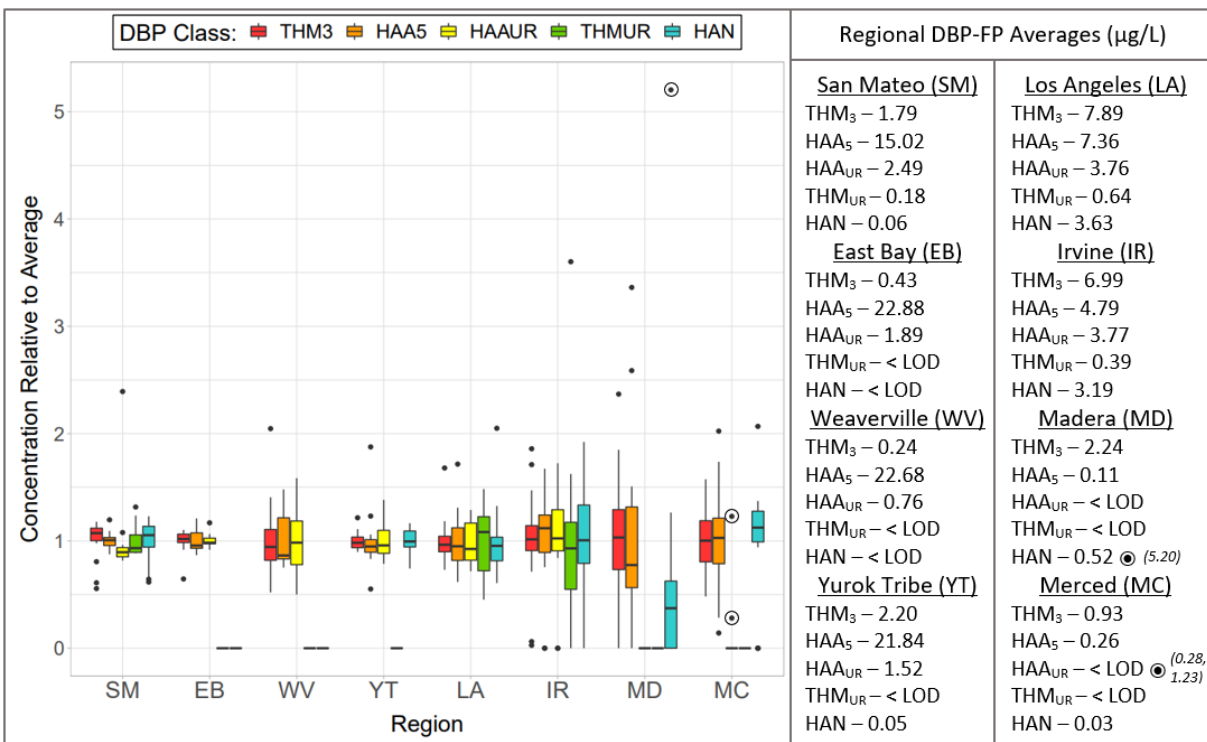
Figure 6. Spatial Variability of DBP Classes in Winter Season (January – April 2020)

3.4 Estimated AREc32 Total Oxidative Stress Induction

The total oxidative stress (TOS) on the AREc32 breast cancer cell line was calculated for each sample using detected concentrations, $EC_{IR1.5}$ values (Stalter et al., 2016), and equation 1. Figure 7 shows the minimum, 1st quartile, 2nd quartile, 3rd quartile, and maximum calculated TOS within each region on the 3–D bar plot (left) as well as the compounds driving toxicity in the median on 100% stacked bar plot (right). Los Angeles and Irvine had the highest TOS response across both seasons which was driven primarily by detected dBAN and BCAN concentrations. BAN was also detected in Los Angeles and Irvine summer samples however this compound did not have an $EC_{IR1.5}$ value and was not included in TOS calculations. For Los Angeles, the 3rd quartile sample was 181% and 128% more toxic than the 1st quartile sample in summer and winter, respectively. Irvine had a similar result with 147% and 170% in summer and winter, respectively. Despite overall lower DBP concentrations, TOS response was highest for Los Angeles and Irvine in the winter. This was due to a slight increase in dBAN concentrations detected in the winter. DBAN concentrations in the summer for LA and IR ranged from 0.15–0.77 µg/L while winter concentrations ranged from 1.25–6.86 µg/L. *Thus, the slight increase in more potent unregulated compounds such as dBAN can have a significant impact on overall toxicity.*

The other regions had TOS response with medians ranging from 0.37–6,486 across both seasons. This was significantly lower than the medians for Los Angeles and Irvine which ranged from 8,876–102,960 across both seasons. Due to lower calculated TOS response, compounds driving toxicity were more diverse in the other regions and included the following: TCM, mono-HAAs, dCAA, BCAA, BdCAA, HANs, and IAA. These results support observed DBP sensitivity to HANs, mono-HAAs, and iodo-DBPs (Stalter et al., 2020, 2016) as well as confirm

their presence in California drinking water. While extrapolated chloroform was identified as a driving compound in some regions, the TOS response overall was insignificant compared to regions such as Los Angeles and Irvine. In addition, outliers represented as the maximum had a significant increase in TOS response in some regions. *This indicates that high spatial DBP variability also corresponds to high toxicity variability in the AREc32 breast cancer cell line.*
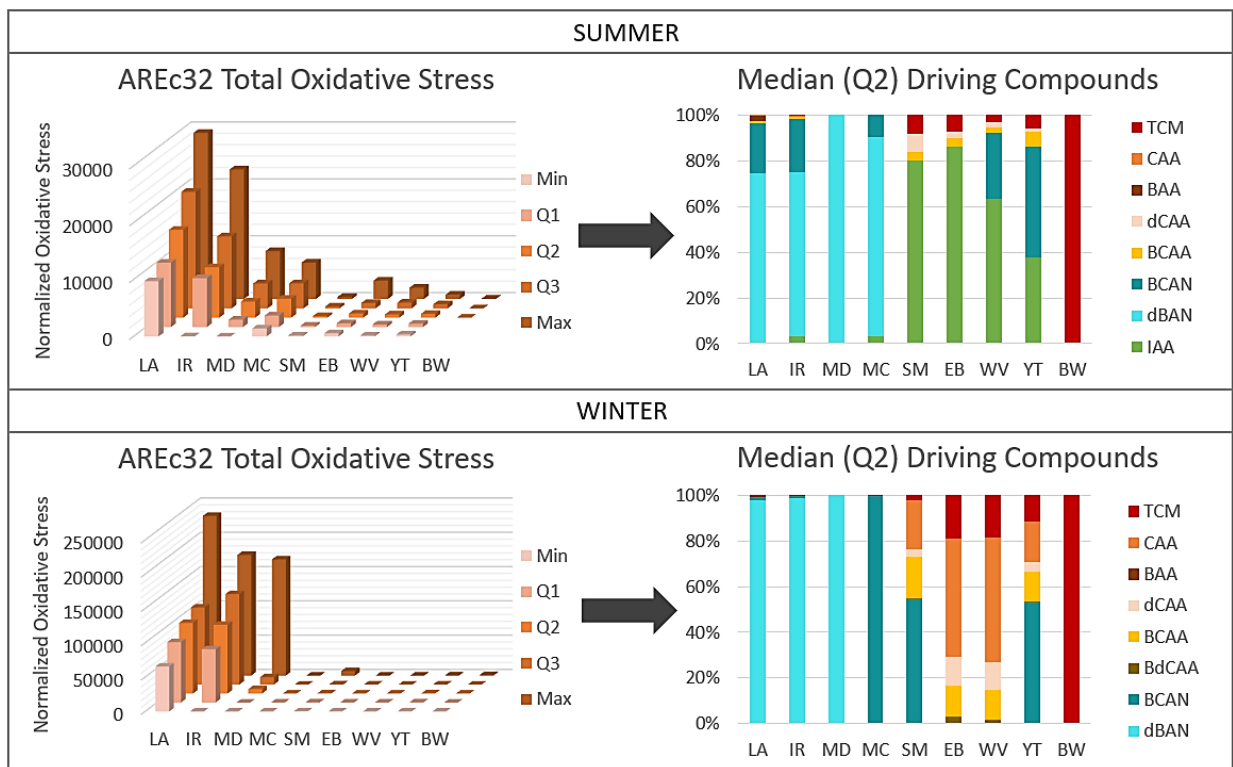
Figure 7. Estimated AREc32 Total Oxidative Stress Induction and Driving Compounds

4. CONCLUSION

The purpose of this study was to better understand disinfection by-product formation potential (DBP–FP) in California drinking water across different public water systems and source water types. DBP spatial and seasonal variability within each public system was also investigated as well as estimated total oxidative stress (TOS) induction in the AREc32 breast cancer cell line. Based on our results, Los Angeles and Irvine had the highest unregulated DBP–

FP concentrations and associated AREc32 calculated TOS with dBAN and BCAN driving toxicity. These results could be attributed to the complex nature of their water sources including high salinity water, brackish groundwater, and direct or indirect potable reuse of recycled water. While public water systems utilize such complex water sources to increase long-term water supply reliability and sustainability, the increase in DBP formation and speciation raises concern. Additional DBP-FP experiments on each individual water source could help to elucidate the role played by each water source in producing the observed DBP speciation in these regions. Recommendations and future directions for DBP mitigation at the treatment plant level and household level are provided in Section S4.

In addition, our results support the highly variable nature of DBPs within the distribution system as reported in the literature. Concentrations by DBP class ranged from ND to 430% of the regional average, suggesting that IDSE monitoring locations and locational running annual averages (LRAA) may not provide an accurate representation of household level exposure to DBPs. Outliers across both methods indicate hot spots or drops within the distribution system which corresponded to variations in AREc32 calculated TOS by orders of magnitude. This shows that high variability in DBP concentrations could have a drastic impact on overall potential toxicity.

While the AREc32 assay is highly sensitive to HANs, mono-HAAs, and iodo-DBPs (Statler 2020; Statler 2016), our results confirm the occurrence of these compounds in California drinking water and their role as drivers of toxicity in the MCF7 breast cancer cell line. While there are limitations with TOS estimation methods, namely the inability to account for unknown DBPs or mixture effects, the results of this study support the prioritization of dBAN, BCAN, and IAA for further research on long-term exposure and associated breast cancer risks.

Overall, the results of this study add to the discussion about whether regulatory approaches provide an accurate representation or effective control of DBP exposure considering the emergence of more toxic unregulated DBPs (Richardson and Plewa, 2020). With increasing climate change, water shortages, anthropogenic impacts on source waters, and use of recycled water, levels of DBP precursors (organic matter, iodide, bromide, and nitrogen) are likely to increase, exacerbating these issues. As drinking water treatment plants face challenges with balancing effective microbial disinfection and DBP formation mitigation, many are switching to alternative disinfectants such as chloramination which can increase the formation of more toxic iodo-DBPs and N-DBPs (Dong et al., 2019; Richardson, 2011; Richardson and Plewa, 2020). As these more toxic unregulated compounds are emerging in California drinking water, current regulatory methods may not be capturing household DBP exposure and toxicity.

REFERENCES

Alexandrou, L., Meehan, B.J., Jones, O.A.H., 2018. Regulated and emerging disinfection by-products in recycled waters. Sci. Total Environ. 637, 1607–1616. https://doi.org/10.1016/j.scitotenv.2018.04.391

Baribeau, H., 2006. Formation and decay of disinfection by-products in the distribution system. AwwA Research Foundation.

Bougeard, C.M.M., Goslan, E.H., Jefferson, B., Parsons, S.A., 2010. Comparison of the disinfection by-product formation potential of treated waters exposed to chlorine and monochloramine. Water Res. 44, 729–740. https://doi.org/10.1016/j.watres.2009.10.008

Charisiadis, P., Andra, S.S., Makris, K.C., Christophi, C.A., Skarlatos, D., Vamvakousis, V., Kargald, S., Stephanou, E.G., 2015. Spatial and seasonal variability of tap water disinfection by-products within distribution pipe networks. Sci. Total Environ. 506, 26–35. https://doi.org/10.1016/j.scitotenv.2014.10.071

Chen, B., Westerhoff, P., 2010. Predicting disinfection by-product formation potential in water. Water Res. 44, 3755–3762. https://doi.org/10.1016/j.watres.2010.04.009

Chowdhury, S., Rodriguez, M.J., Serodes, J., 2010. Model development for predicting changes in DBP exposure concentrations during indoor handling of tap water. Sci. Total Environ. 408, 4733–4743. https://doi.org/10.1016/j.scitotenv.2010.07.006

Chu, W., Fang, C., Deng, Y., Xu, Z., 2021. Intensified Disinfection Amid COVID-19 Pandemic Poses Potential Risks to Water Quality and Safety. Environ. Sci. Technol. 55, 4084–4086. https://doi.org/10.1021/acs.est.0c04394

Cuthbertson, A.A., Kimura, S.Y., Liberatore, H.K., Summers, R.S., Knappe, D.R.U., Stanford, B.D., Maness, J.C., Mulhern, R.E., Selbes, M., Richardson, S.D., 2019. Does Granular Activated Carbon with Chlorination Produce Safer Drinking Water? From Disinfection Byproducts and Total Organic Halogen to Calculated Toxicity. Environ. Sci. Technol. 53, 5987–5999. https://doi.org/10.1021/acs.est.9b00023

Dong, H., Qiang, Z., Richardson, S.D., 2019. Formation of Iodinated Disinfection Byproducts (I-DBPs) in Drinking Water: Emerging Concerns and Current Issues. Acc. Chem. Res. 52, 896–905. https://doi.org/10.1021/acs.accounts.8b00641

Drinking Water and Health,: Volume 2, 1980. . National Academies Press, Washington, D.C. https://doi.org/10.17226/1904

Escher, B.I., Dutt, M., Maylin, E., Tang, J.Y.M., Toze, S., Wolf, C.R., Lang, M., 2012. Water quality assessment using the AREc32 reporter gene assay indicative of the oxidative stress response pathway. J. Environ. Monit. 14, 2877–2885. https://doi.org/10.1039/c2em30506b

Farré, M.J., Gernjak, W., 2021. Disinfection byproducts in potable reuse, in: Comprehensive Analytical Chemistry. Elsevier, pp. 139–161. https://doi.org/10.1016/bs.coac.2021.01.005

Guilherme, S., Rodriguez, M.J., 2015. Short-term spatial and temporal variability of disinfection by-product occurrence in small drinking water systems. Sci. Total Environ. 518, 280–289. https://doi.org/10.1016/j.scitotenv.2015.02.069

Guilherme, S., Rodriguez, M.J., 2014. Occurrence of regulated and non-regulated disinfection by-products in small drinking water systems. Chemosphere 117, 425–432. https://doi.org/10.1016/j.chemosphere.2014.08.002

Krasner, S., IWA Publishing, Water Environment Research Foundation, 2008. Contribution of wastewater to DBP formation. Water Environment Research Foundation ; IWA Publishing, Alexandria, Va.; London.

Krasner, S.W., Weinberg, H.S., Richardson, S.D., Pastor, S.J., Chinn, R., Sclimenti, M.J., Onstad, G.D., Thruston, A.D., 2006. Occurrence of a New Generation of Disinfection Byproducts. Environ. Sci. Technol. 40, 7175–7185. https://doi.org/10.1021/es060353j

Krasner, S.W., Westerhoff, P., Chen, B., Rittmann, B.E., Nam, S.-N., Amy, G., 2009. Impact of Wastewater Treatment Processes on Organic Carbon, Organic Nitrogen, and DBP Precursors in Effluent Organic Matter. Environ. Sci. Technol. 43, 2911–2918. https://doi.org/10.1021/es802443t

Li, X.-F., Mitch, W.A., 2018. Drinking Water Disinfection Byproducts (DBPs) and Human Health Effects: Multidisciplinary Challenges and Opportunities. Environ. Sci. Technol. 52, 1681–1689. https://doi.org/10.1021/acs.est.7b05440

Melnick, R.L., Hooth, M.J., 2011. Carcinogenicity of Disinfection Byproducts in Laboratory Animals, in: Encyclopedia of Environmental Health. Elsevier, pp. 516–523. https://doi.org/10.1016/B978-0-444-52272-6.00099-4

Neale, P.A., Escher, B.I., 2019. In vitro bioassays to assess drinking water quality. Curr. Opin. Environ. Sci. Health 7, 1–7. https://doi.org/10.1016/j.coesh.2018.06.006

PHI and CBCRP, 2012. California Breast Cancer Mapping Project: Identifying Areas of Concern in California.

Plewa, M.J., Simmons, J.E., Richardson, S.D., Wagner, E.D., 2010. Mammalian Cell Cytotoxicity and Genotoxicity of the Haloacetic Acids, A Major Class of Drinking Water Disinfection By-Products. Environ. Mol. Mutagen. 51, 871–878. https://doi.org/10.1002/em.20585

Plewa, M.J., Wagner, E.D., 2011. Comparative Mammalian Cell Cytotoxicity and Genotoxicity, in: Encyclopedia of Environmental Health. Elsevier, pp. 806–812. https://doi.org/10.1016/B978-0-444-52272-6.00101-X

Plewa, M.J., Wagner, E.D., Muellner, M.G., Hsu, K.-M., Richardson, S.D., 2008. Comparative Mammalian Cell Toxicity of N-DBPs and C-DBPs, in: Disinfection By-Products in Drinking Water, ACS Symposium Series. American Chemical Society, pp. 36–50. https://doi.org/10.1021/bk-2008-0995.ch003

Plewa, M.J., Wagner, E.D., Richardson, S.D., 2017. TIC-Tox: A preliminary discussion on identifying the forcing agents of DBP-mediated toxicity of disinfected water. J. Environ. Sci. 58, 208–216. https://doi.org/10.1016/j.jes.2017.04.014

Priya, T., Mishra, B.K., Prasad, M.N.V., 2020. Physico-chemical techniques for the removal of disinfection by-products precursors from water, in: Disinfection By-Products in Drinking Water. Elsevier, pp. 23–58. https://doi.org/10.1016/B978-0-08-102977-0.00002-0

Richardson, S., Plewa, M., Wagner, E., Schoeny, R., Demarini, D., 2007. Occurrence, genotoxicity, and carcinogenicity of regulated and emerging disinfection by-products in drinking water: A review and roadmap for research. Mutat. Res. Mutat. Res. 636, 178–242. https://doi.org/10.1016/j.mrrev.2007.09.001

Richardson, S.D., 2011. Disinfection By-Products: Formation and Occurrence in Drinking Water★★This article has been reviewed in accordance with the US EPA's peer and administrative review policies and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use by the US EPA., in: Encyclopedia of Environmental Health. Elsevier, pp. 110–136. https://doi.org/10.1016/B978-0-444-52272-6.00276-2

Richardson, S.D., Plewa, M.J., 2020. To regulate or not to regulate? What to do with more toxic disinfection by-products? J. Environ. Chem. Eng. 8, 103939. https://doi.org/10.1016/j.jece.2020.103939

Shanks, C.M., Serodes, J.-B., Rodriguez, M.J., 2013. Spatio-temporal variability of non-regulated disinfection by-products within a drinking water distribution network. Water Res. 47, 3231–3243. https://doi.org/10.1016/j.watres.2013.03.033

Stalter, D., O'Malley, E., von Gunten, U., Escher, B.I., 2020. Mixture effects of drinking water disinfection by-products: implications for risk assessment. Environ. Sci. Water Res. Technol. 6, 2341–2351. https://doi.org/10.1039/C9EW00988D

Stalter, D., O'Malley, E., von Gunten, U., Escher, B.I., 2016. Fingerprinting the reactive toxicity pathways of 50 drinking water disinfection by-products. Water Res. 91, 19–30. https://doi.org/10.1016/j.watres.2015.12.047

Tardiff, R.G., Carson, M.L., Ginevan, M.E., 2006. Updated weight of evidence for an association between adverse reproductive and developmental effects and exposure to disinfection by-products. Regul. Toxicol. Pharmacol. 45, 185–205. https://doi.org/10.1016/j.yrtph.2006.03.001

Tuttle, M.L., Grauch, R.I., 2009. Salinization of the Upper Colorado River—Fingerprinting Geologic Salt Sources. U.S. Geological Survey Scientific Investigations Report 2009–5072.

USEPA, 2005. Fact Sheet: Stage 2 Disinfectants and Disinfection Byproducts Rule.

USEPA, 2003. Determination of Haloacetic Acids and Dalapon in Drinking Water by Liquid-Liquid Microextraction, Derivatization, and Gas Chromatography with Electron Capture Detection.

USEPA, 1995. Analytical Methods Approved for Drinking Water Compliance Monitoring under the Disinfection Byproduct Rules. U. S. Environ. Prot. Agency 28.

Villanueva, C.M., Cordier, S., Font-Ribera, L., Salas, L.A., Levallois, P., 2015. Overview of Disinfection By-products and Associated Health Effects. Curr. Environ. Health Rep. 2, 107–115. https://doi.org/10.1007/s40572-014-0032-x

Wei, J., Ye, B., Wang, W., Yang, L., Tao, J., Hang, Z., 2010. Spatial and temporal evaluations of disinfection by-products in drinking water distribution systems in Beijing, China. Sci. Total Environ. 408, 4600–4606. https://doi.org/10.1016/j.scitotenv.2010.06.053

WHO, 2005. Trihalomethanes in Drinking-Water.

Xiao, R., Duan, Y., Chu, W., 2020. The effectiveness of household water treatment and safe storage in improving drinking water quality: a disinfection by-product (DBP) perspective. J. Water Supply Res. Technol.-Aqua 69, 785–806. https://doi.org/10.2166/aqua.2020.052

Table S1: Targeted Disinfection By-Product Compounds

Class	Compound	Abv.	CAS no.	SMILES	Vendor	Purity
THM$_4$	Trichloromethane	TCM	67–66–3	C(Cl)(Cl)Cl	EPA 551A Mix, Sigma[a]	–
	Bromodichloromethane	BdCM	75–27–4	C(Cl)(Cl)Br	EPA 551A Mix, Sigma[a]	–
	Dibromochloromethane	dBCM	124–48–1	C(Cl)(Br)Br	EPA 551A Mix, Sigma[a]	–
	Tribromomethane	TBM	75–25–2	C(Br)(Br)Br	EPA 551A Mix, Sigma[a]	–
THM$_{UR}$	Bromochloroiodomethane	BCIM	34970–00–8	C(Cl)(Br)I	TRC[b]	97.0%
	Chlorodiiodomethane	CdIM	638–73–3	C(Cl)(I)I	TRC[b]	95.0%
HAA$_5$	Chloroacetic Acid	CAA	79–11–8	C(C(=O)O)Cl	EPA 552.2 Mix, Sigma[a]	–
	Dichloroacetic Acid	dCAA	79–43–6	C(C(=O)O)(Cl)Cl	EPA 552.2 Mix, Sigma[a]	–
	Trichloroacetic Acid	tCAA	76–03–9	C(=O)(C(Cl)(Cl)Cl)O	EPA 552.2 Mix, Sigma[a]	–
	Bromoacetic Acid	BAA	79–08–3	C(C(=O)O)Br	EPA 552.2 Mix, Sigma[a]	–
	Dibromoacetic Acid	dBAA	631–64–1	C(C(=O)O)(Br)Br	EPA 552.2 Mix, Sigma[a]	–
HAA$_{UR}$	Bromochloroacetic Acid	BCAA	5589–96–8	C(C(=O)O)(Cl)Br	EPA 552.2 Mix, Sigma[a]	–
	Bromodichloroacetic Acid	BdCAA	71133–14–7	C(=O)(C(Cl)(Cl)Br)O	EPA 552.2 Mix, Sigma[a]	–
	Chlorodibromoacetic Acid	CdBAA	5278–95–5	C(=O)(C(Cl)(Br)Br)O	EPA 552.2 Mix, Sigma[a]	–
	Tribromoacetic Acid	tBAA	75–96–7	C(=O)(C(Br)(Br)Br)O	EPA 552.2 Mix, Sigma[a]	–
	Iodoacetic Acid	IAA	64–69–7	C(C(=O)O)I	TRC[b]	98.0%
	Chloroiodoacetic Acid	CIAA	53715–09–6	C(C(=O)O)(Cl)I	TRC[b]	95.0%
HAN	Bromoacetonitrile	BAN	590–17–0	C(C#N)Br	TRC[b]	98.0%
	Bromochloroacetonitrile	BCAN	83463–62–1	C(#N)C(Cl)Br	TRC[b]	90.0%
	Dibromoacetonitrile	dBAN	3252–43–5	C(#N)C(Br)Br	TRC[b]	96.3%
	Iodoacetonitrile	IAN	624–75–9	C(C#N)I	TRC[b]	–
Other	1,2,3–trichloropropane	TCP	96–18–4	C(C(CCl)Cl)Cl	Sigma[a]	99.8%
	1,2–dibromo–3–chloropropane	DBCP	96–12–8	C(C(CBr)Br)Cl	EPA 551A Mix, Sigma[a]	–
	1,1,2–trichloroethane	1,1,2–TCE	79–00–5	C(C(Cl)Cl)Cl	Sigma[a]	99.8%
	1,2–dibromoethane	EDB	106–93–4	C(CBr)Br	EPA 551A Mix, Sigma[a]	–
	Tetrachloroethylene	PCE	127–18–4	C(=C(Cl)Cl)(Cl)Cl	EPA 551A Mix, Sigma[a]	–
	1,1,1–trichloroethane	1,1,1–TCE	71–55–6	CC(Cl)(Cl)Cl	EPA 551A Mix, Sigma[a]	–
	Carbon Tetrachloride	CT	56–23–5	C(Cl)(Cl)(Cl)Cl	EPA 551A Mix, Sigma[a]	–
	Trichloroethylene	TCE	79–01–6	C(=C(Cl)Cl)Cl	EPA 551A Mix, Sigma[a]	–

[a] purchased from Sigma–Aldrich
[b] purchased from Toronto Research Chemicals

Section S1: Overview of Studied Public Water Systems and Bottled Water Brands

Table S2: Overview of Public Water Systems and 2020 Consumer Confidence Reports

SOURCE WATER TYPE	REGION	WATER SYSTEM	POPULATION SERVED	DISINFECTION TYPE	CONSUMER CONFIDENCE REPORTS
Large Surface Water	San Mateo	California Water Services, San Mateo	101,004	Chloramination	2020
	East Bay	East Bay Municipal Utility District	1,379,000	Chloramination	2020
Small Surface Water	Weaverville	Weaverville C. S. D.	3,554	Chlorination	2020
	Yurok	Yurok Tribal Environmental Program	< 1,000	Chlorination	2020
Mixed Water	Los Angeles	LA City Department of Water and Power	4,072,307	Chloramination	2020
	Irvine	Irvine Ranch Water District	450,526	Chloramination	2020
Groundwater	Madera	City of Madera	66,082	Chlorination	2020
	Merced	City of Merced	86,750	Chlorination	2020

Figure S1: Sampling Locations for Studied Public Water Systems
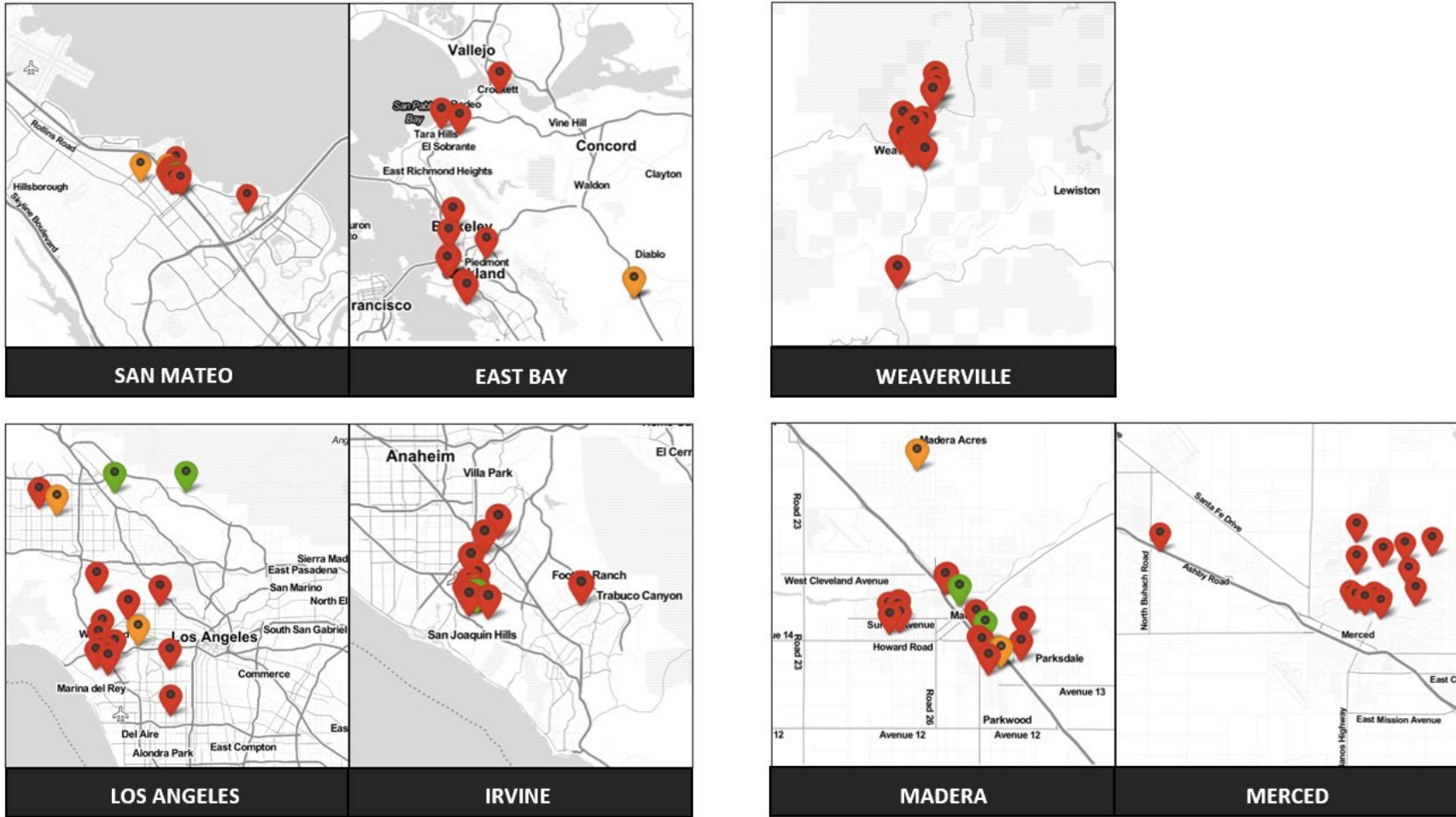
Note: The colors of sampling location icon indicate samples included in both winter and summer sampling (red), winter sampling only (green), and summer sampling only (orange). Differing locations are a result of changes in household participation. Locations of Yurok Tribal Environmental Program samples are not shown.

Table S3: Overview of Studied Bottled Water Brands and Local Refill Stations

NO.	BRAND	TYPE	WATER SOURCE	FILTRATION	REVERSE OSMOSIS	DISTILLATION	DISINFECTION	WQR[a]
1	Dasani	Bottled	Purified	✓	✓			2019
2	Nestle	Bottled	Purified, Spring	✓	✓	✓	✓	2020
3	Arrowhead	Bottled	Spring		✓	✓		2020
4	Kirkland[b]	Bottled	Purified	✓	✓		✓	2020
5	Crystal Geyser	Bottled	Spring	✓			✓	2020
6	Aquafina	Bottled	Purified	✓	✓		✓	2019
7	Smart Water	Bottled	Purified			✓		2018
8	Boxed Water is Better	Boxed	Purified	✓	✓		✓	2018
9	Just Water	Boxed	Spring	✓				2020
10	Ever and Ever	Canned	Purified		✓			N/R[e]
11	Pathwater	Canned	Purified	✓	✓		✓	N/R[e]
12	H20 to Go	Refill	Purified	✓	✓	✓	✓	Davis 2020
13	Safeway[c]	Refill	Purified	✓	✓		✓	Davis 2020
14	Davis Food Co–op[d]	Refill	Purified		✓	✓		Davis 2020
15	Nugget[c]	Refill	Purified	✓	✓		✓	Davis 2020

[a] *Water quality reports (WQR) for most recently reported year*
[b] *Costco's Kirkland signature water is sourced from Niagara*
[c] *Refill Stations include Primo self–serve refill water dispensers*
[d] *Refill Stations include Fresh Pure Waters self–serve refill water dispensers*
[e] *Water quality report not reported or found*

Section S2: Analytical Methods for GC–ECD and GC–MS

Section S2.1: Overview of GC–ECD Methods

```
===============================================================================
                              6890 GC METHOD
===============================================================================


OVEN
   Initial temp:  40 'C (On)            Maximum temp:  325 'C
   Initial time:  10.00 min             Equilibration time:  3.00 min
   Ramps:
      #  Rate   Final temp  Final time
      1  2.50        65        0.00
      2 10.00        85        0.00
      3 20.00       205        7.00
      4  0.0(Off)
   Post temp:  100 'C
   Post time:  0.00 min
   Run time:  35.00 min

FRONT INLET (SPLIT/SPLITLESS)           BACK INLET (VOLATILES)
   Mode:  Splitless                        Mode:  Split
   Initial temp:  210 'C (On)              Initial temp:  50 'C (Off)
   Pressure:  9.17 psi (On)                Pressure:  0.00 psi (Off)
   Purge flow:  30.0 mL/min                Total flow:  45.0 mL/min
   Purge time:  0.75 min                   Gas saver:  Off
   Total flow:  33.6 mL/min                Gas type:  Helium
   Gas saver:  Off
   Gas type:  Helium

COLUMN 1                                COLUMN 2
   Capillary Column                        (not installed)
   Model Number:  Agilent 222-0732LTM
   DB-1701 (G3900-63003)
   Max temperature:  300 'C
   Nominal length:  30.0 m
   Nominal diameter:  250.00 um
   Nominal film thickness:  0.25 um
   Mode:  constant pressure
   Pressure:  9.17 psi
   Nominal initial flow:  0.7 mL/min
   Average velocity:  20 cm/sec
   Inlet:  Front Inlet
   Outlet:  Back Detector
   Outlet pressure:  ambient

FRONT DETECTOR (FID)                    BACK DETECTOR (µECD)
   Temperature:  250 'C (Off)              Temperature:  250 'C (On)
   Hydrogen flow:  40.0 mL/min (Off)       Mode:  Constant column+makeup flow
   Air flow:  450.0 mL/min (Off)           Combined flow:  20.0 mL/min
   Mode:  Constant makeup flow             Makeup flow:  On
   Makeup flow:  45.0 mL/min (Off)         Makeup Gas Type: Argon methane 5%
   Makeup Gas Type: Nitrogen               Electrometer:  On
   Flame:  Off
```

```
   Electrometer:  Off
   Lit offset:  2.0


SIGNAL 1                                  SIGNAL 2
   Data rate:  20 Hz                         Data rate:  20 Hz
   Type:  back detector                      Type:  front detector
   Save Data:  On                            Save Data:  Off
   Zero:  0.0 (Off)                          Zero:  0.0 (Off)
   Range:  0                                 Range:  0
   Fast Peaks:  Off                          Fast Peaks:  Off
   Attenuation:  0                           Attenuation:  0


COLUMN COMP 1                             COLUMN COMP 2
   Derive from back detector                 Derive from back detector

AUX PRESSURE 3                            AUX PRESSURE 4
   Description:                              Description:
   Gas Type:  Helium                         Gas Type:  Helium
   Initial pressure:  0.00 psi (Off)         Initial pressure:  0.00 psi (Off)

AUX PRESSURE 5
   Description:
   Gas Type:  Helium
   Initial pressure:  0.00 psi (Off)


                                          POST RUN
                                             Post Time: 0.00 min


TIME TABLE
   Time        Specifier                    Parameter & Setpoint

                            GC Injector
      Front Injector:
         Sample Washes              2
         Sample Pumps               3
         Injection Volume        1.00 microliters
         Syringe Size             5.0 microliters
         PreInj Solvent A Washes    2
         PreInj Solvent B Washes    2
         PostInj Solvent A Washes   2
         PostInj Solvent B Washes   2
         Viscosity Delay            0 seconds
         Plunger Speed           Fast
         PreInjection Dwell      0.00 minutes
         PostInjection Dwell     0.00 minutes

      Back Injector:
No parameters specified
```

36

Section S2.2: Overview of GC–MS Methods

```
================================================================================
                              6890 GC METHOD
================================================================================


OVEN                                    COLUMN 2
Equilibration time:  0.50 min           (not installed)
Maximum temp:  260 C
Initial temp:  45 C (On)                FRONT DETECTOR (NO DET)
Initial time:  3.00 min
Ramps:                                  BACK DETECTOR (NO DET)
#   Rate  Final temp  Final time
1  10.00      250         5.00          SIGNAL 1
2   0 (Off)                             Save Data:  Off
Post temp:  0 C
Post time:  0.00 min                    SIGNAL 2
Run time:  28.50 min                    Save Data:  Off


                                        THERMAL AUX 2
FRONT INLET (SPLIT/SPLITLESS)           Use:  MSD Transfer Line Heater
Mode:  Splitless                        Initial temp:  280 C (On)
Initial temp:  250 C (Off)
Pressure:  7.3 psi (Off)                POST RUN
Purge flow:  50.0 mL/min                Post Time: 0.00 min
Purge time:  2.00 min
Total flow:  53.7 mL/min                INJECTOR 1
Gas saver:  On                          Solvent Wash Mode:  A, B
Saver flow:  20.0 mL/min                Waste Bottle Use: A Only
Saver time:  2.00 min                   Sample Volume (uL):  2.000
Gas type:  Helium                       Syringe size (uL):      5.0
                                        Pre washes from bottle A:  2
BACK INLET (SPLIT/SPLITLESS)            Pre washes from bottle B:  2
Mode:  Split                            Post washes from bottle A:  2
Initial temp:  140 C (On)               Post washes from bottle B:  2
Pressure:  0.0 psi (Off)                Viscosity delay (seconds):  0
Total flow:  45.0 mL/min                Pre injection dwell (min):  0.00
Gas saver:  Off                         Post injection dwell (min):  0.00
Gas type:  Helium                       Sample skim depth (mm):    0.0
                                        (Off)
COLUMN 1                                Plunger Speed:  Fast
Capillary Column                        Solvent saver:  Off
Max temperature:  320 C
Nominal length:  30.0 m
Nominal diameter:  250.00 um            ALS ERRORS:
Nominal film thickness:  0.25 um        On missing vial: pause
Mode:  constant flow
Initial flow:  1.0 mL/min               TIME TABLE
Nominal init pressure:  7.3 psi         Time(min)      Parameter & Setpoint
Average velocity:  36 cm/sec
Inlet:  Front Inlet
Outlet:  MSD                            Column 1 Inventory Number :
Outlet pressure:  vacuum                Column 2 Inventory Number :



MS ACQUISITION PARAMETERS
```

```
General Information
------- -----------
Tune File               : atune.u
Acquistion Mode         : Scan

MS Information
-- -----------

Solvent Delay           : 0.00 min

EMV Mode                : Relative
Relative Voltage        : 0
Resulting EM Voltage     : 1812

[Scan Parameters]

Low Mass                : 35.0
High Mass               : 300.0
Threshold               : 0
Sample #                : 2         A/D Samples   4
Plot 2 low mass         : 33.0
Plot 2 high mass        : 300.0

[MSZones]

MS Source               : 230 C   maximum 250 C
MS Quad                 : 150 C   maximum 200 C

END OF MS ACQUISITION PARAMETERS

TUNE PARAMETERS for SN: US02080150
----------------------------

Trace Ion Detection is OFF.

EMISSION    :       34.610
ENERGY      :       69.922
REPELLER    :       29.955
IONFOCUS    :       90.157
ENTRANCE_LE :        0.000
EMVOLTS     :     1811.765
Actual EMV  :     1811.77
GAIN FACTOR :        2.07
AMUGAIN     :     2275.000
AMUOFFSET   :      126.000
FILAMENT    :        1.000
DCPOLARITY  :        0.000
ENTLENSOFFS :       25.098
MASSGAIN    :      251.000
MASSOFFSET  :      -10.000

END OF TUNE PARAMETERS
------------------------------------
END OF INSTRUMENT CONTROL PARAMETERS
------------------------------------
```

Table S4: GC–ECD Retention Times (RTs)

Class	Compound	Abv.	CAS no.	RT – Winter (min)	RT – Summer (min)
HAA$_5$	Chloroacetic Acid	CAA	79–11–8	5.37	5.37
	Dichloroacetic Acid	dCAA	79–43–6	20.75	20.82
	Trichloroacetic Acid	tCAA	76–03–9	23.29	23.33
	Bromoacetic Acid	BAA	79–08–3	19.97	20.05
	Dibromoacetic Acid	dBAA	631–64–1	26.29	26.32
HAA$_{UR}$	Bromochloroacetic Acid	BCAA	5589–96–8	24.36	24.40
	Bromodichloroacetic Acid	BdCAA	71133–14–7	25.95	25.99
	Chlorodibromoacetic Acid	CdBAA	5278–95–5	27.72	27.75
	Tribromoacetic Acid	tBAA	75–96–7	29.13	29.17
	Iodoacetic Acid	IAA	64–69–7	29.83	29.87
	Chloroiodoacetic Acid	CIAA	53715–09–6	27.07	27.11

Note: Reported average RTs had RSD ranging from 0.003% – 0.18% across both seasons

Table S5: GC–MS Retention Times (RTs), Quantifiers, and Qualifiers

Class	Compound	Abv.	CAS no.	RT – Winter (min)	RT – Summer (min)	Quantifier (m/z)	Qualifiers (m/z)
THM$_4$	Trichloromethane	TCM	67–66–3	8.15	8.15	83	85, 47, 87
	Bromodichloromethane	BdCM	75–27–4	10.41	10.50	83	85, 129, 87
	Dibromochloromethane	dBCM	124–48–1	12.79	12.85	129	127, 131, 48
	Tribromomethane	TBM	75–25–2	15.08	15.18	173	171, 175, 91
THM$_{UR}$	Bromochloroiodomethane	BCIM	34970–00–8	15.78	15.84	127	129, 131, 175
	Chlorodiiodomethane	CdIM	638–73–3	18.66	18.64	175	127, 177, 302
HAN	Bromoacetonitrile	BAN	590–17–0	11.60	11.77	119	121, 40, 79
	Bromochloroacetonitrile	BCAN	83463–62–1	12.81	12.86	74	76, 155, 153
	Dibromoacetonitrile	dBAN	3252–43–5	15.24	15.32	120	118, 199, 79
	Iodoacetonitrile	IAN	624–75–9	14.87	14.90	167	127
Other	1,2,3–trichloropropane	TCP	96–18–4	15.71	15.79	75	110, 77, 112
	1,2–dibromo–3–chloropropane	DBCP	96–12–8	19.48	19.55	157	75, 155, 159
	1,1,2–trichloroethane	1,1,2–TCE	79–00–5	12.07	12.13	97	83, 99, 85
	1,2–dibromoethane	EDB	106–93–4	13.14	13.19	107	109, 81, 79
	Tetrachloroethylene	PCE	127–18–4	13.34	13.43	166	164, 129, 131
	1,1,1–trichloroethane	1,1,1–TCE	71–55–6	9.10	9.10	97	99, 61, 117
	Carbon Tetrachloride	CT	56–23–5	9.54	9.54	117	119, 121, 82
	Trichloroethylene	TCE	79–01–6	10.50	10.42	130	132, 95, 97

Table S6: LODs, LOQs, Cal Curve Accuracies, and Quality Control RSDs

Class	Compound	Abv.	Winter				Summer			
			LOD (ppb)	LOQ (ppb)	Cal Accuracy (%)	QC RSD (%)	LOD (ppb)	LOQ (ppb)	Cal Accuracy (%)	QC RSD (%)
THM$_4$	Trichloromethane	TCM	0.025	0.25	109 ± 20	N/A[a]	0.1	0.25	101 ± 34	12.8
	Bromodichloromethane	BdCM	0.025	0.1	114 ± 28	18.7	0.05	0.1	110 ± 26	30.5
	Dibromochloromethane	dBCM	0.01	0.1	103 ± 13	17.2	0.05	0.1	103 ± 13	22.6
	Tribromomethane	TBM	0.01	0.5	111 ± 27	23.0	0.05	0.1	103 ± 31	6.1
THM$_{UR}$	Bromochloroiodomethane	BCIM	0.05	0.1	110 ± 33	17.2	0.01	0.025	99 ± 36	11.4
	Chlorodiiodomethane	CdIM	0.1	0.25	93 ± 27	20.0	0.1	0.25	90 ± 16	18.5
HAA$_5$	Chloroacetic Acid	CAA	0.5	1	114 ± 17	62.1	0.5	1	117 ± 20	30.7
	Dichloroacetic Acid	dCAA	0.1	0.25	106 ± 11	21.8	0.1	1	111 ± 15	11.1
	Trichloroacetic Acid	tCAA	0.1	0.25	92 ± 13	15.5	0.05	0.1	100 ± 12	8.1
	Bromoacetic Acid	BAA	0.25	0.25	100 ± 8	16.6	0.1	0.25	111 ± 15	6.6
	Dibromoacetic Acid	dBAA	0.025	0.1	80 ± 19	19.5	0.01	0.1	99 ± 8	6.8
HAA$_{UR}$	Bromochloroacetic Acid	BCAA	0.25	0.25	97 ± 14	20.9	0.25	0.5	114 ± 16	10.4
	Bromodichloroacetic Acid	BdCAA	0.25	1	89 ± 24	11.6	0.05	0.1	81 ± 19	3.9
	Chlorodibromoacetic Acid	CdBAA	0.1	0.25	86 ± 19	44.4	0.1	0.25	85 ± 18	8.9
	Tribromoacetic Acid	tBAA	0.5	1	92 ± 16	36.8	0.5	1	95 ± 16	13.2
	Iodoacetic Acid	IAA	0.5	1	101 ± 14	24.3	0.05	0.1	92 ± 13	6.4
	Chloroiodoacetic Acid	CIAA	0.05	0.1	98 ± 16	39.4	0.05	0.1	101 ± 14	6.1
HAN	Bromoacetonitrile	BAN	0.5	1	110 ± 39	25.0	0.1	0.5	107 ± 28	14.5
	Bromochloroacetonitrile	BCAN	0.025	0.1	102 ± 20	24.4	0.025	0.1	101 ± 20	40.7
	Dibromoacetonitrile	dBAN	0.05	1	107 ± 26	20.0	0.025	0.1	98 ± 39	42.5
	Iodoacetonitrile	IAN	0.5	1	105 ± 24	20.4	0.5	1	95 ± 33	19.8
Other	1,2,3–trichloropropane	TCP	0.25	0.5	111 ± 25	19.4	0.1	0.25	108 ± 15	9.8
	1,2–dibromo–3–chloropropane	DBCP	0.5	1	93 ± 23	20.4	0.25	0.5	95 ± 18	10.2
	1,1,2–trichloroethane	1,1,2–TCE	0.5	1	123 ± 39	18.8	0.1	0.25	115 ± 20	12.2
	1,2–dibromoethane	EDB	0.5	0.5	107 ± 25	18.3	0.025	0.05	115 ± 17	8.6
	Tetrachloroethylene	PCE	0.1	0.5	89 ± 31	13.1	2.5	5	100 ± 19	6.3
	1,1,1–trichloroethane	1,1,1–TCE	0.01	0.1	108 ± 16	N/A[a]	0.01	0.025	110 ± 18	54.4
	Carbon Tetrachloride	CT	0.01	0.1	97 ± 19	N/A[a]	0.025	0.1	97 ± 19	57.2
	Trichloroethylene	TCE	0.25	0.5	97 ± 37	19.1	0.1	0.25	108 ± 12	16.1

[a] Compounds were under solvent peak for winter sampling but later verified during summer sampling

Section S2.3: Description of AREc32 Reporter Gene Bioassay

The AREc32 reporter gene assay is emerging as a useful tool in water quality monitoring and signifies the induction of the Nrf2 mediated oxidative stress response in the MCF7 human breast cancer cell line (Escher et al., 2012). The AREc32 bioassay was developed by Wang et at. (2006), validated by Escher et al. (2012), and modified to a headspace–free setup to avoid volatilization of DBPs by Stalter et al. (2013). The bioassay is based on the MCF7 human breast cancer cell line where eight copies of the antioxidant response element (ARE) are linked to a luciferase reporter gene (Escher et al., 2012). The production of luciferase measured by bioluminescence indicates ARE activation and presence of chemical stressor (Stalter et al., 2016). Reported effect concentrations (EC$_{IR1.5}$) as shown in Table S7 represent the molar concentrations at which individual DBPs induce a 50% effect increase relative to the control (Stalter et al., 2016). It should be noted that there are some limitations with using individual EC$_{IR1.5}$ values, namely the inability to account for mixture or other matrix effects.

Table S7: Compound Effect Concentrations (EC$_{IR1.5}$) and Molecular Weights (MW). The colors signify effect concentrations from least potent (yellow) to most potent (red orange)

Class	Compound	Abv.	CAS no.	MW (g/mol)	EC$_{IR1.5}$ (mol/L)
THM$_4$	Trichloromethane	TCM	67–66–3	119.37	1.40E–02
	Bromodichloromethane	BdCM	75–27–4	163.83	6.10E–03
	Dibromochloromethane	dBCM	124–48–1	208.28	1.90E–03
	Tribromomethane	TBM	75–25–2	252.73	1.40E–03
THM$_{UR}$	Bromochloroiodomethane	BCIM	34970–00–8	255.28	1.20E–04
	Chlorodiiodomethane	CdIM	638–73–3	302.28	2.70E–05
HAA$_5$	Chloroacetic Acid	CAA	79–11–8	94.50	2.70E–04
	Dichloroacetic Acid	dCAA	79–43–6	128.94	6.00E–03
	Trichloroacetic Acid	tCAA	76–03–9	163.38	n.e.≤2E–2[a]
	Bromoacetic Acid	BAA	79–08–3	138.95	5.20E–06
	Dibromoacetic Acid	dBAA	631–64–1	217.84	1.20E–04
HAA$_{UR}$	Bromochloroacetic Acid	BCAA	5589–96–8	173.39	1.40E–04
	Bromodichloroacetic Acid	BdCAA	71133–14–7	207.83	2.00E–03
	Chlorodibromoacetic Acid	dBCAA	5278–95–5	252.29	4.90E–03
	Tribromoacetic Acid	tBAA	75–96–7	296.74	4.40E–04
	Iodoacetic Acid	IAA	64–69–7	185.95	3.60E–06
	Chloroiodoacetic Acid	CIAA	53715–09–6	220.39	2.20E–05
HAN	Bromoacetonitrile	BAN	590–17–0	119.95	n.t.[b]
	Bromochloroacetonitrile	BCAN	83463–62–1	154.39	2.20E–06
	Dibromoacetonitrile	dBAN	3252–43–5	198.84	1.50E–07
	Iodoacetonitrile	IAN	624–75–9	166.95	n.t.[b]
Other	1,2,3–trichloropropane	TCP	96–18–4	147.43	n.t.[b]
	1,2–dibromo–3–chloropropane	DBCP	96–12–8	236.33	n.t.[b]
	1,1,2–trichloroethane	1,1,2–TCE	79–00–5	133.40	n.t.[b]
	1,2–dibromoethane	EDB	106–93–4	187.86	n.t.[b]
	Tetrachloroethylene	PERC	127–18–4	165.80	n.t.[b]
	1,1,1–trichloroethane	1,1,1–TCE	71–55–6	133.40	n.t.[b]
	Carbon Tetrachloride	CT	56–23–5	153.80	n.t.[b]
	Trichloroethylene	TCE	79–01–6	131.38	n.t.[b]

[a] *no effect observed at highest concentration tested*
[b] *compound not tested or included* in (Stalter et al., 2016) *study*

Section S3: Additional Information for Results and Discussion

Table S8: Overview of Detection Frequencies. The colors signify low detection frequency (yellow) to high detection frequency (red orange)

Class	Abv.	Winter Detection Frequency (%)									Summer Detection Frequency (%)								
		SM	EB	WV	YT	LA	IR	MD	MC	BW	SM	EB	WV	YT	LA	IR	MD	MC	BW
THM$_4$	TCM	100	100	100	100	100	100	13	100	60	100	100	100	100	100	100	13	93	60
	BdCM	100	100	100	100	100	100	13	100	47	100	100	100	100	100	100	13	100	33
	dBCM	100	0	0	100	100	93	81	100	40	0	93	40	100	100	100	80	100	27
	TBM	100	0	0	0	100	93	94	100	13	0	13	0	0	100	93	80	100	13
THM$_{UR}$	BCIM	100	0	0	0	100	87	0	0	0	0	0	0	0	100	93	0	0	0
	CdIM	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
HAA$_5$	CAA	100	100	100	100	100	60	0	0	0	0	20	0	0	100	73	0	0	0
	dCAA	100	100	100	100	100	87	0	87	7	100	100	100	100	100	100	0	73	7
	tCAA	100	100	100	100	100	87	0	0	27	100	100	100	100	100	100	0	13	7
	BAA	0	0	0	0	100	53	0	0	7	0	13	7	0	100	13	0	0	7
	dBAA	100	47	29	100	100	87	81	100	13	100	100	100	100	100	100	80	100	33
HAA$_{UR}$	BCAA	100	100	57	100	100	87	0	7	7	100	100	53	100	100	93	0	33	0
	BdCAA	93	100	100	100	100	87	0	53	7	100	100	100	100	100	93	0	0	0
	CdBAA	53	0	0	29	93	87	13	0	0	0	20	20	0	100	93	20	13	0
	tBAA	0	0	0	0	0	33	0	0	0	0	0	27	0	21	0	7	0	0
	IAA	7	0	0	0	0	0	13	0	0	100	100	93	87	7	93	0	87	0
	CIAA	100	0	0	0	0	20	0	0	0	0	0	0	0	0	0	0	0	0
HAN	BAN	0	0	0	0	0	0	0	0	0	40	0	0	0	100	60	0	7	0
	BCAN	100	0	0	100	100	87	19	80	0	0	20	100	100	100	93	40	100	7
	dBAN	0	0	0	0	100	87	69	7	0	7	20	27	0	100	93	73	100	0
	IAN	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other	TCP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	DBCP	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	1,1,2–TCE	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	EDB	0	0	0	0	0	0	0	0	0	7	0	0	0	0	0	0	0	0
	PCE	20	13	0	0	13	7	63	0	7	0	0	0	0	0	0	0	0	0
	1,1,1–TCE	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	CT	27	87	0	14	60	100	44	0	40	0	73	7	93	7	0	20	0	0
	TCE	7	0	0	0	0	0	13	0	0	7	0	0	0	7	0	13	7	0

Table S9: DBP–FP Interquartile Ranges (IQRs) (µg/L)

SUMMER

DBP Class	SM	EB	WV	YT	LA	IR	MD	MC
TCM	22.20	17.50	19.76	12.16	3.94	7.35	< LOD	0.94
THM$_3$	0.27	0.76	2.05	0.61	1.56	4.34	0.78	0.55
THM$_{ur}$	< LOD	< LOD	< LOD	< LOD	0.12	0.21	< LOD	< LOD
HAA$_5$	2.01	4.24	5.95	1.53	1.75	1.28	0.13	0.08
HAA$_{ur}$	0.13	0.32	1.05	0.12	1.17	0.26	< LOD	0.03
HAN	< LOD	< LOD	0.16	0.04	0.87	0.38	0.09	0.16
Total	20.22	17.99	21.95	10.86	8.96	12.93	0.89	1.33

WINTER

DBP Class	SM	EB	WV	YT	LA	IR	MD	MC
TCM	2.97	1.95	5.46	10.18	0.94	1.71	< LOD	0.27
THM$_3$	0.21	0.03	0.07	0.22	1.14	1.62	1.25	0.36
THM$_{ur}$	0.03	< LOD	< LOD	< LOD	0.32	0.24	< LOD	< LOD
HAA$_5$	1.12	3.29	8.70	2.57	2.24	1.67	0.08	0.11
HAA$_{ur}$	0.21	0.10	0.31	0.32	1.30	1.45	< LOD	< LOD
HAN	0.01	< LOD	< LOD	0.01	0.80	1.73	0.32	0.01
Total	1.97	4.60	10.06	11.71	4.49	6.53	2.00	0.79

Section S4: Recommendations

The following section provides recommendations, treatments strategies, and future directions for mitigating DBP formation in California drinking water:

Section S4.1: Drinking Water Treatment Plant Level

Section S4.1.1: Precursor Control and Removal

One way to mitigate DBP formation includes reducing precursor levels at the drinking water treatment plant level. Such treatment strategies to remove organic matter in source water include enhanced coagulation, electrocoagulation, membrane pressure processes (microfiltration (MF), ultrafiltration (UF), nanofiltration (NF), and reverse osmosis (RO)), and adsorption processes (granular activated carbon and ion exchange resin) (Priya et al., 2020). Granular activated carbon (GAC) is less efficient at removing dissolved organic nitrogen and bromide and thus the increased formation of more toxic N-DBPs and bromo-DBPs has been observed

(Cuthbertson et al., 2019). Pressure membrane processes such as reverse osmosis are effective at removing organic matter as well as bromide and iodide (Priya et al., 2020) however large scale applications may be impractical due to high costs and amount of water wasted.

In recycled waters, poor nitrification efficiencies at wastewater treatment plants have been linked to increased formation of toxic N-DBPs including HANs (Krasner et al., 2009). Thus, more efficient nitrification processes may be needed to ensure lower amine levels in recycled waters that may be later used for indirect potable reuse.

Section S4.1.2: Alternate Disinfection Processes

Another way to potentially mitigate DBP formation at the drinking water treatment plant level includes the use of alternative disinfection methods. While chlorine is the most widely used, other chemical disinfectants include chlorine dioxide, chloramine, or addition of ammonia to a preexisting chlorination system. Ozonation and UV are also increasing in use however they require the application of a secondary disinfectant to maintain disinfection within the distribution system (Richardson and Plewa, 2020). While the use of alternative disinfection processes may reduce overall DBP formation, increases in more toxic unregulated compounds has been observed (Krasner et al., 2006). For example, while the use of chloramines have significantly reduced DBP levels, the increased formation of more toxic iodo-THMs and HANs has been observed (Richardson, 2011; Richardson and Plewa, 2020). Promising disinfection approaches in mitigating DBP formation and toxicity as suggested by (Richardson and Plewa, 2020) include:

- Granular activated carbon (GAC) + low dose of chlorine
- Membranes + low dose of chlorine
- UV + low dose of chlorine
- Chlorine dioxide

Section S4.2: Household Level Treatment

Due to high variability and unknown formation within the distribution system, treatment at the household level may be key to mitigating DBP exposure, especially in regions that use recycled water or source waters that are heavily impacted by anthropogenic activity. House water treatment strategies include point-of-use filtration (reverse osmosis, activated carbon, ion exchange, microfiltration), thermal methods such as boiling, and water storage (Xiao et al., 2020). While these methods have been shown to significantly reduce THM_4 and HAA_5 concentrations, their effectiveness for unregulated compounds such as HANs, HALs, and HNMs are not well known or studied (Xiao et al., 2020). The idea of taking tap water and further purifying it is similar to bottled water and refill station purification processes which overall had relatively low DBP–FP concentrations in our results.

Water storage as a treatment alternative has some ambiguities. Increases in THM formation have been observed when tap water is stored in a closed container at 4°C between 4 to 48 hours (Chowdhury et al., 2010). This is due to extended reactions between precursors and disinfectants. However, THM levels were reduced when stored with an open container due to their high volatility (Chowdhury et al., 2010).